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Original Contributions

Value of Succinylcholine Chloride in Electroconvulsive Therapy

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IN RECENT months succinylcholine chloride (Anectine*) has become established as the most practical and valuable muscle relaxant for use with electroconvulsive treatment (E.C.T.). Every physician who uses electroconvulsive therapy should familiarize himself with this drug and be able to use it if the occasion demands, though he may or may not wish to adopt it as a routine procedure. One never knows when he will encounter one of those desperate situations when urgently needed electroconvulsive therapy is contra-indicated by some physical abnormality (e.g.—an acutely distributed patient with a fresh fracture). The purpose of this paper is to report on our experience and to outline a variety of techniques for use under varying circumstances.

Succinylcholine chloride is a white crystalline water-soluble solid made up of two molecules of acetylcholine chloride linked together at the alpha methyl groups. When injected intravenously, it rapidly produces voluntary muscle relaxation or flaccid paralysis. It acts at the motor end plate, as does acetylcholine, blocking myoneural conduction; this action is of short duration (one-half to three minutes) as the drug is rapidly hydrolyzed into the normal metabolic products of choline and succinic acid. The flaccidity develops in an order similar to that with curare, viz: (a) eyelids, (b) face, (c) extremities, (d) back, (e) intercostals, (f) diaphragm. Disappearance of the paralysis is in the reverse order. It is possible to give electroconvulsive therapy with the patient so completely paralyzed that almost no muscle movement occurs.[3]

Following E.C.T., the average patient usually begins to breathe promptly—as if no succinylcholine had been used. However, prolonged apnea (three to twenty minutes) may occur in unusual cases. There is no known antidote; prostigmine is contra-indicated. Artificial respiration by manual chest compression is insufficient in such cases of prolonged apnea. Because of this possibility, one should not use succinylcholine chloride unless he is skilled in the administration of oxygen with a positive pressure mask.

The mechanism of prolonged apnea is not completely understood. There is some evidence that the cholinesterase of the plasma may play a part. Bourne and Evans[2,4,5] observed that eight patients with delayed recovery showed a low plasma cholinesterase level, suggesting that caution be used in patients with liver damage. As an added safety precaution it may be advisable to have a suction apparatus, airway, and intubation tray available. If the physician is inexperienced or the patient's condition warrants it, an anesthesiologist could be called to assist in the first few treatments.

There may be minor but definite side effects and after effects. When the drug is given rapidly, the paralysis is often preceded by a transient stimulatory phase, involving some muscle twitchings. Murray[9] reported chest soreness, burning in the throat, and pain in the jaw and back of the neck as the three occasional complaints of his patients. He injected himself with 20 mgm. of succinylcholine chloride and confirmed the above symptomatology referred to by his patients. The pain and burning diminished gradually over a

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*There are other drugs such as succinylcholine iodide which are reported to have essentially the same physiological action, but we have had experience only with the succinylcholine chloride. ('Anectine' is the Burroughs Wellcome and Company brand name.)

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period of twelve hours. On the other hand Mayrhofer[7] gave himself a series of injections and experienced no side effects whatsoever.

As far as the evidence indicates at the present time, succinylcholine chloride has no observable detrimental effects on the cardiac status. Nowill, Wilson and Borders[10] report some changes in electrocardiogram, blood pressure, and pulse recordings, which were however all transient (less than three minutes) and partially eradicated by the proper dosages of atropine sulfate (gr. 1/75 or higher) administered before the treatment. As a result succinylcholine chloride has been proposed as the drug of choice for cases with increased cardiac risk. In such cases we may use bag and mask controlled respirations with high oxygen concentrations before and/or after electroconvulsive therapy.

There are several possible techniques of administration. Most writers[1,6,8,11] advocate succinylcholine chloride-sodium pentothal mixtures. Murray[9] advocates the use of succinylcholine chloride alone, feeling that the addition of sodium pentothal needlessly complicates the treatment procedure.

The basic technique of the current University Hospitals plan involves the use of succinylcholine chloride by itself. This is modified as the need arises, as will be described later. As usual the patient gets no breakfast. Atropine, gr. 1/75 (0.8 mgm.) is given hypodermically thirty minutes before treatment. Everything is made ready before the injection of succinylcholine; that is, head band and mouth protector are in place, and the shock machine is warmed up and set. We place no roll under the patient's back, nor do we hold him.

With this technique the time factor is critical if the patient is to be spared unpleasant sensations. We inject the succinylcholine chloride rapidly intravenously; that is, in about three to five seconds. Verbal reassurance is given to the patient during the few seconds that it takes the drug to act. For example, he may be told that he will feel the medicine beginning to take effect but that he will be asleep very soon. Twenty-five seconds to thirty seconds after termination of the injection, the treatment is given. The hand-grip disorganization sign employed by Murray[9] had not been consistently reliable in our experience.

Ten mgm. (0.5 cc.) is the usual initial dosage. If this fails to produce adequate relaxation, the dosage of subsequent treatments is increased. We have seldom found it necessary to go beyond the 20 mgm. level. Once the dose is established for a given patient it has been found to remain constant.

Modifications of the basic technique are sometimes utilized. If a patient is fearful before the treatment or restless after it, sodium amytal, gr. iii orally one hour beforehand, is helpful. If this is insufficient and especially if the patient is markedly upset or complains strongly of muscle twitchings and/or smothering sensations, intravenous pentothal is used instead. Sufficient pentothal (from 50 to 200 mgm.) is injected to make the patient drowsy; this is followed by administration of succinylcholine chloride via the same needle but from a different syringe; then forty-five to sixty seconds later, the treatment is given. Some clinicians[6] employ succinylcholine chloride-pentothal mixtures in the same syringe. This method may work if the mixture is administered at once. However, since succinylcholine chloride hydrolyzes rapidly when mixed with pentothal, the technique would appear unreliable, as the effective amount of succinylcholine which actually reaches the patient will be unknown and variable.

During the period from July 1, 1953 to June 30, 1954, we have treated 114 patients with a total of 957 electroconvulsive treatments. A breakdown of the 957 treatments revealed the following:

137—Treatments with sodium pentothal alone. Of these 130 were given during the first part of our 12-month period. Later when the staff had become impressed with the value of succinylcholine, almost everyone was given its benefit.

667—Treatments with succinylcholine chloride alone. In 23 treatments the patient was given sodium amytal, gr. iii, orally one hour beforehand.

110—Treatments with succinylcholine chloride and sodium pentothal in combination.

43—Treatments with no medication.

Since we are stressing the role of succinylcholine chloride in this report, we shall discuss our findings on the bases of (1) complaints of the patient concerning the immediate effect of the drug; (2) post-treatment complaints, and (3) degree of modification of the grand mal seizure.

1. In about one-quarter of the treatments, there were complaints relative to the immediate effect of succinylcholine just before the administration of electroconvulsive therapy. Of those who

had complaints, about half voiced them immediately; the other half told us about them later in the day, that is, they remembered well how they had felt. Most of these complaints were minor, consisting of vague "discomfort," mild restlessness and moderate anxiety. Several patients on one or more occasions revealed considerable apprehension, spoke of twitchings of the muscles (especially of the face) and smothering sensations. One patient in describing her distress said, "I couldn't breathe: I wanted to talk and scream but I couldn't." About 80 per cent of these latter difficulties were resolved, either by sodium amytal orally beforehand or by the use of a pentothal-succinylcholine combination. The above complaints were more numerous during the first few months of our experience. As our technique improved and as our staff gained confidence, the complaints occurred less and less frequently.

2. Probably most patients who receive E.C.T. suffer from some degree of post-treatment discomfort such as headache, nausea, occasional vomiting. These complaints occurred in many of our patients irrespective of the type of medication used. In addition to the above, several patients had complaints which appeared to stem from the use of succinylcholine chloride: (a) dryness of the throat, (b) pain in the neck, chest, and back, and (c) jaw and calf pain. Patients who did not receive succinylcholine chloride did not complain of these symptoms. These complaints persisted with decreasing severity for only a few hours. They were seldom mentioned spontaneously but could be elicited by questioning after about 15 per cent of the treatments.

3. Modifications of the grand mal seizures were arbitrarily divided into the classifications good, fair, poor or none. We considered the modification to be good if there was very little tonic and clonic reaction. We did not feel it was necessary that the convulsive seizure be completely eliminated as would be possible with this drug. If the patient had only slight modification the reaction was classified as poor. On a dosage of 10 mgm. a trifle more than half of the treatments fell into the fair or poor categories; but when the dosage was raised to 15 or 20 mgm., 80 per cent of the modifications were good and 15 per cent were fair. In two cases, when intravenous pentothal was used in combination, it was necessary to raise the dose of succinylcholine (once up to 25 mgm.) to obtain a satisfactory modification of seizure. The succinylcholine had no apparent effect whatsoever in two other cases in spite of 20 and 25 mgm. doses. We did not go higher. Once a satisfactory dosage was obtained for a given patient, we found this to produce a constant effect. There was one exception—in this patient for some unknown reason the same dosage brought different responses on different days.

It is not part of our routine to obtain spine x-rays before E.C.T., and we take post-shock x-rays only if the patient has physical complaints referable to a possible skeletal injury. In the above-reported cases, there was no clinical evidence of fractures or dislocations. X-rays were done in the case of four patients all of whom complained rather markedly of back pain; they were all reported as negative.

We had a few special problems. One female patient was apneic for seven minutes. She had been given 250 mgm. pentothal, followed by 10 mgm. succinylcholine, preceding the E.C.T. She received oxygen via the bag-breathing method until she began breathing again. A second female patient appeared to have recovered satisfactorily but suddenly became apneic in the recovery room at least five minutes after E.C.T. had been administered. She was apneic for two minutes, was not given oxygen, recovered spontaneously. These two patients experienced no difficulties in succeeding treatments. The first treatment of another female patient, utilizing 10 mgm. succinylcholine, produced a complaint of severe pain in the neck with muscle spasm, necessitating cessation of therapy for ten days. Treatments were then resumed with 15 mgm. succinylcholine without further difficulty. Whether or not this was related to the succinylcholine, or even to the E.C.T., is open to question.

It is of some interest to note that the patient who had the greatest difficulty in this series was one who received no succinylcholine but rather pentothal alone (150 mgm.). He was apneic for 10 minutes and suffered severe laryngospasm. He was breathed by the bag method until he took over control himself. Subsequent treatments without any medication led to mild laryngospasm but much less severe in nature.

It would be well to mention that the use of succinylcholine chloride did not appear to influence the therapeutic effect of E.C.T. in any way.

Summary

Succinylcholine chloride has proven to be an ideal muscle relaxant for routine use with electroconvulsive treatment. Because of its rapid but short-lived action, it is far superior to most other drugs. Its use allows the physician to administer electroconvulsive therapy to a larger variety of patients than heretofore; aged persons, those with orthopedic complications, and cardiac cases, can be treated with less risk.

We have described our treatment procedures: as a routine, we have found it safer and simpler to give succinylcholine alone. This is satisfactory in the majority of cases. For those patients who are unduly fearful or anxious, the procedure may be modified by the use of a barbiturate. Because of the occasional occurrence of prolonged apnea, a positive pressure oxygen apparatus must be available.

Although the patients occasionally complain of certain side effects, we wish to stress that these are minor in nature. They occurred much less frequently as we gained experience and our technique smoothed out.

With a dosage of 10 to 25 mgm., we have found succinylcholine to sufficiently modify or eliminate the convulsive reaction in almost everyone. There were two exceptional cases in which it had no effect whatsoever.

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NUMBER OF PHYSICIANS REACHES NEW PEAK

A record graduation of 6,861 physicians during the past year by the nation's medical schools has boosted the ratio to an all-time peak of one physician for every 730 persons in the United States. This ratio will be lowered even more in the next few years, as the number of medical graduates is expected to rise due to the continued expansion of the country's medical schools.

Today's physician population has now reached approximately 220,100. The record graduation figures, as released in the 54th annual report on medical education in the United States by the American Medical Association's Council on Medical Education and Hospitals, include the following:

Enrollment of 28,227 is the largest number of medical students in the history of the United States.

Freshman class enrollment of 7,449 also is a record.

More than 76 million dollars was spent during 1953-1954 for new facilities, remodeling or completion of buildings for medical instruction.

Budgets for medical schools during 1954-1955 total more than 143 million dollars.

A total of 21,328 physicians did volunteer teaching without pay during the year.

Ten new four-year schools are in construction or planning stages and will be in operation within the next few years These schools will be at the Universities of California, Mississippi, Miami, Missouri, Florida, West Virginia, Kentucky, North Dakota and Yeshiva University of New York and Seton Hall University. In addition, three other medical schools are being considered.

Hearing Loss in Children

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FOR THE past two years the Committee on Conservation of Hearing of the Minnesota State Medical Association has been engaged in formulation of a program which is directed toward preservation of hearing among the people of Minnesota. Five population groups, each of which has somewhat different problems, are being considered. The categories are: (1) children of preschool age, (2) children of school age, (3) adults, (4) hearing loss in industry and (5) hearing loss among the aged.

Initial emphasis is being placed on the children of school age. This group was selected owing to means of locating individuals through the agencies of the public health services and the school systems in our communities. The program for this group is threefold: location of individuals, information to physicians, and information to parents of handicapped children.

For purposes of location the state was divided into seven districts. Each district was assigned to a member of the committee residing in that district. Committee members asked public health agencies and school systems within the individual districts for the names of children with hearing handicap. For purposes of this survey, handicapping loss of hearing was arbitrarily set at a loss of 30 decibels or more in both ears in one or more of the speech frequencies of 500, 1,000 and 2,000 cycles per second. The co-operation given by these agencies has been admirable. The names of 1,106 children have been submitted to the committee. This number is not complete. Facilities for testing are not available in all communities. One district reported by code number only.

Detection

Early detection is the key to successful therapy or rehabilitation of children who have hearing loss. Our therapeutic victories will be confined to those patients whose hearing loss is due to inflammation of the middle ear or to lymphoid hypertrophy in the nasopharynx. The success of therapy depends on achieving remission of the pathologic process before permanent changes occur. The results of rehabilitation programs are less dramatic, but are no less dependent on early recognition of the handicap with early institution of training measures.

The parents are usually the first to suspect impairment of hearing in the young child. The sense of hearing is present at birth. Some newborn infants will respond to a handclap, although response is inconsistent. It is not usual for children to be brought to a physician for evaluation of hearing before the age of one year. Only severe defects of nerve function are detected at early age. Unilateral or partial loss of hearing may escape detection by the parent. Parents are quite likely to be aware of the bilateral partial loss of hearing associated with bilateral inflammation.

The examination of hearing of young children requires patience, skill and equipment. Froeschels and Beebe found that thirty-one of thirty-three newborn infants responded to tests using Urbantschitsch's whistles as the stimulus. Response was usually by acousticopalpebral reflex. No newborns tested responded to tuning forks. The authors attributed this difference to the strong stimulus required to overcome poor middle-ear function in the newborn.

Ewing and Ewing studied children with normal as well as those with defective hearing. They reported that from birth to two months of age percussion sounds elicit reflex response; that between two months and one year the voice becomes the better stimulus; that moderate voice is better than loud or whispered voice; that learned

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From the Section of Otolaryngology and Rhinology, Mayo Clinic and Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

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responses replace reflex responses at this age; and that children older than one year respond best to the quiet voice. Various toys were used to hold the child's attention and to evaluate response to spoken stimuli. Similar tests were used for children suspected of deafness. Reflex responses were replaced by learned responses at a later age than in children with normal hearing. Residual hearing for loud sounds was determined by reflex response to loud stimuli. The authors concluded that if a child does not respond to voice by the end of one year he is deaf or has some other gross defect; that if he never turns his head toward the voice of a familiar person at three feet he is totally or severely deaf; and that all children more than the age of one year who fail to respond to a loud voice are totally deaf.

Quantitative hearing tests for young children require special equipment. Bordley and associates reported favorable results from the use of the psychogalvanometer. Dix and Hallpike developed another conditioned-reflex test using a "peep show."

Occasionally hearing loss is first noted by teachers after the child attains school age. Poor progress and lack of attention in school may suggest deficient hearing.

The screening type of tests carried out by school and public health organization is responsible for recognition of many children with partial loss of hearing. These tests are of tremendous value. In my experience, errors tend to be in the direction of indicating too great a loss of hearing. This is no doubt largely due to inattention on the part of the child.

Otologic evaluation is the final step in detection. The otologist has the experience in diagnosis necessary to select those children suitable for therapy from the group requiring rehabilitation measures. He also has the special equipment and skill needed for evaluation of the hearing defect and for following the progress of therapeutic efforts. The experience of the otologist in treatment of otic infection is necessary in those patients who fail to respond to usual measures. The otologist is the only physician who is willing to treat chronic infections of the ear. Chronic infections merit his care as soon as they are recognized. Adequate, early treatment of chronic infection may prevent continuing destruction of the sound-conducting mechanism.

Treatment

Diagnosis of the cause of hearing loss is prerequisite to treatment. The conditions which cause hearing loss in children and which are amenable to therapy are occlusions of the external auditory canal by cerumen, inflammation, foreign bodies or atresias, and interference with the function of organs in the tympanic cavity. Reversible causes of middle-ear dysfunction are acute otitis media, serous otitis media, some cases of chronic otitis media, and impaired function of the eustachian tube. Hypertrophy of the lymphoid tissue in the nasopharynx is an important causative factor in lesions of the middle ear. Infections of the nose and tonsils less frequently cause disease of the ear.

Causes of hearing loss in children not amenable to therapy are the end result of middle-ear inflammation and the loss of nerve function. The latter may be congenital in origin owing to heredity, rubella infection in the mother during the first trimester of pregancy, erythroblastosis or syphilis, or acquired, owing to mumps, trauma, meningitis or syphilis.

The outstanding approach to relief of hearing loss in children is management of lymphoid hypertrophy in the nasopharynx. Adenoidectomy is the principal weapon. In order to achieve relief of middle-ear and eustachian-tube dysfunction, adenoidectomy must be done carefully and thoroughly. The philosophy that adenoidectomy is an adjunct procedure secondary in importance to tonsillectomy is untenable. Blind adenoidectomy is to be avoided. It is imperative that the nasopharynx be subjected to painstaking direct visual inspection and that all remaining lymphoid tissue be carefully removed before the operation is completed. The palate retractors of Lothrop and Senturia are effective instruments. Finger palpation is valuable in locating bits of lymphoid tissue high in the nasopharynx. The Kelly direct-vision adenotome is also a valuable instrument. Adequate exposure requires movement of the patient's head to bring all areas of the nasopharynx into view. Sharp alligator biting forceps such as those designed by Meltzer facilitate removal of tissue from the lateral bands. For removal of the main adenoid mass, a series of La Force adenotomes in various sizes is essential. One cannot work efficiently with an instrument either too large or too small for the nasopharynx.

Each surgeon should plan and follow a definite routine in adenoidectomy which assures clean removal of all lymphoid tissue. The final step should be a searching inspection of the surgical field.

Age is not a contraindication to adenoidectomy if sufficient reason exists for doing the operation. The adenoid is not confined in a capsule. Some lymphoid tissue remains after the most painstaking operation; therefore, recurrence may be anticipated when operation is done in very young children. It is well to advise the parents of this possibility, as secondary operation may be necessary a few years later.

Irradiation of the nasopharynx has limited use in treatment of lymphoid hypertrophy. Small residual lymphoid masses may be reduced in size by irradiation although this agent is not satisfactory for removal of larger masses. Irradiation is not a substitute for adenoidectomy.

Finger massage of the nasopharynx has not proved effective for control of lymphoid overgrowth and control of infection.

Acute suppurative otitis media usually responds favorably to the use of antibiotic drugs. The outstanding cause of failure is inadequate dosage of the drug which results in temporary masking of the symptoms followed by recurrence of the disease. An adequate program is one which provides effective concentrations of the drug at the site of the infection for a period of time sufficient to eliminate the infection. Inadequacy may result from small doses, infrequent doses, or discontinuation before the infection has subsided.

Myringotomy, once the principal weapon in treatment of acute otitis media, is no less valuable today. Institution of surgical drainage of the middle ear early in the course of acute suppuration promotes early healing and decreases the possibility of permanent changes in the middle ear. Myringotomy certainly should be done in every instance of delayed resolution of acute otitis media.

Serous otitis media, if repeated or prolonged, may have permanent influence on hearing. Vigorous therapy is indicated. A few patients respond to the use of antihistamine drugs or inflation of the eustachian tube. Direct removal of secretions from the middle ear by myringotomy and suction or by needle puncture is the preferred direct treatment. Treatment of adenoid hypertrophy is of primary importance. Treatment of nasal infection or of allergy is effective in some patients. Mastoidectomy has been advocated for persistent disease. In my experience the results of mastoidectomy have been unsatisfactory.

Chronic otitis media is basically a destructive process. Early evaluation and treatment by an otologist may in some instances arrest the destruction of tissue and avoid further loss of hearing. Treatment by antibiotic drugs administered systemically is unsatisfactory. Local treatments directed toward removal of debris, promotion of free drainage, and control of infection by topical application of effective drugs are useful. Modified radical mastoidectomy effectively eliminates the disease and at the same time preserves hearing in suitable patients.

Other methods of treatment which are of value in prevention of ear disorders which lead to loss of hearing are treatment of nasal disorders, infections, allergies, or obstruction, and immunization against or preventive management of the acute diseases of childhood.

There is no convincing evidence that nerve deafness benefits from vitamin or mineral therapy.

Rehabilitation

Rehabilitation of the hard of hearing is not the province of the physician; however, the physician can be of great service to his patients by advising them of the possibilities, the aims, the methods and the availability of rehabilitation.

Areas in which advice of the physician is of especial value to the patient or his parents are those of the hearing aid, parental instruction, and available school instruction for the handicapped child.

Selection of Hearing Aids

The hearing aid has a single function: to increase the loudness of sounds reaching the ear. The simplest hearing aid is the ear trumpet or speaking tube. The diaphragm-and-rod prosthesis is useful for a limited number of individuals. This system is more adaptable to use by adults than by children. The electric hearing aid has many features which make it the most widely used prosthetic device for the hard of hearing.

The electric hearing aid is a miniature system made up of the component parts of the public address system, microphone, amplifier, power source and speaker. It is essential that all component parts be included in the instrument for practical function. Advertisements for hearing aids without batteries, cords or earpieces are misleading.

Receivers

Persons with conductive deafness and normal nerve function may be fitted with a bone-conduction receiver. The air-conduction receiver is however, more efficient and should be advised unless disease of the middle ear or canal interferes with wearing the insert. Hidden-type ear molds sacrifice efficiency by reducing intensity and introducing distortion into the signal. These factors are roughly proportioned to the length of the air column between the receiver and the ear. Day has advocated wearing the receiver in the ear where it can be seen. This prompts speakers to enunciate clearly.

The standard hearing aids are very similar in their performance characteristics. Much of the difference in cost between different makes of hearing aids is attributable to the service of dealers in assisting and encouraging new users of hearing aids. For this reason the service policies of the dealer are more important than the make of instrument, especially for the person wearing an aid for the first time.

Since the hearing aid is a signal booster only, it cannot be expected to amplify wanted sounds and suppress unwanted sounds, or to clarify poorly enunciated speech. Amplified sound is most helpful for persons with conduction losses, but is of limited benefit for persons with perception loss. Amplification is of least benefit to the ear with sharp dips or drops in the pure-tone curve, or the ear which distorts the signal as in patients with Ménière's disease.

Persons whose threshold for the better-hearing ear is 35 decibels or more will benefit from use of a hearing aid. Those whose loss is between 35 and 75 decibels usually experience satisfactory results. With losses between 75 and 95 decibels the results justify wearing the instrument.

A hearing aid is of doubtful value to the patient whose hearing threshold for the speech frequencies in the better ear is 95 decibels or more.

The most recent major development in hearing aids is substitution of the transistor for the vacuum tube. This results in a noteworthy decrease in power consumption, and thus in battery costs. Laboratory reports indicate that transistor instruments compare favorably with vacuum-tube instruments in range and fidelity of amplification for speech. However, clinical comparisons reveal a preference for the tone qualities of the vacuum-tube instruments. Instruments in which only the power tube is replaced by a transistor combine the best features of the two types.

Instruction by Parents

Instruction in the home by parents is of utmost importance to the hard-of-hearing child, particularly for the child of preschool age. It is important that training begin as soon as the defect is recognized.

The basic objectives of training in the home are fourfold: (1) talk to the child as much as possible, taking care to talk at close range and to face the child when talking, (2) associate words with objects and actions, (3) encourage the child to speak, (4) encourage contact with hearing children. Do not build up the idea that the hard-of-hearing child differs from other children.

Parents need help in order to instruct the child effectively. Advice to parents is available through the following: Institute for Parents, University of Minnesota; local hearing societies, Minneapolis, St. Paul, Duluth and Rochester; Audiology Clinics, University of Minnesota and Mayo Clinic; Committee on Conservation of Hearing, Minnesota State Medical Association; and John Tracy Institute, University of Southern California, Los Angeles, California.

School Instruction

School instruction plays a vital role in training of the hearing-handicapped child. School instruction is available in Minnesota.

Nursery schools for hearing children are valuable in developing the social contacts of the hard-of-hearing child, and should be used where special schools are not available. Special nursery schools are maintained by the Minneapolis Hearing Society and the St. Paul Hearing Society. Private boarding-school facilities are available at the Robey-Allen School in Faribault.

The public school systems of many communities maintain speech and hearing therapists to aid the hearing-handicapped child.

(Continued on Page 27)

Minnesota Maternal Mortality Study

By the MATERNAL MORTALITY COMMITTEE
of the Committee on Maternal Health,
Minnesota State Medical Association

THIS REPORT is the fourth concerning the state-wide Minnesota Maternal Mortality Study. The material to be presented, which has not been previously reported, includes all maternal deaths occurring from April 1, 1952, through March 31, 1953, hereafter referred to as the "1952 Survey." References will be made to previous reports dealing with the study in 1941-42, 1950 and 1951.[1,2,3] Details regarding the method by which the study is carried out have been published.[2]

As in previous years, all deaths associated with pregnancy and those occurring during a period of three months postpartum are included regardless of cause of death (Table I). Deaths due to chorionepithelioma are also included. The study thus includes many deaths in which there is no obvious direct relationship between pregnancy or the postpartum state and the death, but the study has made it amply clear that death certification information cannot be relied upon in attempting to distinguish "obstetric" from so-called "non-obstetric" deaths.[4] Exclusion of any maternal deaths from detailed study would therefore necessarily be done on an arbitrary basis. It has been considered preferable to include all deaths and to report a gross or over-all mortality rate. For purposes of comparing the Minnesota Maternal Mortality rate with that of the United States generally the rate excluding deaths considered "non-obstetrical" by the committee is also shown in Table I. As indicated in Table I the mortality rate for 1952 closely approximated that for 1951. The low rate is considered further evidence of the continuing excellence of the obstetric care being administered by Minnesota physicians.

TABLE I. MATERNAL DEATHS IN MINNESOTA. STUDIES FOR 1941, 1950, 1951 AND 1952

	Number Deaths	Number Births	Gross Mortality Rate Per 1000 Live Births†	Maternal Mortality Rate Per 1000 Live Births§	U. S. Maternal Mortality Rate Per 1000 Live Births
1941	112	55,293	2.03	1.68	2.8*
1950	68	76,074	0.89	0.59	0.83**
1951	57	80,099	0.71	0.45	0.71**
1952	58	78,990	0.73	0.42	

*Average for 1941 and 1942
**Unofficial
†Including all maternal deaths from whatever cause
§Excluding deaths considered as non-obstetrical by the maternal mortality committee

TABLE II.
SOURCE OF REPORT OF MATERNAL DEATHS

	Number	Per Cent
Cases found from death certificates (with or without preliminary reports from physician, hospital, etc.)	135	73.8
Sources other than death certificates	48	26.2
Cross-match of death with birth certificate 24-13.1%		
Others 24-13.1%		
Total Deaths 1950, 1951, 1952	183	100.0

Source of Reporting

The source of reporting of the maternal deaths for the past three years is shown in Table II. The percentage in each category for each of the three years has been approximately the same. It can be seen that approximately one-fourth of the cases were found by other than the usual means and therefore would not be included in the ordinary reporting of the maternal mortality rate. Many of the deaths so discovered are so-called non-obstetric deaths, but many others are not. In the past year they have included such cases as an air embolus from attempted self-induced abortion, a death from arteriolosclerotic toxemia occurring three weeks postpartum and a death from postpartum hemorrhage.

Cross matching of all death certificates of women aged fifteen to forty-five years with birth certificates has led to the discovery of twenty-four cases in three years. Another twenty-four cases were found through a variety of sources including reports of hospitals and physicians. Such reports are now required by the State Department of Health within three days of the time of death of a patient dying in association with pregnancy

TABLE III. ANALYSIS OF DEATH CERTIFICATES

	No.	Per Cent	No.	Per Cent
Death certificates correct and complete in all details	57	31		
Death certificate correct in all details reported	54	30	111	61
Death certificates incorrect			72	39
Death certificates incorrect as to cause of death			52	28
Total maternal deaths (1950, 1951, 1952)			183	100

TABLE IV. PREVENTABILITY OF MATERNAL DEATHS IN MINNESOTA

	1941		1950		1951		1952	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Preventable	82	73.2	22	32.4	24	42.1	10	17.2
Not preventable	27	24.1	45	66.1	32	56.1	47	81.0
Not determinable	3	2.7	1	1.5	1	1.8	1	1.7

TABLE V. OBSTETRIC AND NON-OBSTETRIC DEATHS

	1941		1950		1951		1952	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Obstetric deaths	93	83.0	46	67.6	39	68.4	33	56.9
Non-obstetric deaths	19	17.0	22	32.4	18	31.6	25	43.1

or the postpartum state. It is felt that no maternal deaths in Minnesota are now being missed with the possible exception of some associated with pregnancies of less than twenty weeks duration in which the filing of birth certificates is not required and occasional cases occurring in undelivered patients. It seems likely that a correction factor of considerable magnitude is applicable to rates being reported from states where similar efforts at case findings are not carried out. In reports arrived at by ordinary means this error is added to that involved in the exclusion of "non-obstetric" deaths. Further gross errors follow the use of unchecked death certificates for information concerning maternal mortality rates (Table III).

Inaccuracies of Death Certificates

The data in Table III indicate that it is hazardous at best to arrive at conclusions based on information derived from unchecked death certificates. Only 31 per cent were complete and correct and 39 per cent were incorrect in some detail. Perhaps the most significant feature is the fact that thorough evaluation of the cases has led the committee to change the diagnosis of the cause of death in 28 per cent of the cases. The inaccuracies were of many types, and not always unintentional. A common error was to make a positive diagnosis on the death certificate when none was possible. There have been several deaths each year in which the true cause of death could not be determined even after complete autopsies were done. The cause of death in such cases should have been listed as unknown, yet in each instance a definite diagnosis was inserted on the death certificate. In 1952 the cause of death in one instance was listed as pulmonary embolism and the patient actually died of postpartum hemorrhage. In another instance a patient known to have a normal heart died as a result of drowning. The death certificate was signed by a lay coroner with the cause of death being listed as acute cardiac failure actually due to mitral heart disease. Another patient whose death certificate showed the cause of death as pulmonary embolism actually died of air embolism from air pumped into her veins under pressure through an empty transfusion bottle. Still another patient whose death certificate showed epilepsy as the cause of death actually died of postpartum hemorrhage. Many more examples from each year's survey could be cited.

Results of 1952 Survey

A most notable achievement of 1952 was the reduction of the number of deaths considered preventable to ten or 17.2 per cent of the total (Table IV). This would scarcely have been believed possible eleven short years ago when there were no less than eighty-two preventable deaths or 73.2 per cent of the total for that year. Part of the reduction in numbers of preventable deaths may be accounted for by the fact that as shown in Table V there was a further reduction in the number of deaths due to "obstetric" causes. The majority of the preventable deaths each year are in this group. On the other hand, the reduction of the absolute numbers of deaths due to obstetric causes is itself a real indication of improvement in care.

TABLE VI. TYPE OF DELIVERY

	1950		1951		1952	
	No.	Per Cent of Delivered Patients	No.	Per Cent of Delivered Patients	No.	Per Cent of Delivered Patients
Undelivered	17		21		27**	
Spontaneous	24	46.2	16	44.4	13	40.6
Operative	28	53.8	20	55.6	19	59.4
Indicated	19	8		16		
Not Indicated	9	12		3		
Totals	69*	100.0	57	100.0	59	100.0

*One set twins.
**Includes seven delivered by post-mortem cesarean section.

TABLE VII. SEROLOGIC TESTS FOR SYPHILIS

	1941		1950		1951		1952	
	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients
Unregistered	18		6		13		12	
Not obtained	64	68.1	38	61.1	25	56.8	20	43.5
Obtained	30	31.9	24	38.9	19	43.2	26	56.5

TABLE VIII. RH DETERMINATIONS

	1950		1951		1952	
	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients
Unregistered	6		13		12	
Not obtained (regist. pts.)	23	37.1	17	39.5	8	17.4
Obtained at hospital	11	17.7	10	23.3	9	19.5
Obtained early in pregnancy	28	45.2	16	37.2	29	63.0
No data			1			
Totals	68	100.0	57	100.0	58	99.9

There are other evidences of improvement in care such as is indicated by a review of the operative deliveries (Table VI). While the number of operative deliveries in 1952 did not change significantly, the remarkable fact is that only three (15.8 per cent) were considered not indicated. This compares with approximately 40 per cent in the years 1950 and 1951 combined. Only four patients who died in 1952 were delivered by cesarean section. Adequate indication existed for three of these operations. One unindicated mid-forceps and one unindicated version and extraction were done. Of interest were seven post-mortem cesarean sections all said to have been done within five minutes of death. No living infants resulted. In previous years of the study five other post-mortem cesarean sections were done again with failure in all.

Serologic tests for syphilis were done in greater numbers in 1952 (Table VII). A much larger increase took place in the number of Rh determinations carried out (Table VIII). This was done early in pregnancy in twenty-nine cases or 63 per cent of the patients registered for care, and in five of the cases where it was not done early the patients were primigravidas and the indication therefore not so urgent.

The number of autopsies performed is shown in Table XIV. The percentage in each category for the past three years has been approximately the same. While 38.8 per cent adequate autopsies is considered good and is, of course, a vast improvement over the 8 per cent obtained in 1941, there is room for more improvement here. The majority of the failures to obtain autopsies are due to physician inertia. The twenty-one cases in which autopsy was not requested in 1952 include five coroners' cases.

More consultations were obtained in 1952 (Table IX), and it was thought indicated in only eight of the cases in which it was not obtained. However, further consultation was indicated in the case of four patients seen by one consultant and in many other instances consultation was obtained too late to be of value.

TABLE IX. CONSULTATIONS

	1941		1950		1951		1952	
	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients
None	63	56.2	29	42.6	28	49.1	22	37.9
Indicated		17		14		8		
Not indicated		12		14		14		
Adequate	10	9.0	22	32.4	17	29.8	30	51.7
Inadequate	39	34.8	17	25.0	12	21.1	6	10.3
Totals	112	100.0	68	100.0	57	100.0	58	99.9

TABLE X. PELVIC MEASUREMENTS

	1941		1950		1951		1952	
	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients	No.	Per Cent of Registered Patients
Unregistered	18		6		13		12	
Not measured or incompletely measured	77	81.9	40	64.5	32	72.7	25	54.3
Measured	17	18.1	22	35.5	12	27.3	21	45.7

TABLE XI. PRENATAL CARE

	1941		1950		1951		1952	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Adequate	2	1.9	15	24.2	6	13.6	10	21.3
Faulty, contributory	55	52.9	14	22.6	10	22.7	5	10.6
Faulty, not contributory	47	45.2	33	53.2	28	63.6	32	68.1
None	8		6		13		11	
Totals	112	100.0	68	100.0	57	99.9	58	100.0

The data in Table X shows a very gratifying increase in the frequency with which pelvic measurements were done. Notwithstanding the improvement, measurements were not done or were incompletely done in more than one-half of the patients who died. It is difficult to understand why this is true. The evaluation of the pelvis necessary to satisfy the minimum requirements as defined by the committee include measurement of the transverse diameter of the outlet (satisfied by the "fist" test), a measurement of the diagonal conjugate and palpation of the sacrum and ischial spines.

The amount of time and effort involved in this evaluation beyond that required for an ordinary pelvic examination is negligible. The rewards involved do not require repeating here. There is evidence that some re-education regarding the value of the various pelvic measurements is needed in some areas. In the past two years fifteen of eighteen incompletely measured pelves were evaluated only with respect to the external measurement including the interspinous, intercristal, intertrochanteric and Baudelocque's diameters. These measurements are now widely recognized as being valueless in estimating the capacity of the true pelvis although they have some value for other purposes. Yet in each instance the physician involved apparently felt that he was satisfying the necessary requirements regarding pelvic mensuration.

The data regarding care given in the prenatal period, during labor and delivery and in the puerperium to patients who died during the four years of the study are shown in Tables XI, XII and XIII. The minimum requirements for adequate obstetric care adopted by the committee have been published.[2] Adequate care was given in a significantly higher percentage of cases in 1952, but there is room for much improvement. In the most significant categories, faults in care which contributed to the patients' deaths, particularly gratifying reductions took place in 1952, For example, in only 9 per cent was faulty care in labor and delivery contributory to the patient's death as compared to 68 per cent in 1941.

TABLE XII. CARE IN LABOR AND DELIVERY

	1941		1950		1951		1952	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Adequate	7	7.8	21	38.9	11	25.5	23	51.1
Faulty, contributory	61	67.8	26	48.1	23	53.5	4	8.9
Faulty, not contributory	22	24.4	7	13.0	9	21.0	18	40.0
None	22		14		14		13	
Totals	112	100.0	68	100.0	57	100.0	58	100.0

TABLE XIII. POSTPARTUM CARE

	1941		1950		1951		1952	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Adequate	4	4.8	18	37.5	14	42.4	16	55.2
Faulty, contributory	67	79.8	27	56.3	15	45.5	9	31.0
Faulty, not contributory	13	15.5	3	6.2	4	12.1	4	13.8
None	28		20		24		29	
Totals	112	100.1	68	100.0	57	100.0	58	100.0

TABLE XIV. AUTOPSIES

	1941		1950, 1951 & 1952	
	No.	Per Cent	No.	Per Cent
Obtained, adequate	9	8.0	71	38.8
Obtained, inadequate	27	24.1	6	3.3
Not requested	55	49.1	70	38.3
Permission refused	21	18.8	36	19.6
Totals	112	100.0	183	100.0

Causes of Death

The primary causes of death are listed in Table XV. Significant of the trend in obstetrics in Minnesota is the fact that over 40 per cent of the deaths associated with pregnancy or the postpartum state were due to non-obstetric causes. The fact that poliomyelitis accounted for as many deaths as hemorrhage, the leading "obstetric" cause of death, seems particularly noteworthy. The data in the table are largely self-explanatory. The deaths due to hemorrhage in the past three years are considered in a subsequent paragraph. Of the patients who died of toxemia in 1952, two had hypertension preceding the pregnancy. One of these died undelivered of a probable cerebral vascular accident and the other died of uremia three weeks postpartum. Four others died of eclampsia. The eclampsia followed very fulminating pre-eclampsia and three of these deaths were considered non-preventable. The fourth followed severe pre-eclampsia of at least three weeks duration. Both deaths due to amniotic fluid embolism were proven at autopsy. The deaths from air embolism followed attempted self-induced abortions. The inclusion of the two deaths associated with heart disease in the "obstetric" group might be questioned because one died of congestive failure from hypertensive heart disease three months postpartum and the other died of cardiac arrest at surgery for ligation of a patent ductus arteriosis fourteen weeks postpartum. The three deaths listed in the non-obstetric complications as due to intracranial hemorrhage apparently bore no relationship to the pregnancy. None of the three had toxemia. Two deaths oc-

TABLE XV. PRIMARY CAUSES OF MATERNAL DEATHS IN MINNESOTA. 1952 STUDY

Cause of Death		No.	Per Cent
Hemorrhage		11	19.0
Toxemia		6	10.3
Amniotic fluid embolism		2	3.5
Air embolism		2	3.5
Heart disease		2	3.5
Acute pulmonary edema		1	1.7
Septic abortion		1	1.7
Dehydration and electrolyte imbalance		1	1.7
Chorionepithelioma		1	1.7
Probable pulmonary embolus		1	1.7
Acute hepatitis		1	1.7
Unknown		4	7.0
Non-obstetrical complications		25	43.1
Bulbar poliomyelitis	11		
Trauma	4		
Intracranial hemorrhage	3		
Brain tumor	2		
Leukemia	1		
Suicide	1		
Obstruction trachea	1		
Acute glomerulonephritis	1		
Thrombocytopenic purpura	1		
Totals		58	100.1

TABLE XVI. CAUSES OF "OBSTETRIC" DEATHS AND PERCENTAGE DISTRIBUTION OF DEATHS CONSIDERED PREVENTABLE IN 1950, 1951 AND 1952

	No.	Per Cent	Preventable	Per Cent of Preventable Deaths
Hemorrhage	33	28.0	25	44.6
Toxemia	23	19.5	8	14.3
Infection	8	6.8	3	5.4
Anesthesia	7	5.9	6	10.7
Pulmonary embolism	7	5.9	0	0.0
Others	40	33.9	14	25.0
Total	118	100.0	56	100.0

TABLE XVII. ANALYSIS OF DEATH DUE TO HEMORRHAGE, 1950, 1951 AND 1952

		Number	Per Cent
Ruptured uteri and lacerations of cervix		15	45.5
Accouchement force	6		
Pituitary extract	5		
Others	4		
Postpartum atony		8	24.2
Retained placenta		3	9.1
Premature separation of placenta		2	6.1
Ectopic pregnancy		2	6.1
Others		3	9.1
Totals		33	100.1

curred during pregnancy and one on the day of delivery.

The leading cause of death for the second consecutive year and for the third of the four years of the study was hemorrhage and shock. It is quite apparent that it is here that the greatest opportunity for improvement in the mortality rate lies. Hemorrhage is far and away the greatest cause of preventable deaths (Table XVI). It accounts for more than three times as many of these as the next leading cause.

Delivery of patients by accouchement forcé and improper use of pituitary extract during labor are two well-recognized means of inviting trouble. These two accounted for one-third of the deaths due to hemorrhage in the past three years (Table XVII). There is no indication whatever for accouchement forcé. In no instance in which its application led to the patient's death could any justification for the procedure be found. Forceful dilatation of the cervix and forceps deliveries were often carried out in acute emergencies during labor in the unthinking and obviously false belief that delivery of the patients would accomplish something to ameliorate the situation. One patient developed premature separation of the placenta and severe shock during labor. Without any preliminary shock treatment she was anesthetized with chloroform, the cervical dilatation was completed manually and mid-forceps extraction was done. The patient expired from hemorrhage and shock in less than two hours. In another instance cervical dilatation from five centimeters was completed manually followed by a difficult forceps rotation and extraction because of "epilepsy." This patient died of hemorrhage approximately eight hours later. One patient with placenta previa at thirty-one weeks gestation, another with toxemia and a third with probable premature separation of the placenta were delivered by accouchement forcé. All died of hemorrhage. In still another instance a patient was making good progress after eighteen hours of labor when for no understandable reason the physician undertook to complete the dilatation manually and deliver the infant by means of a mid-forceps extraction. This patient died of hemorrhage thirteen hours later.

The abuse of pituitary extract during labor led to five deaths. Two of these resulted from severe lacerations of the birth canal from which patients bled to death while the other three developed frank ruptures of the uterus. In general, the indications for the use of the oxytocics was either inadequate or non-existent. The recognized rules relating to the safe methods of administering pituitary extract were violated in each instance. In the only case in which the pituitrin was given in dilute solution by continuous intravenous drip, the patient was a para 8. The danger of intramuscular use of pituitary extract in any quantity is indicated by the case of a patient who developed a precipitous labor and severe vaginal and cervical lacerations following administration of only two doses of pituitrin of 1 minim each. The patient died of hemorrhage.

Another large group of deaths due to hemorrhage is made up of those resulting from postpartum uterine atony. In this group there was, in general, failure to anticipate hemorrhage in instances of prolonged exhausting labor, difficult forceps deliveries, after deep anesthesia, in the presence of twins or hydramnics and at cesarean section. Treatment was inadequate. Probably the single most effective means for correcting or preventing postpartum hemorrhage due to atony is the administration by continuous intravenous drip of a dilute pitocin solution. This was not used in a single case in this group. The situation was often aggravated by the use of general anesthesia to examine the generative tract or to insert packing. Such anesthesia was not necessary. The utero-vaginal tract was usually inadequately exposed and explored, and packing, when it was used, generally simply obscured the true situation. Packs were used in eight instances in this series of deaths due to hemorrhage and in not one did it serve to arrest the hemorrhage. There was frequently failure to appreciate the true emergency nature of the situation and to secure adequate help.

One other fact stands out when deaths due to hemorrhage are considered. Blood is now so widely available that adequate amounts were on hand in all except two instances. However, in all except three instances the blood administered was given too late and/or in inadequate amounts.

Summary and Conclusions

1. A brief general summary of the results of the Minnesota Maternal Mortality Study for the period April 1, 1952, through March 31, 1953, has been presented. Certain comparisons are made between these results and those of similar studies done in three previous years in Minnesota.

2. There were fifty-eight deaths associated with pregnancy or the postpartum period in the 1952-1953 period among 78,990 live births for an over-all maternal mortality rate of 0.73 per 1,000 live births. The rate when deaths due to non-obstetric complications are excluded was 0.42. This is almost identical with the rate for the preceding year.

3. A most remarkable reduction in the proportion of deaths that were considered preventable took place in the past year. Only 17.2 per cent were considered preventable as compared with 42.1 per cent of the deaths in the preceding year and 73.2 per cent of those that occurred in 1941-1942.

4. There was a further increase in the percentage of deaths associated with pregnancy but due to non-obstetric causes from 17 per cent in 1941-1942 to 43.1 per cent in the most recent survey.

5. Approximately the same proportion of delivered patients who died were delivered by operative means in the most recent year of the study. However, only 15.8 per cent of the operative deliveries were considered not indicated as compared with 60 per cent in the preceding year.

6. The inaccuracies of ordinarily reported maternal mortality rates and of death certificates is discussed.

7. Complete pelvic measurements were obtained in 45.7 per cent of registered patients who died. Of those incompletely measured, external measurements including the interspinous, intercristal, intertrochanteric and Baudelocque's diameter were determined in most instances while the much more significant intertuberous and diagonal conjugate diameters were ignored.

8. The primary causes of maternal deaths in Minnesota in the 1952 study are tabulated. Hemorrhage was again the leading cause of death.

9. An analysis of the cause of "obstetric" deaths in 1950, 1951 and 1952 showed that hemorrhage accounted for 28 per cent of the total and almost one-half of the obstetric deaths which were considered preventable. Hemorrhage accounted for more than three times as many preventable deaths as the next leading cause, toxemia.

10. Some of the factors involved in the deaths due to hemorrhage are discussed. The principal ones included accouchement forcé, improper use of pituitary extracts during labor, and inadequate and delayed replacement of blood.

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Fractures of the Elbow

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OF THE MAJOR extremity joints, the elbow suffers the greatest from injuries to its surfaces. Since the functions of the upper extremity are largely those requiring dexterity, great demands are placed on this joint to provide a wide range of motion of its articulating parts. The articulation between the ulna and the humerus combines great stability with flexion and extension movement through a range of about 150 degrees. The articulation between the radius and the humerus combines this same range of flexion and extension movement with rotation of about 135 degrees at any point throughout the flexion and extension arc. The stability of the joint is further increased by an articulation between the head of the radius and the ulna.

Study of the anatomical relationships of these various articular surfaces reveals a precision of fit which cannot be disturbed more than a small amount if the motions of the elbow are not to be restricted. A thorough knowledge of the anatomy and functions of the elbow joint is a primary requirement for successful treatment of fractures of this joint. To this knowledge must be added the recognition of the soft tissue damage which occurs with fractures in this region. Particular care must be exercised during the recovery period to prevent this damage from leading to permanent disability. No joint in the body suffers more from overly zealous and injudicious manipulations during the recovery period.

The intelligent treatment of fractures of the elbow must be preceded by x-ray studies of good quality. The interpretation of the x-ray must be based upon exact anatomical knowledge of the bony and cartilaginous parts. The operator must have sufficiently good films and he must study those films carefully so that all displacements are visualized before treatment is begun. Certain of the fractures in this region occur primarily in youth while others are usually found in adult life. Some of the more common fractures of the elbow will be discussed according to the age group in which they are usually found.

Fractures Occurring Chiefly in Adults

Head of the Radius.—When falling on the outstretched hand, force is transmitted up the shaft of the radius and directed against the capitellum of the humerus. With an added valgus strain, the radial head may yield. In the presence of pain in the elbow with tenderness over the head of the radius and discomfort on pronation and supination movement, a fracture of the radial head must be suspected, and if routine x-ray studies do not reveal a fracture, further films made in varying degrees of pronation and supination should be made. Three degrees of fracture should be recognized:

1. A small marginal fracture with only mild displacement or a moderate sized marginal fracture with no displacement.
2. Marginal fractures with moderate to severe displacement.
3. Comminuted fractures of the head.

The first type of fracture can be treated with the use of a sling and early gentle exercise. Immobilization with a cast or a posterior plaster splint will give some comfort but should be discontinued within a week. Fractures of the second and third types usualy unite with deformity of the head and are accompanied by damage to the articular surface of the capitellum and should be treated by excision of the head. If excision is to be undertaken, it should be done within the first few days after the accident and should not be delayed until weeks or months later when it becomes obvious that the end result will be poor. Late excision of the head seldom improves the result. In the treatment of these fractures as well as all fractures of the elbow, passive stretching in any manner must be rigidly avoided since this

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delays recovery and frequently leads to unnecessary permanent loss of elbow movement.

Olecranon.—Fractures of the olecranon with separation require accurate reduction and immobilization until healing is complete. This can sometimes be accomplished by closed method, but the majority of such fractures are best treated by open operation and internal fixation employing wire suture or screws. When a screw is employed, it must engage the cortex of the distal fragment in order to obtain sufficient purchase in the bone. If wire is employed, it should pass through a drill hole in the distal fragment, but through the triceps tendon and immediately proximal to the olecranon fragment and not through the easily fragmented proximal fragment. Both a simple loop and a figure of eight loop should be employed, and the wire should be pulled very tight before twisting. The fixation should be so secure that no plaster is needed after the operation. If a plaster cast is applied, the patient awakening from anesthesia may struggle against the plaster and disrupt the reduction in spite of good technique. This will not take place where only elastic bandaging is used.

Comminuted fractures of the olecranon are usually not suited to either open or closed reduction. Excision of the comminuted fragments and suture of the tricepts tendon of the distal fragment leads to good functional result. Instability of the elbow does not result unless the elbow was dislocated anteriorly at the time of the fracture or unless the fragments of the olecranon constitute more than half of the semilunar notch.

Intercondylar Fractures.—"T" and "Y" fractures of the distal end of the humerus commonly display marked rotation of the condyles and separation of the capitellum and the trochlea. If not accurately reduced, proper fitting of the articular surfaces of the radius and ulna is lost leading to considerable disability. These fractures are usually best treated by closed method employing a judicious amount of skeletal traction through the olecranon and molding of the fragments by external manipulation. Open reduction may cause more abundant new bone formation which blocks joint movement and should, therefore, be employed only when necessary and then with a minimum of dissection. The posterior approach gives the best visualization and is the most convenient.

Capitellum.—The force of a fall transmitted upward through the radius may cause the hemispherical capitellum to shear off from the humerus and become widely displaced and free in the joint. This fragment can rarely be accurately replaced by manipulative method, although a single trial is usually justified. Open reduction permits accurate replacement. The pressure of the head of the radius then maintains reduction without the necessity for internal fixation.

Fractures Occurring Chiefly in Children

The proper diagnosis of fractures in children and the assessment of their severity depends upon a knowledge of the ossification centers which are present in the cartilaginous ends of the bones. Without a knowledge of the constant and the inconstant ossification centers about the elbow, serious fractures may be overlooked or a false diagnosis of fracture may be made in a normal elbow. Since many of the fractures of the lower end of the humerous chiefly involve the cartilage at the end of the bone, the actual fracture line may not be visible and the serious nature of the injury not recognized upon casual inspection of the x-ray film. Failure to accurately reduce such fractures commonly leads to greater deformity and physical handicap in adult life than the fracture of the shaft which is permitted to unite in marked angulation. In determining the injury to the elbow of a child, films of the opposite elbow made in the same direction may be exposed and the position and shape of the ossification centers compared.

Supracondylar Fractures.—In the usual type of supracondylar fracture, the distal fragment is displaced and rotated posteriorly. In the lateral x-ray of the elbow, a line drawn through the center of the epiphysis for the capitellum forms an angle of about 40 degrees with the longitudinal axis of the shaft. In this type of fracture, the angle is reduced and the epiphysis may be found to lie in the longitudinal axis or may be displaced posterior to it. Manipulative reduction of the fracture by applying traction and then flexion usually results in a very satisfactory replacement. Immobilization in a flexed position maintains reduction, but the combination of swelling and excessive flexion may lead to ischemia and disaster if not closely attended. If the normal forward tilt of the distal end of the humerus is not accurately restored, there will be loss of either flexion or extension movement to the same extent that this reduction fails to approximate the normal Failure to reduce any angulation seen in the anteroposterior x-ray will lead to the unsightly increase or decrease in the carrying angle of the arm and frequently to tardy ulnar palsy. Residual rotation of the fragments in the axis of the arm may lead to loss of ability to place the hand behind the head or behind the back.

In the more unusual type of supracondylar fracture, the distal fragment is angulated forward. Reduction is accomplished by fully extending the arm and immobilization should be in the same position.

Transcondylar Fractures.—These fractures pass transversely through the olecranon and coronoid fossae. Accurate reduction is essential to avoid bone block and subsequent loss of movement. Closed manipulation similar to that for supracondylar fractures is the method of choice.

Lateral Condyle Fractures.—This fracture line passes largely through cartilage in children and is not seen on the x-ray film. The fragment is commonly rotated up to 90° in the frontal plane and 90° about the longitudinal axis due to the pull of the wrist and finger extensor muscles which attach to the lateral epicondyle. In the presence of this displacement, the anteroposterior x-ray film displays an epiphysis for the capitellum which has the hemispherical shape normally seen in the lateral view. The lateral x-ray film may show the "half pear" shape which is normally seen in the anteroposterior view. When displacement is severe, open reduction is almost always needed. After anatomical replacement of the fragment under direct vision, a suture of catgut is usually sufficient to maintain reduction in the plaster cast. Failure to recognize and accurately replace this fragment results in progressive distortion of the elbow into a position of cubitus valgus and subsequent tardy ulnar palsy.

Medial Epicondyle.—Avulsion of the medial epicondyle may lead to only minimal displacement or to displacements of up to one centimeter. Such disruptions do not require open reduction but heal satisfactorily with fibrous union. When the elbow is forced into severe valgus and dislocated at the time of injury, the medial epicondyle may drop into the joint where it becomes an obstacle to reduction of the dislocation. Closed reduction may be accomplished at times by increasing the valgus of the arm while tensing the flexor muscle mass by extending and supinating the wrist and fingers. When this does not succeed in withdrawing the epicondyle from the joint, open operation is clearly indicated for replacement of the epicondyle and suture with catgut.

Upper Radial Epiphysis.—The head of the radius does not commonly fragment in children, but the neck of the bone more readily compresses, resulting in a greenstick fracture. At times the fracture may lead to severe tilting of the head. The head fragment with its epiphysis must never be excised in children because the loss of the growth center leads to shortening of the bone, loss of support of the radial side of the wrist and the deformity of a club hand. By placing the elbow under a varus strain and by pressing against the displaced head with the thumb, closed reduction is frequently successful. If manipulation does not succeed and the tilt of the epiphysis is more than of moderate degree, open reduction for accurate replacement is essential. Internal fixation is not necessary since reduction is maintained by pressure of the capitellum.

Summary

Fractures of the elbow must be reduced with special care in order to prevent permanent disability. Thorough knowledge of the anatomy and functions of the elbow joint is prerequisite to intelligent treatment. Accurate knowledge of the number, location, and shape of the ossification centers is essential in proper diagnosis and treatment of elbow injuries in children. Some fractures of the elbow must be treated by early open reduction. Passive stretching adversely effects recovery, but active exercise should be encouraged after immobilization is removed.

Anal Pruritus

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THE controversial subject of anal pruritus has flooded the literature with innumerable theories and thoughts as to the etiology and treatment of this condition. Many of these suggestions have received enthusiastic support initially, and then have gradually been abandoned. Some of the established etiologic factors will be reviewed, stressing the importance of the bacterial flora of the colon and diet as contributing factors in the production of anal pruritus. Recommendations for effective therapy will also be considered.

The anal and perianal skin are unusual in that very little stimulation is necessary to produce a perceptible irritation and the sensation of itching. This portion of the body is richly endowed with sensory nerve endings and physiologically displays a maximum readiness to itch. Longo[5] has shown that the only other region of the body which has as great a tendency to itch is the meatus of the external ear. In these two hyperirritable areas only minimal stimuli are necessary to produce itching and the consequent desire to scratch. Scratching, infection and prolonged irritation soon result in lichenification which greatly increases the state of hyperexcitability. This produces the vicious scratch-itch cycle which is very difficult to interrupt. This cycle must be broken, and the patient must refrain from scratching if any results are to be expected from therapy.

The gross pathology of the pruritic area is usually characteristic in appearance. Lichenification is usually present to some degree and the more chronic the process the thicker the skin. The normal radiating anal skin folds are usually found to be raised and edematous and often excoriated from scratching. The skin changes, however, depend upon the chronicity of the disease process, the various forms of medication and treatment that have been used, and the amount of scratching the patient has done.

Anal pruritus in the vast majority of cases is a *symptom complex* and ordinarily is not produced by any one sole factor. Usually a variety of etiologic factors are combined to produce this state of persistent perianal itching. This condition is a definite pathologic entity and should not be treated lightly. Patients with pruritus should not be dismissed with some ointment or suppository nor should they all be labeled as psychoneurotics as is so frequently done. All the possible etiological factors should be carefully studied in each case to make intelligent and effective therapy possible. First, it is very important to obtain an adequate history in these patients. This should include the duration of the disease process, the time of greatest severity of symptoms, as well as the relation to defecation, baths, menstrual cycles, and ingested food and drugs. Dietary habits and the presence or absence of frequent soft stools are very important. The patient should be asked about the presence of athlete's foot or other skin disease elsewhere on the body. It is also important to determine whether allergic manifestations have been present. Next, a detailed proctologic examination should be done which should include stool examinations for parasites and an estimation of the pH of the rectal secretions. A routine laboratory examination of the urine should be done in all cases to rule out diabetes.

1. *Common Ano-rectal Pathology.* — Local pathological conditions of the anus or rectum which allow leakage of rectal secretions onto the surrounding skin are important etiological factors in anal pruritus. Common conditions such as prolapsing internal hemorrhoids, redundant rectal mucosa, anal fissures and fistulae, hypertrophied anal papillae or fibrous polyps which protrude from the anus *all* allow mucus to

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escape onto the perianal skin. Frequently these secretions are very irritating. On purely a mechanical basis, however, even if the discharge was non-irritating, the constant presence of moisture between the buttocks produces a chafing and maceration of the skin. Usually the chief complaint of patients with protruding hemorrhoids and the like, will be in reference to such symptoms as bleeding and protrusion, but if questioned they usually volunteer the information that periodic episodes of itching occur.

All patients with a moist anus, however, do *not* have a true persistent anal pruritus and this is where the other etiological factors are concerned. This is also the reason why the surgical correction of all existing anal and rectal pathology in cases of pruritus ani will sometimes fail to cure the patient of the troublesome itching. External anal pathology such as skin tags and hypertrophied skin folds are also important since they make proper cleansing of the perianal skin very difficult. A factor which is very frequently overlooked in the production of a moist anus is redundancy of the rectal mucous membrane. This represents excessive looseness of the rectal mucosa in its attachment to the muscularis of the rectal ampulla. A certain amount of mobility of the mucous membrane is normally present, but through constipation, straining at stool, age and general wear and tear, the degree of slack between the layers of the rectal wall greatly increases. The excessive mobility and prolapsing of the rectal lining provokes an irritation and hyperemia of the mucous membrane in and above the internal hemorrhoidal area, which in turn effects excessive secretion of mucus. This mucus puddles above the intact sphincter and intermittently leaks through onto the perianal skin. Obvious anorectal pathology should be corrected surgically. One cannot expect a favorable response to therapy in patients with anal fistulae or pathology of an equally obvious nature which causes the skin to be constantly bathed in moist secretions.

The significance of a vaginal discharge is also frequently underestimated. Anal pruritus is often aggravated by vaginal secretions which run backward over the perineum and many of these patients will also have an associated vulvar pruritus. The leukorrhea causes the sensitive perianal skin and perineum to be kept constantly moist, and with chafing and maceration the skin becomes susceptible to secondary infection. The anal pruritus will not respond properly until the vaginal discharge has been eliminated.

2. *Chemical Irritants.*—Chemical irritation is probably the most important of the etiological factors involved in the majority of cases of anal pruritus and until recently its importance has been almost completely overlooked. Tucker and Hellwig[15] many years ago reported that the gross and microscopic changes in the skin in anal pruritus were characteristic of a chemical dermatitis. More recently some very important work has been done on the hypothesis of chemical irritation in pruritus which has given us a much more logical basis for the treatment of this condition. Slocumb,[9] in an exhaustive study of a series of cases of anal pruritus, found that the great majority of these patients had an increased pH of the rectal secretions, and he believed that the pruritus was due to seepage of this alkaline mucus onto the perianal skin. He also found that the increased alkalinity of the rectal contents was directly correlated with the flora of the colon. Through dietary measures he was able to alter the pH of the secretions of the rectum by altering the relative percentages of the Gram-positive and Gram-negative bacteria in the colon.

The second important study was done recently by Friedman, Haskell and Snape[2] who did microchemical studies on secretions obtained from the anal canal of patients with anal pruritus. They separated their cases into the "moist pruritus," or those in which the affected area was reddened, eroded, macerated and moist, and the "dry pruritus," characterized by marked hypertrophy and thickening of the skin folds. They were able to identify trypsin or trypsin-like enzymes in the rectal secretions of approximately 80 per cent of the "moist pruritus." In another group of patients with non-pruritic anorectal disease 20 per cent were found to have significant amounts of proteolytic enzymes in the rectal secretions. A similar figure (approximately 20 per cent) was obtained in the control group with no gastrointestinal pathology. These investigators believed that the proteolytic enzymes in the secretions leaking out onto the perianal skin was primarily responsible for the skin irritation. This group, however, did not indicate whether an elevated pH or increased alkalinity was found.

With these two studies one can come to some very definite conclusions as to the primary etiology in many cases of anal pruritus, and it is possible to treat the condition in a more intelligent and specific manner. Normally the pH of secretions obtained from the rectum vary from pH 6.5 to 7.5. In the majority of patients with anal pruritus the pH reading will be from 7.5 to 9.0. The prolonged seepage of alkaline secretions onto the perianal skin would in itself be an irritant, but the presence of the proteolytic enzymes is probably more important. Trypsin is most active in an alkaline medium with the maximum enzymatic effect being reached at pH 8.0 (Fig. 1) and this effect is maintained as the alkalinity increases. It will be noted that this is also the pH range of the rectal secretions in the majority of patients with anal pruritus. Trypsin is also mildly active in a neutral or mild acid medium but the activity rapidly and progressively decreases from a maximum at pH 8.0 to complete inactivity at pH 5.5.

Nitrazine paper is used for the purpose of determining pH readings. This is a rather rough method of estimating pH values, but it is accurate enough to definitely determine whether or not increased alkalinity is present.

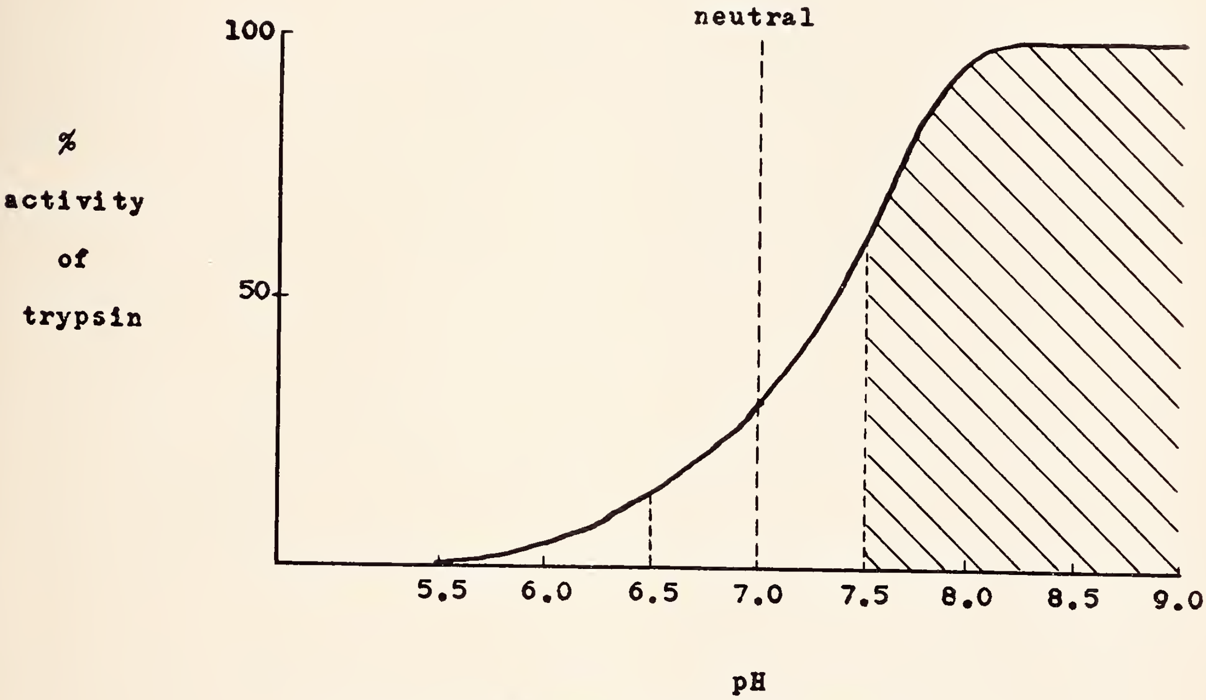

Fig. 1. From *The Journal of General Physiology.*[6]

The irritation of the skin in anal pruritus can be compared in a sense with the irritation of the skin of the abdominal wall following ileostomy or colostomy. High concentrations of proteolytic enzymes are present in the small bowel where they are active in protein digestion. Gradually neutralization of these enzymes occurs in the colon so that usually only minimal amounts can be detected when the fecal material reaches the rectum. In some individuals, for various reasons, inadequate neutralization of these enzymes occurs. We have all experienced a soreness and irritation of the anus following a bout of diarrhea. The increased peristalsis produces liquid stools which allows insufficient time for the neutralization of the proteolytic enzymes. When the diarrheal stool comes in contact with the external skin, the enzymes produce the irritation by a process of digestion. A comparison can also be made with the care necessary to protect the skin about an ileostomy stoma, transverse and sigmoid colostomies. Great care is required to prevent digestion of the skin from ileostomy secretions. Less care is necessary to prevent irritation about a transverse colostomy and with a sigmoid colostomy the skin usually requires little care. Progressive inactivation of the proteolytic enzymes as the fecal mass passes through the colon renders the stools less irritating. The irritation produced about artificial abdominal stomas, however, produces a burning sensation rather than persistent itching. A possible explanation for this may be Longo's study[5] mentioned previously, which showed the marked tendency of the perianal skin to itch. Many patients with anal pruritus will volunteer the information that the itching is severe following a bowel movement. The increased irritation following defecation is usually due to improper cleansing of the anus. Small amounts of stool containing minute quantities of proteolytic enzyme lodge between the radial perianal skin folds and irritate the skin until either the stool is removed or the action of the enzyme is spent. This is the reason why proper anal hygiene is so important in patients with pruritus ani.

Another recent factor in anal pruritus has been the generalized use of oral antibiotics. Aureomycin and terramycin seem to be the greatest offenders but persistent perianal itching is also seen following the use of others. The rectal secretions obtained from pruritic patients who have received these agents are uniformly quite alkaline in reaction (pH 8.0—9.0) and it may be assumed that proteolytic enzymes are also present. The high pH is due to a reduction in the number of the Gram-negative colon bacteria which normally form lactic acid in the intestinal tract by fermentation of carbohydrates.

Rettger[9] has likened the large intestine to "a veritable culture tube in which definite bacterial types appear to be constantly struggling to gain supremacy." As the colonic flora gradually returns following sterilization of the bowel by antibiotics, an overgrowth of Gram-positive bacteria may occur, which suppresses the growth of the normally predominant Gram-negative organisms. The alkaline contents of the small bowel is then no longer neutralized by the Gram-negative acid-producing colon bacteria, and the fecal mass passes through the large bowel maintaining a high alkaline pH. The inactivation of the proteolytic enzymes is likewise impeded. According to Zinsser's Bacteriology,[16] the Gram-negative colon bacteria normally constitute about 75 per cent of the intestinal flora, with the Gram-positive rods and cocci forming the remainder of the colonic organisms. Kendall,[4] Rettger,[9] and others have proven that definite changes may be brought about in the relative percentages of colon bacteria by purposefully adjusting the diet. Slocumb's[10] study routinely showed a predominance of Gram-positive instead of Gram-negative bacteria in his patients with elevated mucosal pH and anal pruritus. Through dietary measures he was able to reverse this ratio, and when the Gram-negative bacilli again becomes most numerous, the pH of the rectal secretions also was found to have been reduced to the normal range. With additional supportive measures the anal pruritus disappeared. Recurrences were reported and stool cultures again showed a predominance of Gram-positive instead of Gram-negative bacteria. Individual dietary habits over prolonged periods of time with a resultant change in the flora of the colon may well be one of the fundamental factors in the etiology of anal pruritus. Dietary habits are also the probable explanation for the fact that anal pruritus following oral anti-biotic therapy or otherwise, is rarely seen in children except for those with oxyuriasis infestation.

3. *Fungus Infections.* — Fungus infections play a definite role in anal pruritus, but they are important mainly as secondary invaders. Terrell and Shaw[15] reported an incidence as high as 60 per cent in cultures taken from the perianal area. Fungi can occasionally be cultured from normal skin but they usually do not grow and produce symptoms unless certain definite conditions are present for their growth. The perianal skin and the interdigital spaces between the toes offer ideal places for active growth of these organisms because of the moisture, heat and improper ventilation of the areas. Another factor which makes the perianal skin particularly vulnerable to fungus infections is the presence of the apocrine sweat glands, or scent glands. These glands produce a secretion having a higher pH, as well as a higher percentage of carbohydrate and protein than is found in ordinary perspiration.

Harry[3] and other investigators have stressed the important role played by pH in the defense mechanism of the skin. All the cells of the normal epidermis have an acid reaction (average pH 5.4) which serves as a protection to the skin. As Prinz and Ereaux[8] stated, "many pathogenic bacteria and fungi are very sensitive to acids, but grow abundantly in a slightly alkaline environment." In rubbing or scratching the perianal skin, if the epidermis is penetrated even to a small degree, weeping occurs and the adjacent skin immediately assumes the alkaline pH of blood plasma (pH 7.4). This again offers ideal conditions for the growth of pathogenic bacteria and fungi. The two most common types of fungi involved are the Monilia and Epidermophyton organisms. Occasionally cases of anal pruritus can be cleared by using fungicidal therapy alone but this is the exception rather than the rule. Usually additional therapeutic measures are necessary to give the patient lasting relief. Some clinical improvement can be expected, however, if the secondary fungus infection can be eradicated or reduced in severity.

4. *Allergy.*—Allergy is difficult to rule out as a causative factor in anal pruritus. If the patients do not respond to other measures, they are then studied from an allergic standpoint. Elimination diets and skin tests may be employed to attempt to identify the allergan and a therapeutic trial with antihistaminic drugs may be tried. With the introduction of the antihistaminics, allergy was strongly implicated as an etiologic factor and numerous enthusiastic reports were published with very high curative rates. For a period of time these drugs were administered routinely to all patients with pruritus ani, but the results which others had reported could not be duplicated. The use of antihistaminic drugs is now restricted to those cases in which a definite allergic element is present or seems to be present or in those cases where other means fail. Allergy has been definitely established as an etiologic factor in pruritus ani but the number of cases is small.

5. *Intestinal Parasites.*—These are not major factors in anal pruritus in adults, but pinworm infestation should be considered especially in the night scratchers. Abel[1] has stated that pinworms are the usual cause of anal pruritus in children. Tests to determine the presence of pinworm eggs on the perianal skin should be a routine procedure in adults as well as children, since positive findings are common enough to justify the consideration of their presence. If the pinworm ova are found, the patient should be given adequate treatment with oral gentian violet. The uncomplicated cases will clear up immediately. In adults, however, if oxyuriasis is found in a full blown case of anal pruritus, the presence of the parasites is probably only contributory and additional methods of treatment will be necessary.

6. *Mechanical Factors.*—The anal region is constantly being exposed to trauma of one type or another. The passage of hard stools, the use of rough toilet tissue, improperly fitting underclothing which traumatize the area on walking, etc., are all factors. The wearing of a perineal pad during the menses will often irritate the perianal skin and usually is very aggravating to an existing anal pruritus. This group can be readily identified and is but one of the several factors usually involved in producing this condition.

7. *Psychogenic Factors.*—Stokes[11] and others maintain that there are always tension factors in the background of chronic anal pruritus, "the itching being the focal manifestation of a neurotically predisposed skin." They maintain that the patient sets up a vicious cycle of projection of his anxieties upon the perineum. Some authors associated with large clinic groups have considered 90 per cent of the cases of anal pruritus to be a neurodermatitis. In the last edition of Ormsby,[7] "Diseases of the Skin," anal pruritus is discussed under the neuroses. This is a grave injustice to the majority of patients suffering from this condition. Although it is true that patients with chronic pruritus ani are frequently very nervous, it is often found that as soon as the itching has been alleviated, the degree of nervousness is greatly reduced. While in most cases, nervousness and tension may be contributory in anal pruritus, they do not form the underlying basis for the persistent anal itching. Nervous tension and chronic anxiety may serve to amplify and perpetuate otherwise brief periods of itching by keeping the patient unusually alert or attentive to sensations from the perianal region. Trauma from scratching and the unusual awareness of the condition may start the vicious scratch-itch cycle. Once this cycle has been established therapy is usually ineffective unless the patient can stop scratching the area.

8. *Constitutional Disease.*—Diabetes is probably the most common constitutional disease with secondary anal pruritus, although other constitutional diseases with skin manifestations may involve the perianal area. Patients with eczema

and psoriasis should probably also be included in this category. If the diseased perianal skin is only a part of a dermatitis involving other portions of the body, it is fruitless to attempt to treat only the pruritus ani *per se*.

Treatment

1. *General Considerations.* — Patients with anal pruritus should be reassured and told the nature of their trouble at the first consultation. The type of treatment to be used should be carefully explained so that the patient realizes the value of the instructions that are given him. The futility of scratching the area must be stressed, since any additional rubbing or scratching only serves to aggravate the scratch-itch cycle and makes the condition worse. Underwear and trousers should be worn loosely to avoid any additional stimulation. Woolen underwear is especially bad as a mechanical irritant in these people. Women should be instructed to discard girdles and corsets which bind the buttocks tightly together. These foundation garments aggravate the condition by allowing improper ventilation between the buttocks and produce chafing and irritation.

2. *Anal Hygiene.*—The patient must definitely alter the manner used to cleanse the anus after bowel movements. No harsh toilet tissues are allowed since nothing will start the scratch-itch cycle as readily as vigorous rubbing of the anus. The patient is instructed to use a wad of toilet tissue or cotton soaked in water to cleanse the area. The anus should be thoroughly washed with the wet pledgets to remove all traces of fecal matter and then dried by blotting with dry tissues.

No soap should be used about the anus, since it is a definite irritant if not completely removed. The alkaline soap also removes the protective oily film and creates an alkaline medium favorable to the growth of secondary invaders. Many patients are instructed to apply vaseline to the skin about the anus before bathing to protect the area from soapy water.

3. *Diet.*—Adherence to a strict diet is most important, at least during the early phase of treatment. A modified acid-ash diet should be followed since in leaving an acid residue it will reduce the excessive alkalinity of the large bowel. This diet should include those foods which produce lactic acid on fermentation in the intestinal tract. Especially important are dairy products such as cultured buttermilk, yoghurt, cottage cheese and other cheeses. Cultured buttermilk should be consumed in as large quantities as possible. It is sometimes desirable to add beta-lactose to the buttermilk because of its ready fermentation to lactic acid. Bread, cereals of all kinds and spaghetti are other carbohydrates which may be used. Of the fruits, only prunes, plums and cranberries are considered to have an acid residue because of the formation of benzoic acid. Although meat and eggs are considered to be acid-ash foods, Kendall[4] has shown that the excessive consumption of proteins will promote the growth of the putrefactive flora of the bowel and tends to inhibit the growth of the desirable Gram-negative colon organisms. For this reason a moderate reduction in the consumption of protein is required.

The addition of viable Lactobacillus acidophilus organisms to the diet, in one form or another, is also very beneficial. This is done routinely in the postantibiotic cases of pruritus and proctitis. Many forms of these cultures are available, but recently Lactinex tablets have been used with success. These compressed tablets, composed of a combination of viable L. acidophilus and L. bulgaricus organisms, are consumed with whole milk. Rettger[9] as shown that the simultaneous administration of lactose and L. acidophilus bacteria will replace the putrefactive flora of the colon with an aciduric flora. Lactobacillus acidophilus organisms will also establish residence in the colon. It is not possible to secure implantation of Lactobacillus bulgaricus in the bowel, which is the organism most commonly used in the production of cultured buttermilk. Although the action of L. bulgaricus is transient, with prolonged use it also enhances the growth of the desirable Gram-negative bacteria by also forming lactic acid on fermentation of carbohydrates.

Foods which produce an alkaline residue should be restricted. These include fruits and leafy vegetables in general, with the exception of the fruits mentioned above. Citrus fruits are very common offenders. They greatly increase the alkalinity of the stool and even the moderate use of orange juice and grapefruit juice may aggravate an anal pruritus. Peppers and heavily spiced foods should be omitted because of the irritant effect on the digestive tract. Alcohol is another offender, and beer should be completely restricted if there is a tendency toward the production of soft stools. This dietary regime is starchy and not too tasty but the patients seem to tolerate it quite well.

4. *Rectal Irrigations.*—Slocumb[10] advocated the use of a mild lactic acid solution for rectal irrigations. He found lactic acid to be more effective than dilute hydrocholoric or boric acid in maintaining a pH of about 6.5 in the rectal ampulla. The purpose of this irrigation is twofold. First, it reduces the alkalinity of the rectal ampulla by neutralizing the alkaline secretions and, secondly, it cleanses the rectum of any stool and mucus which may be present. It will be recalled that Friedman et al[2] found proteolytic enzymes in the rectal secretions of a large percentage of patients with anal pruritus. The washing away of these enzyme containing secretions is probably the greatest function of the irrigations. Slocumb[4] recommended that this irrigation be used once daily.

A modification of this technique has been found to be more effective in some cases. One pint of a .15 to .20 per cent lactic acid solution is used as a rectal douche. This solution can be easily prepared by adding one teaspoonful of a 20 per cent lactic acid solution to a pint of tap water. The frequency of the irrigations or low enemas depends upon the acuteness of the individual case. In patients with a moist, red and eroded perianal skin it has been found advantageous to use the irrigation three or four times daily until the acute symptoms have begun to subside. Local applications and supportive treatment must, of course, be used along with the cleansing acid enemas. The average chronic case of anal pruritus is instructed to irrigate himself twice daily at the onset of therapy. After a period of time when the itching subsides and the skin has begun to approach the normal, the irrigations may be reduced to once daily, once every second day, and then discontinued if the patient will adhere to the acid-ash diet. If dietary indiscretions cause a recurrence of the itching, the patients are told to resume the irrigations until the symptoms again subside. These lactic acid irrigations are not a "cure-all" for anal pruritus, but it has been found to be one of the most effective methods of therapy at our disposal, giving more benefit to a greater number of patients than any other single method of treatment..

5. *Local Applications.*—In the very acute types of pruritus with the typical red, weeping skin nothing is more beneficial or gratifying than the use of cold moist packs. Usually a 1:10 Burow's solution or 1:4000 potassium permanganate is used. The potassium permanganate has the advantage of also being effective against the secondary fungus infection which may be present. In the acute type of case the application of ointments usually only serves to aggravate the condition. A mechanism which will quickly bring relief from severe itching is the application of an ordinary ice collar to the perineum, holding it in place with a belt or T-binder. This may be used in conjunction with the cold moist packs. The maintenance of subnormal temperatures anesthetizes the nerve endings and very soon stops the itching. This is a very useful procedure which should be explained to the patients.

The majority of the cases of anal pruritus which are seen have been grossly overtreated. In an effort to stop the itching the patient has usually tried numerous home remedies and proprietary medicines. The majority of medications sold across the counter in drug stores contain symptomatic antipruritics in excessively high concentrations. The effectiveness of these preparations in controlling the itching soon wears off and a secondary irritation is produced which eventually makes the condition worse. In general it may be stated that the mildest medications which will give the patient relief will ultimately give the best results.

New preparations are constantly being introduced for the treatment of anal pruritus; however, some of the old time-worn remedies which have generally been discarded have not lost their excellent qualities. Unna's Soft Zinc Paste (pasta zinci mollis U.S.P.) has been found to be one of the most effective preparations available, forming an excellent water-repellent protective application. Because of the mildness of this paste it may be used indefinitely to protect the perianal skin.

Fungicidal therapy is indicated if the pruritic skin appears to be complicated by a fungus infection. As stated previously, most mycotic infections in anal pruritus are secondary and additional therapeutic measures are necessary, but improvement can be expected if the fungus infection can be eradicated or reduced in severity. Mild preparations of gentian violet, malachite green, Castellani's paint, and other dyes have been used, and all are quite satisfactory for local application. The fatty-acid fungicidal preparations (Desenex, Sopranol, etc.) have also been used. The commercially available preparations of these fungicides, however, are far too strong for use on the sensitive perianal skin. They should be diluted to one-half or one-third their initial strength with an appropriate ointment base. Tar preparations are often quite valuable in the treatment of chronic cases with lichenification of the skin. They are usually used in strengths of 2 to 5 per cent. The tar ointments are generally effective in reducing the chronic inflammation and thickness of the skin. Ointments containing local anesthetics are unnecessary in the treatment of anal pruritus. The itching can be stopped with other means and the eczematoid dermatitis often produced by sensitivity reactions to the anesthetic agents can be avoided.

Recently hydrocortisone preparations have been widely used in the treatment of many dermatoses including anal pruritus. In many cases it promptly reduces the inflammation and edema in the involved skin and stops the itching. Local hydrocortisone therapy, however, is no panacea or one-shot treatment for all cases of chronic anal pruritus. Several cases have been seen in which the application of hydrocortisone has made the condition worse. Sulzberger[13] has reported similar experiences. It is fruitless to expect hydrocortisone ointment, or any other ointment, to be effective in the acute, red, weeping types of anal pruritus. Conventional methods must first be used to relieve the acute inflammatory reaction before local hydrocortisone therapy is attempted. The 2.5 per cent hydrocortisone ointment is much more effective in the treatment of anal pruritus than is the 1.0 per cent preparations. A very small amount of the 2.5 per cent ointment should be applied to the involved area two or three times daily and thoroughly massaged into the skin. In cases where a favorable response occurs, improvement is usually evident within two to three days after treatment is begun. In some cases the itching will stop within a few hours. If no improvement is noted within seven days, the hydrocortisone therapy should be discontinued since no further beneficial effect can be expected. After the pruritus has subsided, the 1.0 per cent ointment is usually adequate to keep the itching under control, but its daily use is often necessary to prevent recurrences. In the presence of scattered excoriations in chronic cases the addition of Neomycin to the hydrocortisone seems to be helpful in eradicating the secondary infection and promoting more rapid healing of the eroded areas.

Sulzberger[13] has stated that the therapeutic action of locally applied hydrocortisone is based on a local hormonal action of the steroid itself on the skin. The local effectiveness is also obtained without undesirable systemic effects and no contraindications were found to its continued use on small body areas in a large series of cases.

Hydrocortisone is a valuable adjunct to the treatment of anal pruritus but its use should be combined with dietary changes and other therapeutic measures at our disposal to produce lasting cures. Treating the symptoms rather than the disease as a whole cannot be expected to produce satisfactory long-lasting results.

6. *X-ray Therapy.*—Local x-ray therapy has been used extensively in the treatment of pruritus ani. It is most effective in the chronic cases with lichenification, and it acts by decreasing the hyperexcitability of the pruritic skin and helps to break the scratch-itch cycle. Many patients will respond favorably to this type of therapy and then become refractory to it. If the x-ray treatments are continued too long a radiodermatitis will ultimately develop. For this reason the indiscriminate use of x-ray should be eliminated. Before resorting to x-ray every effort should be made to control the condition with other means. There are too many patients suffering from both the pruritus and a radiodermatitis. When this unfortunate combination is found, the usual type of therapy is ineffective and radical procedures such as a subcutaneous perianal neurotomy must be used to give the patient relief.

7. *Surgical Methods.*—The injection of long-lasting local anesthetics beneath the perianal skin is used occasionally to break the scratch-itch cycle when the patient has insufficient will power to keep from scratching. This will stop the itching for a sufficient period of time so that adequate general and local therapy can be effec-

tively started. Usually an aqueous preparation of Diothane and Benzyl alcohol is used. Fewer complications in the form of slough and abscesses can be expected with this preparation than with the long lasting local anesthetics in oil.

If redundancy of the mucous membrane exists, it may be corrected as an office procedure with the injection of sclerosing solutions, or surgically at the time of removal of other coexisting anorectal pathology. Sclerosing solutions injected into the submucosa promote fibrosis and fixation of the mucosa to the muscularis. Usually five per cent phenol in olive oil is used for this purpose although other sclerosing solutions are also effective. If the injections are carefully given and intramucosal injections are avoided, sloughs and other complications are quite rare.

Radical operative procedures for anal pruritus, such as Ball's subcutaneous perianal neurotomy, have been abandoned. However, when surgical procedures are done on pruritic patients for the removal of obvious anorectal pathology, the perianal skin is routinely undercut at the same time. Severing the perianal sensory nerve fibers at this time is a relatively simple procedure, and it does not prolong the healing period. If the undercutting is thoroughly done, the patient will be completely relieved of any itching until sufficient time has elapsed for the nerve fibers to grow back. This usually requires several months during which time adequate measures can be taken to treat the skin and otherwise restore normal physiology to the area.

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HEARING LOSS IN CHILDREN

(Continued from Page 8)

Day schools for the hard of hearing are maintained in Minneapolis, St. Paul and Duluth.

Speech clinics are available at the University of Minnesota, the University of Minnesota at Duluth, St. Cloud State Teachers College, and Moorhead State Teachers College.

The Minnesota School for the Deaf at Faribault is a state-maintained boarding school for hearing-handicapped children. Its enrollment is limited.

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Carcinoma of the Common Duct

Report of Two Cases

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ACCORDING to Renshaw, Durand Fardell first described carcinoma of the common duct in 1840. Since that time numerous authors have reported small series of cases. Kirshbaum and Kozell[2] reported thirty-two cases in 13,330 consecutive autopsies at the Cook County Hospital. Niebling[4] found sixty cases in 14,000 operations on the biliary tract and 12,000 autopsies at the Mayo Clinic. McGlinn[3] found five in 9,000 routine post-mortem examinations and Glen and Davis[1] report three from the New York Hospital from 1932 to 1951. From these series it is readily seen that the disease is relatively infrequent even in the larger medical centers. For this reason it seemed to me that two cases occurring within three months' time in a small community hospital would be of interest to the practicing physician. Of further interest is the fact that only 345 operations on the biliary tract were performed during the years 1949 through August 1954. No other cases were found during this period.

The sex distribution of carcinoma of the common duct is found to be more equal than in carcinoma of the gall bladder with most authors reporting a slight preponderance on the male side. Also, in contrast to gall bladder carcinoma, biliary calculi are found to be present less frequently, but they do occur in about fifty per cent of the cases. This is still a rather high incidence and for this reason many authors suggest a possible etiological relationship between biliary calculi and carcinoma of the bile ducts. Other workers, notably Boyd, Barlow, and Dick[4] have pointed out the similarity of normal bile constituents to known carcinogenic agents.

The clinical manifestations of carcinoma of the common duct are indistinguishable from any other obstructive type of jaundice. The most common manifestation is a progressive jaundice usually without pain although pain does occur with surprising frequency. The jaundice is accompanied by acholic stools, anorexia, weight loss, and pruritus. In one of our two cases the patient had no complaints other than jaundice and pruritus. In various series a palpable gall bladder was present in only thirty per cent or less of the cases. The carcinomas are predominately adenocarcinomas with the infiltrating type most prevalent in the common duct. Microscopically three general types are noted: (1) scirrhous, (2) mucous, and (3) a papillary type. Metastasis occurs early both regionally and at a distance and recurrence following surgery is common.

The early diagnosis of these tumors is difficult and seldom made. In fact, the exact preoperative diagnosis is usually obscure and it remains for laparotomy to disclose the type of obstruction. Consequently many of these tumors are discovered unexpectedly or in a late phase when regional and distant spread has occurred beyond any possible hope of surgical cure.

Palliative surgery to relieve the jaundice and its complications and pain has not been encouraging.

Local extirpation of the lesion followed by reconstruction of the bile ducts has been tried with varying success. It is distressing to report that both of these cases died, one surviving her surgery for a period of two months. It is to be hoped that with the further development of radical surgical techniques and of the biochemical anti-carcinogenic armamentarium more long-term survivals will be effected.

With these brief remarks on the disease itself, I should like to present two cases of carcinoma of the common duct occurring at the Winona General Hospital within a three-month period. Both cases presented the classical picture of jaundice and in neither case was the diagnosis made preoperatively.

Case 1.—A seventy-two-year-old white woman was first seen November 23, 1953, complaining of jaundice.

Awarded Southern Minnesota Medical Association Medal for best case report submitted at the annual meeting, Winona, Minnesota, September 13, 1954.

She gave a history of weight loss, anorexia, and tiredness, of one month's duration. Her husband had noticed the yellow discoloration of her skin. She gave no history of nausea, vomiting, or pain. Her general health was always good and she gave no history of having had any type of infection within the past year.

Physical examination revealed a well-developed, well-preserved white woman who was markedly jaundiced. Examination was essentially negative except for the abdomen. There was spasm of the entire right rectus muscle with the sensation of fullness in the right upper quadrant of the abdomen. No definite mass could be outlined. Pelvic and rectal examinations were essentially negative.

Laboratory examination at the time of the admission to the hospital revealed the hemoglobin to be 12.5 grams, red blood count 3,800,000, white blood count 7,200 with 76 per cent polymorphonuclears and 24 per cent lymphocytes. The plasma proteins were 6.05 grams, thymol turbidity 3.9 units, and the prothrombin time 17.2 seconds with a control at 12.2 seconds. The cephalin flocculation test was negative in twenty-four hours and one plus in forty-eight hours. The total serum bilirubin was 12.6 mgm. per cent and the urine urobilinogen was negative in a one to ten dilution. The stool was negative for occult blood.

X-ray examination of the chest revealed no evidence of intrathoracic pathology. Examination of the upper gastro-intestinal tract by means of a barium meal showed a small ulcer in the first portion of the duodenum and distortion of the duodenum by an extrinsic mass. A diagnosis of obstructive jaundice most probably malignant in origin was made and the patient was prepared for surgery.

The patient was operated upon on December 7, 1953. At laparotomy, the gall bladder was found to be markedly distended. The upper portion was thin-walled whereas the lower half was thick-walled and firmly attached to a mass located at the junction of the cystic and hepatic ducts. The hepatic ducts proximal to the mass were markedly dilated. The intrapancreatic portion of the common duct was of normal caliber. Biopsy of the liver near the gall bladder revealed adenocarcinoma. Biopsy of the mass surrounding the common duct also revealed adenocarcinoma. The gall bladder, cystic duct and the mass involving the first portion of the common duct were excised *in toto* and the continuity of the common duct was re-established by primary anastomosis over a T tube.

The patient's postoperative course was quite stormy but she gradually showed a decrease in the amount of jaundice and a slight increase in her appetite. She was discharged with the T tube in place. Her course at home was gradually down hill although she was able to be up and around part of the day and to eat three meals a day. She died on February 19, 1954, following an episode of coffee ground emesis. Her jaundice had cleared at the time of her death. A post-mortem examination was not obtained.

The pathologist's report of the tissue removed at surgery revealed adenocarcinoma of the common duct with regional infiltration into the liver and gall bladder.

Case 2.—This patient was operated upon by Dr. Roger Hartwich of Winona who very kindly provided the clinical data for this paper. The patient was a sixty-two-year-old white man who was first seen on January 2, 1954 complaining of jaundice and anorexia. He had been well until December 29, 1953, when he had complained of nausea. He had had no vomiting, no abdominal pain, no chills or fever. About one week later he noticed dark urine, light colored stools, and weakness. His past history was essentially negative and there was no history of any *injection* in the past year. He continued to work as a shipping clerk feeling fairly well on a high protein low fat diet. Because of the persistent jaundice and pruritus he was admitted to the hospital on January 28, 1954, for exploration.

Physical examination on admission revealed a well-developed, well-nourished white man who was obviously jaundiced and complained only of severe generalized itching. Entire physical examination resulted in negative findings save for the marked jaundice and evidence of pruritus.

Laboratory examination revealed a hemoglobin to be 78 per cent, the white blood count 6,000 with a normal differential. The prothrombin time was eighteen seconds with a control at fourteen seconds, alkaline phosphatase was 10 Bodansky units, cephalin flocculation one plus in seventy-two hours, and the total serum bilirubin 11 mgm. per cent. A twenty-four-hour stool specimen showed no fecal urobilinogen and the urine was positive for bile. The total protein was 7.4 grams with a normal A/G ratio.

This patient was operated upon February 4, 1954, at which time a mass involving the common duct and cystic duct was found. The tumor mass extended from the junction of the cystic and common hepatic ducts along the entire length of the common duct to the Ampulla of Vater where it stopped. The duct system proximal to the mass was markedly dilated. A biopsy of the tumor was taken which on frozen section proved to be adenocarcinoma. The duct was incised and drainage was established by means of a T tube.

The patient did well following his operation but expired suddenly on his fourth postoperative day. Postmortem examination revealed a large pulmonary embolus. Examination of the common duct and pancreas at the time of post-mortem examination revealed the common duct completely surrounded by tumor from the Ampulla of Vater approximately to its junction with the cystic duct. The pathologist's final diagnosis was adenocarcinoma of the common duct.

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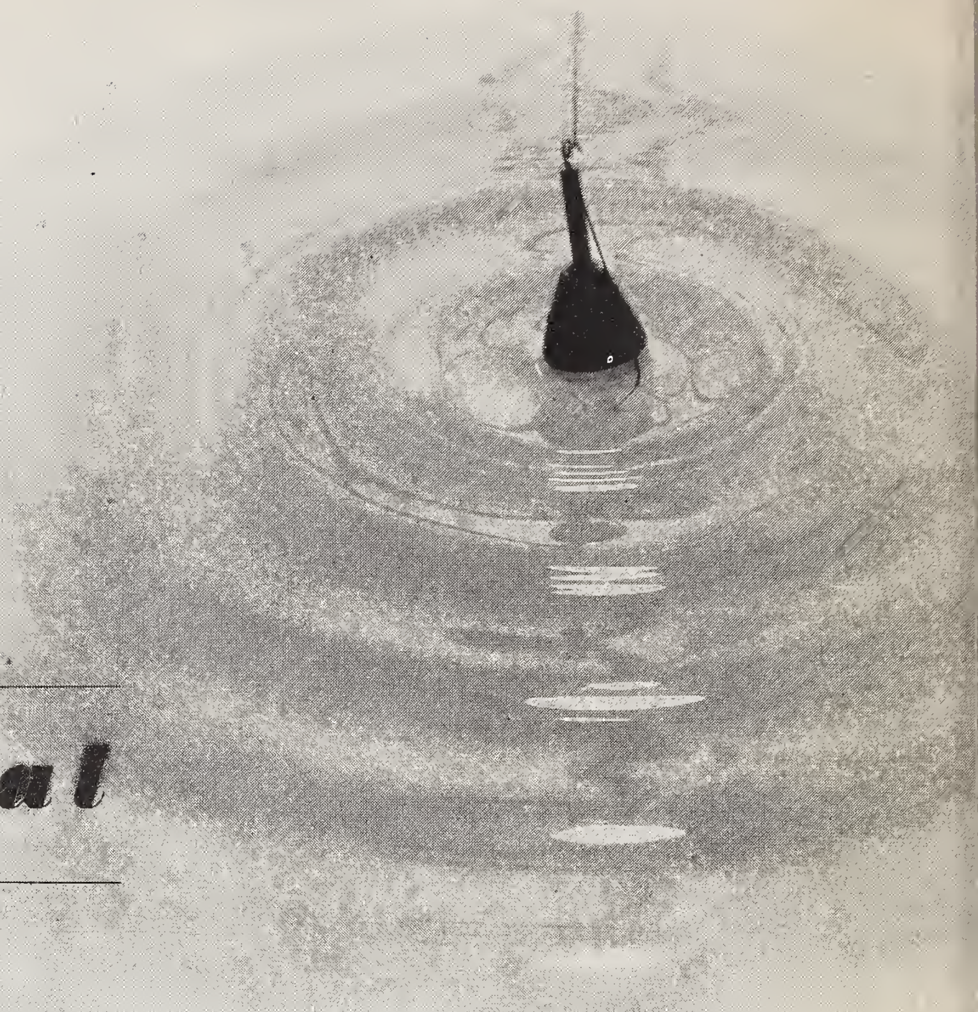

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Retroperitoneal Cavernous Hemangioma Associated with Hemangiomas of the Skin in a Newborn

Case Report and Brief Review of Literature

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DONALD H. PETERSON, M.D.
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HEMANGIOMAS ARE the most common benign tumors of the newborn. They occur most frequently on the skin of the face and the lower extremities. The lesions are often multiple. Rambar[16] reported the occurrence of hemangiomas of the skin in 11.6 per cent of a large group of premature infants. The incidence is believed to be smaller in full term newborns. According to Fitzwilliams[6] about 16 per cent of individuals with hemangiomas of the skin have multiple lesions. Sometimes, hemangiomas can occur in two different tissues of the same individual. The simultaneous presence of hemangiomas of the liver and spleen or skin and brain has been repeatedly recorded in literature. It is these combined lesions that have given rise to many eponyms and non-descriptive diseases such as Osler-Rendu, Sturge-Webber, and Lindau's etc. Numerous reports of these combinations of hemangiomas have been recorded.

A rare site for these congenital lesions is the kidney. Up to 1951, only seventy-three cases of hemangiomas of the kidney had been reported (McCrea[11]). Still rarer in occurrence is the presence of hemangiomas in the perirenal retroperitoneal spaces. Only three such cases have appeared in the medical literature up to the present time. The extreme rarity of a retroperitoneal hemangioma is such that no mention of it appears in the standard texts and reference books on pathology. Caffey[1] fails to mention it in the differential diagnosis of abdominal tumors of children. Similarly, Willis[25] and Ewing,[5] in their comprehensive treatises on the neoplastic diseases of man take no notice of the possible occurrence of hemangiomas in the retroperitoneal spaces. For this reason, the following case is recorded, and a brief review of the literature on this subject is attempted.

Case Report

Baby D, white male, was born at 3:58 P.M. on October 3, 1953. Delivery of the head and shoulders was accomplished without difficulty, but there was some delay in releasing the hips.

Physical Examination at birth revealed the following positive findings:

1. The abdomen was distended by a large oval mass which could be palpated in the right lower quadrant and flank. The mass appeared to extend down into the right pelvis.
2. Bilateral hydroceles of both testes.
3. Two dark strawberry colored tumors, each measuring four centimeters in diameter, were seen, one on the upper lateral aspect of the right thigh, and the other on the skin over the right patella. A firm subcutaneous mass measuring two centimeters in diameter was also found in the right lateral surface of the thigh.

Family History.—Non-contributory.

Laboratory Data.—Examination of the blood revealed a positive Coomb's test, a hemoglobin of 10 Gm. with an erythrocyte count of two million. Many normoblasts in ratio of 8 per cent of the erythrocytes were present. Transfusion via the umbilical cord was carried out. Four hundred and eighty-five cc. of blood was withdrawn, and 556 cc. of whole blood was injected along with 5 cc. of calcium gluconate. The excess of blood was given because of the low initial erythrocyte count. This procedure was well tolerated.

Fig. 1. *(upper left):* Scout film of the abdomen. Note the large soft tissue mass in the right lower quadrant pushing the gas filled loops of intestine to the left.

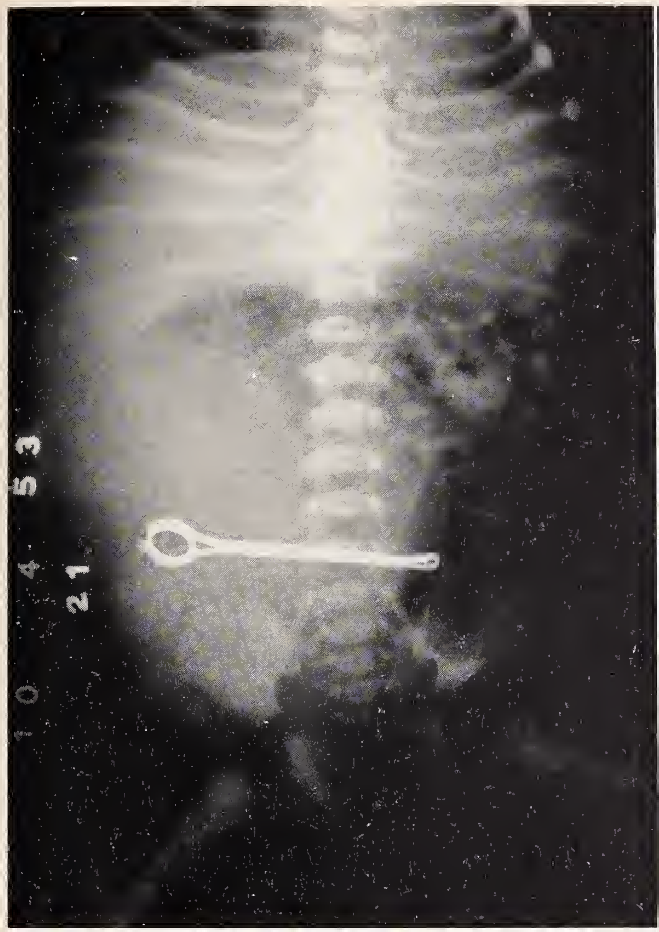
Fig. 2. *(upper right):* Spot roentgenogram obtained during examination of the colon by means of barium enema. Forward and medial displacement of ascending colon is demonstrated. Note also the dye-filled pelves and calyces of both kidneys.

Fig. 3. *(lower left):* Photograph of the microscopic section of the tumor. Note the cavernous blood spaces. Similar picture was seen in the section of the nevi of the skin on postmortum examination.

Fig. 4. *(lower right):* Arrows point to minute calcific densities in the nevus over the right patella.

From the Department of Radiology, Ancker Hospital, Saint Paul, Minnesota.

Dr. Aurelius is Chief of the Department of Radiology, Dr. Peterson is Associate Radiologist, and Dr. Niknejad is University of Minnesota Fellow in Radiology, at Ancker Hospital, Saint Paul, Minnesota.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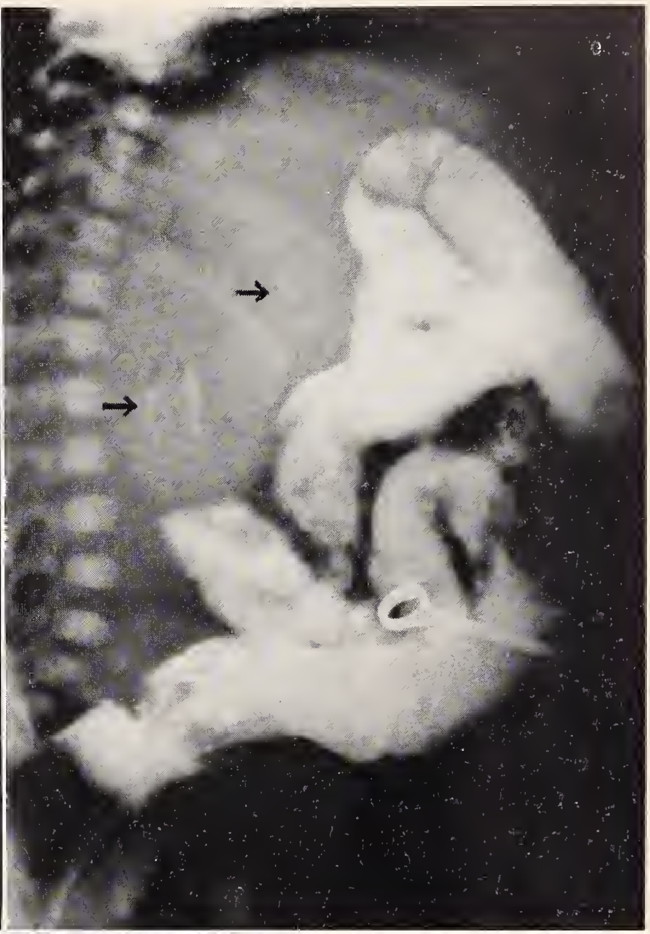

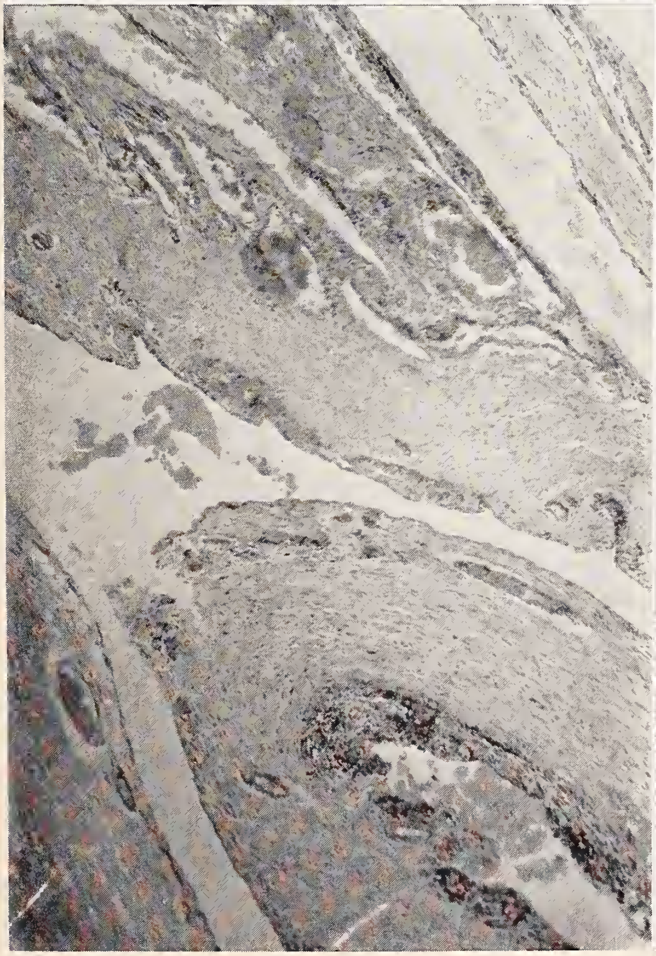

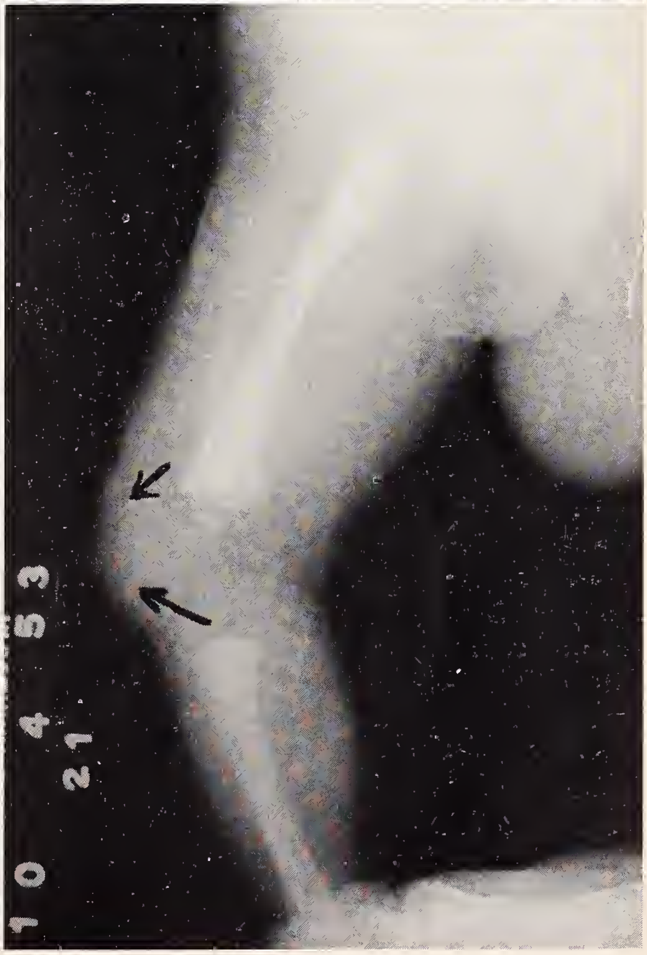
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Roentgenological Examination.—The initial scout film of the abdomen (Fig. 1) revealed a soft tissue mass in the right lower quadrant. The mass appeared to displace the normal gas containing loops of large and small intestines to the left. There was no radiological evidence of intestinal obstruction. A roentgenogram of the right leg showed soft tissue masses which were noted on physical examination. In addition, definite calcification was noted in the tumor over the right patella. Examination of the skull disclosed a normal calvarium with no evidence of metastasis.

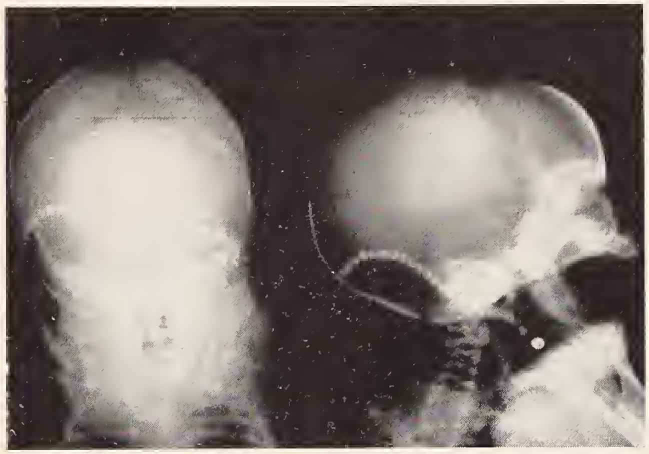

Fig. 5. Antero posterior and lateral roentgenograms of the skull show no evidence of metastasis.

In view of the presence of multiple hemangiomas of the right leg, it was postulated that the mass in the right lower quadrant could also represent a lesion of similar nature. Study of the genito-urinary tract, and examination of the colon for better evaluation of the abdominal mass were suggested. These were carried out on the following day. About 5 cc. of the urographic contrast medium was injected into the subcutaneous tissues of both buttocks. Roentgenograms obtained subsequently showed a normal urogram on the left. No contrast medium could be elicited in the right kidney. At this time, on the insistence of the surgical staff who were anxious to do an exploratory laporatomy as soon as possible, further follow-up urographic studies had to be abandoned. However, films taken during the examination of the colon, which was performed subsequently, fortuitously revealed normal urograms bilaterally (Fig. 2). It was then assumed that the retroperitoneal mass in the right lower quadrant was probably separate from the right kidney, or at least did not involve its collecting system. Thus, the previous impression with regard to the nature of the lesion was confirmed. Incidentally, the barium enema showed medial displacement of the ascending colon.

Findings at Laporatomy.—A mass of tissue measuring 9 x 7 x 2 cm. was found in the right retroperitoneal space which surrounded the anterior aspect of the right kidney, but did not invade it. A line of cleavage could easily be made between the tumor and the right kidney. The inferior portion of the mass extended to the right pelvis.

Surgical Pathology Report.—The specimen consisted of a mass of tissue measuring 9 by 7 centimeters in its broadest dimensions and 2 cm. in thickness. On section, the tissue was composed of tortuous dilated venous spaces filled with clotted blood. The largest of these spaces measured 3 cm. in diameter.

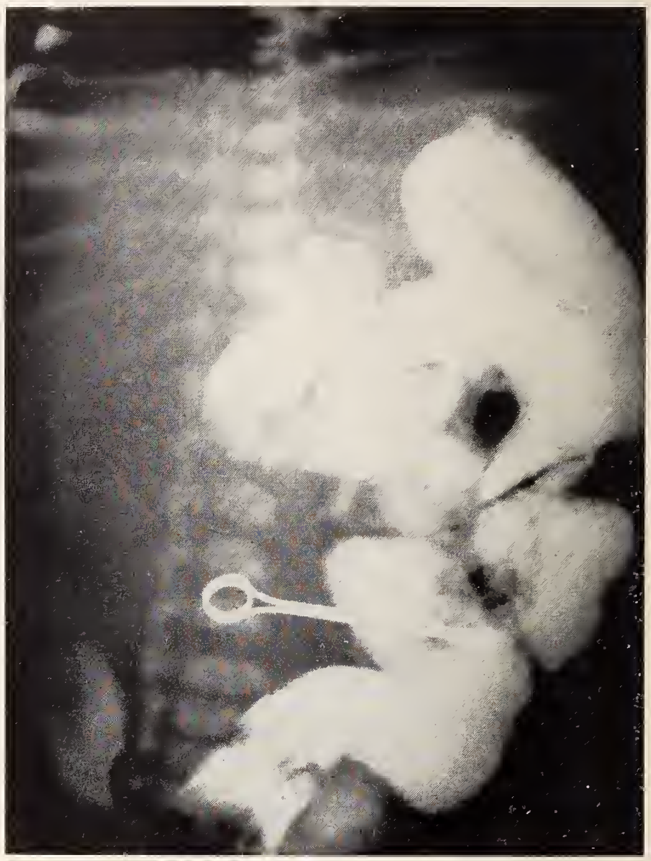

Fig. 6. Barium enema shows marked displacement of the cecum and ascending colon to the left produced by the soft tissue mass in the right retroperitoneal space.

Microscopic Description.—Sections of the tumor showed it to be composed of cavernous blood spaces many of which contained red blood cells (Fig. 3). There was no evidence of malignancy.

Pathological Diagnosis.—Cavernous hemangioma of the abdomen.

Postoperative Course.—The infant tolerated surgery well, and was returned to the recovery ward in fair condition. However, about three hours later, his respiration ceased. Artificial breathing and cardiac stimulants failed to revive the infant.

Post-mortem Examination disclosed atelectasis of both lungs. Microscopic study of the lesions of the skin revealed cavernous hemangiomas. No other finding of note was observed.

Discussion

Watson and McCarthy[24] studied a series of 1,056 cases of angiomas of the skin. Some of these cases had multiple lesions. Of this latter

TABLE I. CLINICAL SUMMARY OF THE PRESENT CASE AND THE THREE PREVIOUSLY REPORTED CASES OF RETROPERITONEAL CAVERNOUS HEMANGIOMA

Age	Sex	Symptoms	X-Ray Findings	Diagnosis Made	Associated Lesions and Fate of Patient	Case Reported By
59 yrs.	White Male	Dull aching pain in the left flank	"Slight displacement of the left ureter toward mid-line, distortion of the pelvis and calyces due to changed axis of the kidney and apparent distortion of the kidney substance in the lower pole . . ."	After Operation	Cardiac enlargement and murmur Patient died suddenly on 10th postoperative day (no autopsy)	Harris[8] 1929
32 yrs.	White Male	Generalized abdominal pains of six months duration	"Barium sulfate enema showed the cecum and ascending colon displaced obliquely as far as, and even somewhat beyond the mid-line."	After Operation	No associated lesions. Patient survived surgery and apparently cured	Millman[13] 1944
5 mos.	White Male	Rectal Bleeding	Barium enema negative	After Operation	Two hemangiomas, one overlying the right buttock and the other over the mid-sacral crest. Patient died during surgery.	Ward and Stewart[23] 1950
Newborn	White Male	Right abdominal mass discovered on physical examination (no symptoms)	Scout film of the abdomen and barium enema showed a large soft tissue mass in the right lower quadrant displacing the cecum and loops of small intestine medially.	Diagnosis suggested prior to operation	Three hemangiomas of the right thigh over the approximate distribution of cutaneous branches of L4. Died about three hours after operation.	Aurelius et al.

group, two had associated hemangiomas of the viscera. One had a hemangioma of the right kidney, and the other had a hemangioma of the sigmoid colon. Dean and McCarthy[4] state that "hemangiomas of the unusual location or extent are often part of a larger congenital pathologic complex." The validity of this statement is attested by the present case and the case reported by Ward and Stewart.[23] In both of these patients, the retroperitoneal cavernous hemangiomas were part of a diffuse similar process involving an extremity. Furthermore, Dean and McCarthy[4] call attention to the possibility of a vascular tumor of the viscera in patients with multiple hemangiomas of the skin who have bleeding of an undetermined origin.

The first retroperitoneal cavernous hemangioma was reported by Harris[9] in 1929. Since then, only two additional cases have appeared in the medical literature.[13,23] Stout[18] mentions a case reported by Hilse[10] in 1929. This, however, was actually a retroperitoneal lipoma which had areas of cavernous hemangiomas within it. This tumor recurred following surgical removal. The case of bilateral cystic lymphangiomas of both kidneys which was reported by Dasco[3] cannot be classified as hemangiomas. Retroperitoneal cavernous hemangiomas of the connective tissues of the pelvic floor have also been recorded by Frank[7] and Tapfer and Just.[19]

For the sake of brevity, the clinical history, and roentgenological findings of the four reported cases of retroperitoneal cavernous hemangiomas have been outlined in Table I.

An analysis of this table discloses an apparent predominence of these lesions in the male. However, in view of the small number of reported cases, no definite conclusion is warranted. To our knowledge, no sex affinity has been observed in the large reported series of the hemangiomas of various locations. Greater incidence of hemangiomas of the skin of the premature white infants as compared with negroes has been noted by Rambar.[16]

Due to the rarity of the visceral hemangiomas, the diagnosis is seldom made prior to surgical exploration or post-mortem examination. In the three previously reported cases, the diagnosis was made only after surgery. In the fourth case, which is the subject of the present report, the possibility of a retroperitoneal cavernous hemangioma was suggested because of the presence of several large colored lesions of the skin on the same side of the body on which the abdominal tumor was palpated and demonstrated by the roentgenogram.

Pathogenesis.—The great majority of hemangiomas are congenital in origin and are present at birth. Willis[25] believes that "angiomas are not true tumors." He considers them to be congenital malformation originating from embryonic mesenchymal cells which have the ability to proliferate and form blood vessels and also a variety of other tissues as well. Ewing,[5] on the other hand, maintains that hemangiomas are due to developmental anomalies of certain blood vessels which retain their embryonic characters. Virchow[22] attributed the formation of hemangiomas to the action of irritants on imperfectly formed blood vessels. Thoma[21] believed that these lesions are the result of mechanical disturbance such as increased blood pressure. A greater incidence of hemangiomas of the skin in premature infants in whom the temperature regulating mechanism of the body is defective led Rambar[16] to suggest that there may be a relationship between the development of angioma and the peripheral vascular needs of the infants. Finally Ribbert[17] is of the opinion that hemangiomas have their origin in embryologic rudiments.

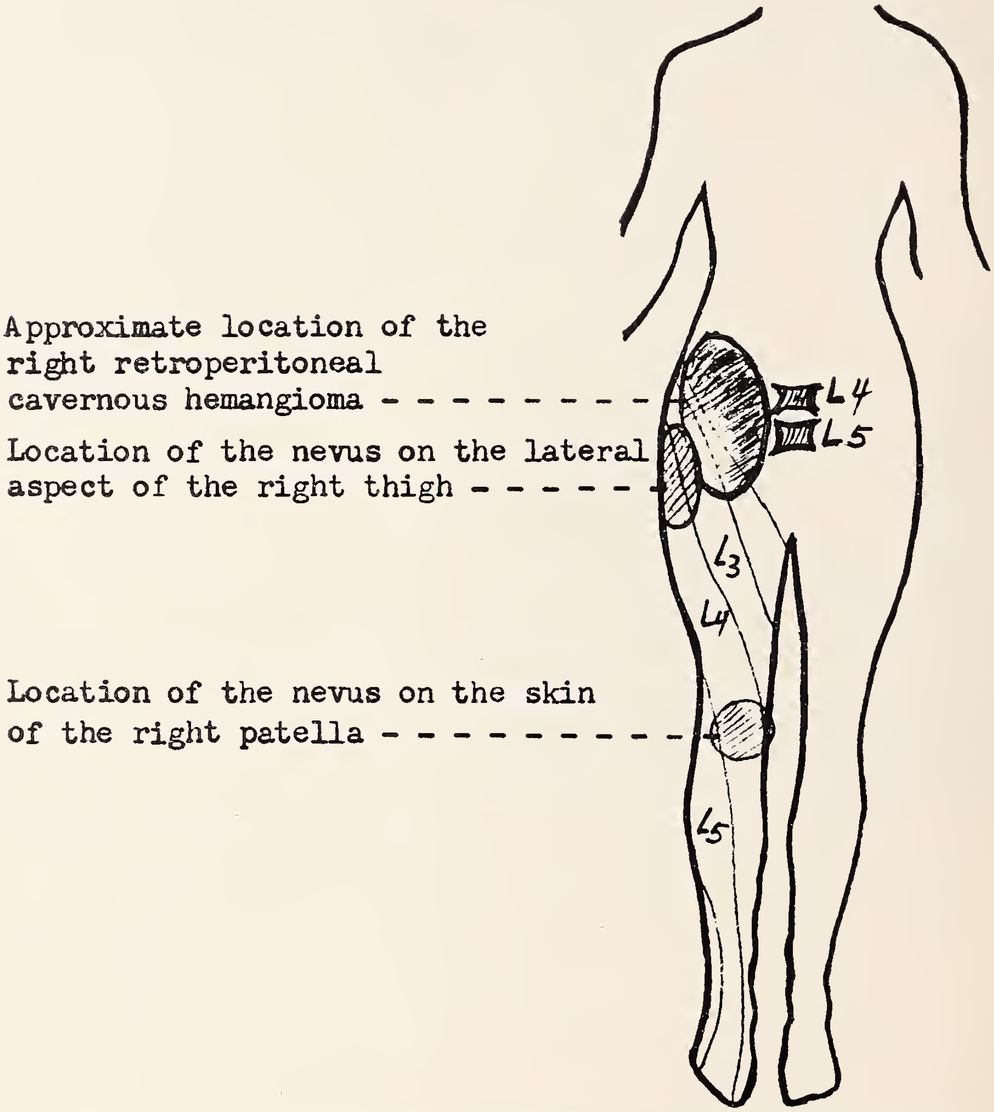
Approximate location of the right retroperitoneal cavernous hemangioma
Location of the nevus on the lateral aspect of the right thigh
Location of the nevus on the skin of the right patella
L4
L5
L3
L4
L5

Fig. 7. Diagram of cutaneous nerve distribution of the right leg. Notice the distribution of cutaneous branches of L4, and location of the hemangiomata. (Redrawn from J. J. Keegan, Anat. Record, 1948).

Differential Diagnosis.—The possibilities of a Wilm's tumor and sympathicoblastoma, two of the most common malignant abdominal tumors of infancy, were considered. Absence of metastasis to the lungs, and lack of derangement of architecture of the right kidney as would be expected from a tumor of this size were against the diagnosis of Wilm's tumor. Sympathicoblastoma was thought unlikely due to lack of any discernible metastasis in the long bones and in the skull. Since the bulk of the tumor appeared to be located below the inferior pole of the right kidney, the possibility of a supra-renal hemangioma, as recorded by several authors[12,14,20] was held improbable. Other possibilities that could not be excluded by conventional roentgenographic methods were teratoma, and retroperitoneal hematoma secondary to rupture of a cortical hemangioma of the right kidney. Several instances of the latter occurrence are found in the medical literature.[2,14]

Likewise, the presence of a retroperitoneal chyle cyst as reported by Gerster[8] could not be ruled out by the available diagnostic tools.

Distribution. — Thought-provoking conjecture can be made about the interesting distribution of the hemangiomas in our patient. Yakovlev and Guthrie[26] in a scholarly analysis of ectodermoses in epilepsy draw attention to peculiar predilection of the angiomatous growths for the tissues of ectodermal origin. They believe that obvious analogies exist between the angiomatosis of the brain and the two other types of congenital ectodermoses, namely, that of Recklinghausen's and Bourneville's diseases. A case of hemangioma of the spinal cord associated with four port-wine nevi of the skin of the corresponding neuromere reported by Cobb is cited[26] as "the classical example" of the neuromeric distribution of these lesions. As early as 1911, Fitzwilliams[6] recognized this distribution and stated: "their extension and growth is intimately connected with the nerve supply of the part." In our case, the location of the retroperitoneal hemangioma opposite to the body of the 4th lumbar vertebra corresponds quite well with the distribution of the nevi which were located in the areas of the right thigh supplied by the cutaneous branches of the 4th lumbar nerve as illustrated in Figure 7.

Thus the presence of hemangiomas of the skin and a study of their distribution may help in the differential diagnosis of a visceral tumor whose nature is not known with certainty.

Summary

A case of retroperitoneal cavernous hemangioma associated with similar lesions of the skin has been presented. The literature on the subject has been reviewed. The association of hemangiomas of the skin with similar lesions in the viscera has been stressed, and their neuromeric distribution has been discussed.

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Facts about Nursing in Minnesota

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IN CONSIDERING our common problem—meeting the nursing personnel shortage—perhaps a few facts and figures will give us a broader understanding of the problem and aid us in finding a new solution. This question of the shortage of nursing service, particularly in hospitals, is not alone the responsibility of nurses, but of doctors, hospital administrators, and other health workers as well.

The solution of our problem most frequently advanced is: "more schools of nursing will produce more nurses." Let us look at the figures (Table I).

TABLE I. PROFESSIONAL NURSING SCHOOLS

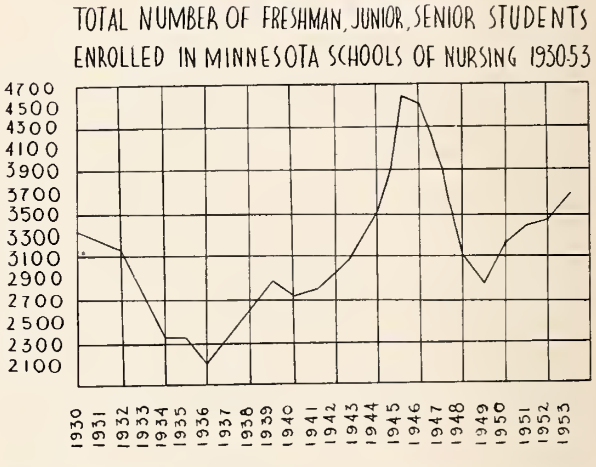
(Minnesota State Board of Examiners of Nurses)

	Number Accredited	Student Enrollment	
January, 1954	23	3,701	
" 1949	27	2,891	
" 1945	26	4,601	(peak)
" 1944	26	3,539	
" 1939	27	2,866	
" 1934	45†	2,359	

†11 Schools were in process of closing.

In 1934, there were forty-five accredited schools of professional nursing with 2,359 students enrolled. As the number of schools has decreased, the number of students enrolled has increased. In 1954, there were twenty-three accredited schools with 3,701 students enrolled. During the years 1943-1948, the United States Public Health Service through the U. S. Cadet Nurse Corps program financially supported Nursing Education and that is the reason for the increased enrollment shown for the year 1944-1945.

The total number of freshman, junior, and senior students enrolled in Minnesota schools of nursing shows a steady increase since 1936 to 1953. Again, it is necessary to keep in mind that the increased enrollment from 1943 to 1948 is the result of the Cadet Nurse Corps program. The increased enrollment from 1951 to 1953 reflects the number of students receiving assistance under the State Scholarship Aid program. More will be said about that later.



(From Minnesota State Board of Examiners of Nurses.)

Today there are twenty-six accredited basic professional nursing programs in Minnesota in twenty-three schools of nursing. Of these, twenty are three-year diploma programs, and six are four-year programs leading usually to a Bachelor of Science in Nursing degree. (Three of the six collegiate nursing schools have both Bachelor degree and diploma programs.) Eighty per cent of the students are enrolled in the diploma programs and 20 per cent in the degree programs. One school, the University of Minnesota, offers programs to graduate nurses leading to both a Bachelor's degree and a Master's degree. In addition to the twenty-three schools, thirty-two hospitals and health agencies in Minnesota provide one or more approved courses in nursing by affiliation.

Nationally the picture is the same.

You will note that since 1930 the number of schools has decreased from nearly 2,000 to a little over 1,000 in 1953, while the number of students enrolled has increased from around 80,000

Presented at the annual meeting of the Southern Minnesota Medical Association, Winona, Minnesota, September 13, 1954.

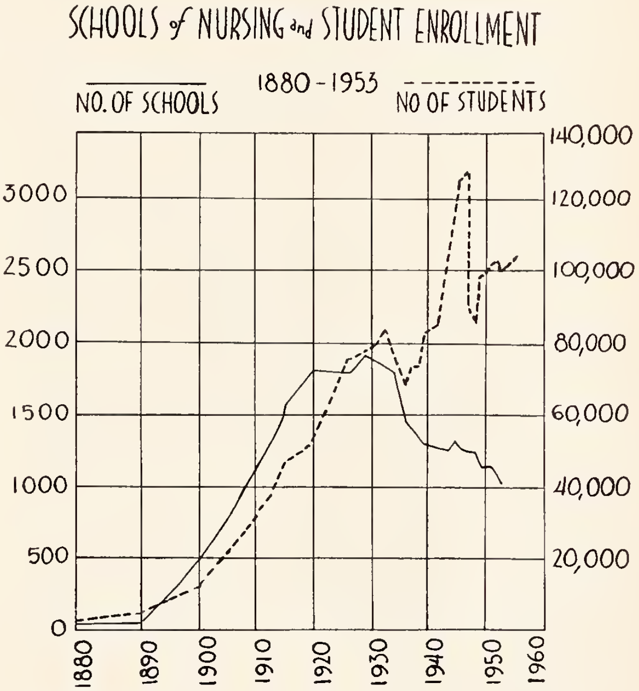
in 1930 to over 100,000 in 1953. (The peak between the years 1943 and 1948 was the result of the U. S. Cadet Nurse Corps program.)

A recent study by the American Nurses Association† shows that as of January 1, 1953, Minnesota had the seventh highest total student nurse enrollment among the forty-eight states. (Yet 1950 census figures show that Minnesota ranked eighteenth among the states in population.) The same source showed that Minnesota admitted the eighth highest number of student nurses, and graduated the seventh highest number.

In the fiscal year 1953-54, Minnesota licensed 1,466 professional nurses—980 by examination and 486 by interstate endorsement. Minnesota endorsed to other states 820 registered nurses. As of July 1, 1954, 36,603 R.N. licenses had been issued in Minnesota since the licensing law was passed in 1907. A total of 12,680 registered nurses had renewed licenses for 1954. (Many of these, however, reside outside of Minnesota or are inactive.) The A.N.A. source quoted previously† indicated that Minnesota had the sixth highest number of new registered nurses licensed by examination. Yet in 1952, Minnesota had only the twelfth highest average hospital census.

Practical nursing in Minnesota is a much newer venture, the Licensed Practical Nurses licensing law having passed in 1947. As of September 1, 1954, there were thirteen accredited practical nursing programs in Minnesota with a total enrollment of 300 students. Size of the student groups vary from eight to fifty-two; eight schools have less than twenty students enrolled. Minnesota ranks fifth highest among the states as to the number of practical nursing programs. In the fiscal year 1953-54, Minnesota licensed 300 practical nurses by examination and eleven by interstate endorsement. As of July 1, 1954, 2,958 licenses had been issued to practical nurses since 1947. A total of 2,541 had renewed their licenses for 1954. (Some of these, however, are inactive or reside outside Minnesota.) The A.N.A. study previously quoted,† indicated that Minnesota had the sixth highest number of new licensed practical nurses licensed by examination in 1952.

Many of the Minnesota Schools of Nursing, both professional and practical, could accept more students than apply for admission. Thus we can assume that no qualified applicant is denied admission to a Minnesota school of nursing because all classes are filled. Furthermore, there are at present, listed with the Minnesota Nurse Counseling and Placement Service, about twenty-five vacancies on Minnesota School of Nursing faculties. Also, many of the faculty positions are at present filled by nurses not fully qualified by education or experience for these important teaching positions. Opening more schools of nursing would not, therefore, increase student enrollment and would obviously deplete the present school faculties to a danger point. Studies that have been completed regarding the cost of a school of nursing to the hospital have indicated in most instances that the schools are not self-supporting. One study completed in Minneapolis at the Swedish Hospital School of Nursing for the year 1951, concluded that the cost to the hospital for each student in the professional school was $639.36 over and above the tuition that the student pays and the services that she contributes in her learning experience. Thus the operation of a school of nursing is an added cost to most hospitals.



(From "Facts about Nursing, 1953," American Nurses Association.)

Why are we faced with this alarming shortage of nurses? The shortage is the product of trends in medical care which we neither can nor wish to reverse. The shortage might better be expressed as an increased demand for nursing services. This increased demand has been influenced by many factors such as pre-paid hospital insurance plans, new and better medical techniques requiring more nursing service, and increased hospital construction, to mention only a few. For example, during the past five years, over 5,000 beds have been added to Minnesota hospitals.

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†"Facts about Nursing—1953," American Nurses Association.

What have we been doing about the problem?

The Minnesota Nurses Association through its state and district committees has carried out an active student nurse recruitment program. In the year 1953-54, a total of 119 high schools of the 484 in Minnesota were visited and nursing information was distributed on career days; high school counselors were visited; five films on nursing were shown in forty schools; 5,000 brochures were distributed; MNA co-sponsored the special vocational issue of the *Minnesota Daily* providing information about the various health fields. This special edition was distributed to every high school senior in Minnesota. And these recruitment activities were financed entirely by the nurses of Minnesota!

Despite the increasing competition for women in other professional fields and in industry: (a) more nurses are practicing their profession than ever before; (b) a larger proportion of girls are entering nursing than ever before—except during the U. S. Cadet Nurse Corps period 1943-48; and (c) the average working life of a nurse is longer than ever before, largely because many more married nurses are working.*

The state Scholarship Aid program has provided assistance to 795 student nurses since the law was passed in 1951. All but two counties in Minnesota have students in schools of nursing who are recipients of the state scholarships. The criteria for awarding these scholarships is based on the financial need of the student and her ability to complete her nursing course. The maximum amount of a scholarship for the professional nursing candidate is $600, and for the practical nursing candidate is $300. In return for this scholarship, the student must practice nursing in Minnesota for one year. We plan to introduce the scholarship bill in the 1955 legislative session and would like to have the continued support of the bill by the Minnesota State Medical Association. In addition to the state scholarships, hospitals, medical groups, or communities in general might well establish local nursing scholarships, the recipients of which would be required to return to work in that community for a specified time.

If it costs a hospital $636 per student to operate a school of nursing, why isn't it just as economical for the student to be subsidized to that amount providing she returns to that community or hospital for employment as a graduate? Such an *annual* scholarship would help build up a supply of nursing personnel for the local hospital.

If "more schools of nursing" is not the answer to our problem, what is the solution? Obviously we cannot continue to maintain the status quo and wait for the return of the "good old days." We must learn to utilize the personnel we have more effectively. To do this we must cast off the shackles of tradition and scrutinize each task within the hospital to see whether it is really necessary for the welfare of the patient and whether it is being performed in the most economical manner from the standpoint of time and choice of personnel. This takes planning, not only in the nursing department but with the medical profession and hospital administrators as well. We must learn to use the knowledge and skills of each individual—professional nurse, practical nurse, attendant, nurse aide, and orderly—to its maximum.

We must see that professional nurses are assigned only those functions which require the type and amount of training they have had; that practical nurses are used in situations where some formal training is necessary but not to the same degree as the registered nurse; that attendants, nurse-aides and orderlies have an on-the-job training adequate to prepare them for their jobs. Numerous hospitals already are making progress in this regard. The key to the solution, however, lies within the individual hospital, since no two institutions present identical problems.

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*"Meeting Your Hospital Personnel Shortage"—August, 1953. Office of Defense Mobilization, Health Resources Advisory Committee.

Sudden, Apparently Unexplained Deaths during Infancy

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SUDDEN, APPARENTLY unexplained deaths during infancy are always tragic events with painful consequences for everyone concerned. If they are not handled properly the parents may develop an acute sense of guilt over their possible negligence in the situation.

An infant in apparently good health is put to bed in a crib or buggy or in bed with its parents. Several hours later the infant is found dead. Sometimes he is lying on his face. Sometimes bed clothes are over his head. Often in the excitement the exact conditions are unnoted. The physician who is confronted with such a death is likely to assume that the baby has smothered or that he has died because of an enlarged thymus.

Upon examining 167 autopsies of children under one year of age who died suddenly, Werne and Garrow[2] (1947), were not able to find one case in which they could prove mechanical suffocation by bed clothes. It is probable that mechanical suffocation may occur as a terminal event during a debilitating disease in a child, but there is no risk of accidental suffocation from the bed clothes in a healthy child. There is also no relationship between the size of the thymus and adrenal glands and sudden death. Rabson[1] (1949) showed the relationship between sex and unexpected natural death in New York City. In the age group from birth to four years, males outnumbered females 2.7 per cent to 2.0 per cent, while in the age group from five to nine years the sexes were equally represented. During the first five years, four times as many boys as girls died suddenly. No girls were counted among the few cases of sudden unexpected death in the second period. This is another confirmation of the often remarked lethality of boy babies. Werne and Garrow,[3] in a study of 299 cases, have shown that pathologically there are microscopic inflammatory lesions of the upper and lower respiratory tracts and vascular changes in the respiratory tract, spleen, lymph nodes and thymus in these cases. The mononuclear pneumonia which is usually found is thought to be agonal, as it also is seen following a violent death in a child. The vascular changes are usually thrombi, focal inflammation, edema and hemorrhage. According to Werne and Garrow, the chief pathological findings in sudden apparently unexplained death during infancy are acute inflammation of the upper or lower respiratory tracts; gross exudate in the middle ears and mastoid antra; acute suppurative tonsillitis; congestion of the meninges; excess subarachnoid fluid; minute focal subarachnoid hemorrhages; congestion of the mucosa of the larynx, trachea, and bronchi; foci of necrosis in the larynx and trachea; hyperemia and hemorrhage in the tracheobronchial and cervical lymph nodes; pulmonary congestion and edema; emphysema; pulmonary capillary congestion and bronchitis or bronchiolitis; focal accumulation of macrophages and lymphocytes within the alveoli and pharyngeal filling with regurgitated vomitus. The vomitus is thought to be an agonal phenomenon.

In most cases, the etiology of a sudden death in an infant is fulminating respiratory disease. Other causes of apparently unexplained death during infancy are: meningococcemia; cerebral hemorrhage; adrenal hemorrhage; obstruction from a laryngeal papilloma; coronary artery disease; anaphylaxis; anesthesia; and postoperative sepsis.

Table I is a summary of seven case reports of children who died suddenly. Pathologically, they all showed bronchopneumonia.

In summary, the etiology of sudden apparently unexplained death during infancy is usually fulminating respiratory disease. This occurs

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Presented at the Regional Meeting of the American College of Physicans at Bismarck, North Dakota, September 11, 1954.

TABLE I.

Case	Sex	History	Age at Death	Postmortem
1. Ryckman No. 109523 E	Male	B. wt. 7 lbs. 11 oz. Normal fullterm. Spontaneous delivery.	2 days	Bronchopneumonia
2. Tibesar No. 79975 G	Male	B. wt. 4 lbs. 3 oz. Bronchopneumonia, 3 mos.	4 mos.	Bronchopneumonia
3. Kielty No. 115656 A	Male	Erythroblastosis. Basilar meningitis. Cortical atrophy. Bronchopneumonia, 1 yr.	14 mos.	Bronchopneumonia. Internal hydrocephalus.
4. Hatzenbuhler No. 122472 B	Female	Myasthenia gravis (?)	8 mos.	Bronchopneumonia
5. Hellman No. 123893 A	Male	Common bile duct atresia. Frequent bronchitis.	5 mos.	Bronchopneumonia. Biliary cirrhosis. Biliary atresia.
6. Brady No. 130898	Male	B. wt. 6 lbs. 9 oz. Normal fullterm. Spontaneous delivery.	5 weeks	Bronchopneumonia
7. Miller No. 109338 E	Female	Congenital hemolytic anemia, 2 days. Splenectomy, 4 mos.	2 8/12 yrs.	Bronchopneumonia

more commonly in males. Agonal phenomena seen are: the presence of bed clothes over the face and around the neck; pharyngeal vomitus and a mononuclear pneumonia. In order to make a correct diagnosis a careful postmortem examination must be made in every case.

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"NO PRACTICAL VALUE"

Gamma globulin as a preventive of paralytic poliomyelitis has been found "of no practical value," says Gaylord W. Anderson, M.D., director of the School of Public Health, University of Minnesota.

The supposed prophylactic effects of GG were studied during 1953 under very carefully controlled conditions by a committee appointed by the U. S. Public Health Service. More than 235,000 children were involved in these studies. The report of the committee showed that beneficial effects were not demonstrated either in the mass inoculation of children in epidemic areas or in the inoculation of family and other close contacts of patients already ill with polio.

These findings, as well as a review of the current status of gamma globulin supplies on hand and of requirements for globulin, led to the recent decision by the Office of Defense Mobilization to discontinue the rationing of GG, a program carried on by that agency since 1953. This co-ordinated distribution of globulin (for poliomyelitis) through state health departments will not be continued after December 31, 1954. According to the ODM, an increase in supply of globulin has taken place during the last two years to a point where all foreseeable demands can be met and rationing is no longer necessary.

GG is already available for purchase once again in drug stores for use by physicians in any way they desire. In addition to this source of supply, the Minnesota Department of Health expects to continue a limited program of distribution of globulin to physicians for certain uses, such as the prevention or modification of measles in young children and the protection of household contacts of cases of infectious hepatitis. Globulin has definite value in these conditions. GG that is distributed by the State Department of Health is furnished without charge through the American Red Cross and the National Foundation for Infantile Paralysis, Inc.—*Minnesota's Health,* December, 1954.

LABORATORY CONTROL OF ANTICOAGULANT THERAPY

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THE USE of the one-stage prothrombin test (Quick prothrombin time) in the control of therapy with anticoagulants of the dicumarol type is an excellent example of the role of the laboratory in modern medicine. In contrast to the screening procedures that occupy much of the time of the clinical laboratory, each prothrombin-time test in the control of anticoagulant therapy provides information specifically required and obtainable in no other way. A number of papers have appeared on this subject; the evidence behind most of these is somewhat less than impressive. Elsewhere I[2,3] have reviewed in some detail the evidence behind the rather strong and dogmatic comments that are to follow.

The prothrombin-time test consists in measurement of the clotting time of plasma on recalcification in the presence of standardized thromboplastin. The test is not merely rapid and convenient, as daily clinical laboratory tests must be, but is also highly accurate when it is properly performed. Nevertheless, unsatisfactory experiences with this test have been reasonably frequent; otherwise there would be no indication for this editorial report. These failures have been due to flagrant disregard of repeatedly emphasized technical principles, some of the more important of which follow:

1. The thromboplastin reagent used must be not only reproducible in its activity with respect to normal plasma but also specifically sensitive to the effect of dicumarol. The principal effect of dicumarol is not on prothrombin but on a substance called the "stable prothrombin conversion factor"; it is this substance and not prothrombin that is measured by the prothrombin-time test when it is properly performed. Retention of the terms "prothrombin time" and "hypoprothrombinemia" to describe the effect of dicumarol is nevertheless inevitable after the years of use of this terminology. The stable prothrombin conversion factor, even in minute amounts, reacts with thromboplastin to form a stable complex (cothromboplastin reaction). Preparations of thromboplastin that have undergone this reaction are specifically insensitive indicators of the effect of dicumarol. At least two such preparations have been offered commercially by means of advertising resembling that extolling certain patent medicines. Since it must be assumed that few clinical laboratories will prepare their own thromboplastin, a step necessary for maximal accuracy, the practical suggestion is offered that no thromboplastin be purchased unless it is specified to be prepared from rabbit brain only, with no added substance whatsoever. It is a sad and rather surprising fact that no commercial manufacturer of thromboplastin ever has seen fit to consult the originator of the prothrombin-time test, Dr. Quick, according to his own statement.

2. The specimen of blood for the prothrombin-time test must be meticulously collected. Incipient coagulation before mixture with oxalate, and likewise an excess of oxalate, must be avoided. The former gives an erroneously short prothrombin time; the latter may cause a false result of incoagulability. It is a good practical rule never to report a prothrombin-time test as showing "no clot" without examining another specimen of blood, since an incorrect report of incoagulability is obviously a serious error. Although one might think that such obvious errors should be rare, ample experience has shown that when specimens of blood are not collected by experienced labora-

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From the Section of Clinical Pathology, Mayo Clinic and Mayo Foundation.

This is the twenty-third in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

tory personnel they are often unsuitable for the prothrombin-time test. This probably has been an important factor in unsatisfactory experience with dicumarol therapy in large teaching hospitals in which specimens may be collected by medical students and interns.

3. The apparatus for the test must provide for control of temperature without sacrifice of visibility of the clot, preferably by means of a glass water bath. The light should be adjustable to the maximal advantage of the technician. There also must be a means of accurate measurement of the clotting time, preferably an electric stop clock operated by a foot pedal, which leaves the hands free for the necessary manipulation of pipet and test tube. Elaborate water baths that do not allow a good view of the clotting process and photoelectric apparatus that hampers the rapid addition of reagents and the control of temperature are not improvements.

4. Perhaps most important of all, the test must be performed by a person properly grounded in laboratory discipline who has had sufficient practice with this specific test under supervision. Such a person must be allowed to perform the test with reasonable freedom from interruption. Coagulation tests, involving precise timing, are peculiarly susceptible to error due to interruptions, such as the innumerable telephone calls that many clinical laboratories receive. Far too often, errors are attributed to basic inadequacies of technique when they are actually due to impossible working conditions.

Dogmatism will not be carried so far as to specify the precise version of the Quick prothrombin test that should be followed; reference is made to a technique that has given excellent results over a fifteen-year period.[1] Several other versions appear to have given good results but no modifications or gadgets are likely to circumvent the basic laboratory requirements with respect to reagents, specimens, apparatus, personnel and time.

This brief account is concerned only with the laboratory techniques involved in the control of anticoagulant therapy, not with the therapy itself. It may properly be noted, however, that the originators of such therapeutic methods emphasized from the outset the absolute necessity of precise laboratory control. It is thus surprising that authors who have condemned anticoagulant therapy as hazardous often have failed to mention how they performed the prothrombin-time test or even have stated that due to lack of technical personnel they were unable to perform this test accurately. This attitude resembles that of a surgeon who would condemn the operation of cholecystectomy on the basis of a series of strictures of the common bile duct.

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EARNINGS OF REHABILITATED VETERANS

Disabled veterans rehabilitated under a special VA training program have more than doubled their pre-war incomes. This is one of the findings of a follow-up survey conducted by the Veterans Administration. It shows that before service the men averaged $32 a week, but after the special training, they averaged $72 a week. In the postwar years, the average pay of all non-veterans went up 19 per cent, that of able-bodied veterans 50 per cent, but the average veteran who completed the special training increased his earnings 75 per cent in this period. Medical education was one of the fields where the veterans had a high rate of rehabilitation, 76 per cent. Medical disabilities of the veterans surveyed were classified as follows: orthopedic 41 per cent, nervous or mental 24 per cent, circulatory and heart 7 per cent, and tuberculosis or other respiratory 8 per cent.

The special training is restricted to service-connected cases where the disability has or could seriously interfere with the veteran's employment in his established occupation. Training is limited to four years. The average in the group studied twenty-one months.

Editorial

ARTHUR H. WELLS, M.D., *Editor*
HENRY G. MOEHRING, M.D.
JOHN F. BRIGGS, M.D.

"I'M NOT MARRYING YOUR MOTHER"

Well, maybe you aren't marrying your future spouse's mother, but you might almost as well be. And except for the less certainty about paternity, you might almost as well be marrying the (apparent) father, too. Setting aside all the personal characteristics that your spouse might acquire from parents via merely growing up with them, the Metropolitan Life Insurance Company's *Statistical Bulletin* for September, 1954, points out in its lead article on "Family History and Longevity" the following interesting facts.

The mortality among 15,000 policyholders who had two or more close relatives with cardiovascular-renal disease acquired before age sixty, was about 40 per cent higher than for standard ordinary risks as a whole. Most of the excess mortality was from diseases of the heart and vascular system.

In two large companies, among insured who, in addition to having a family history of cardiovascular-renal disease, either were slightly over weight or had a slight elevation in blood pressure, the mortality was 70 per cent higher than the average for ordinary standard risks.

The work of Dr. May Wilson and her associates in New York City indicates an hereditary susceptibility to rheumatic fever and rheumatic heart disease.

The prevalence of coronary heart disease was considerably higher among the parents of a group of young people suffering from coronary heart disease than it was among parents in a control group.

In policyholders reporting two or more cases of cancer in parents or siblings under age sixty, the mortality from all causes was about average, *but* the mortality from cancer, which accounted for one-third of all the deaths in this group, was more than twice the average.

The mortality from diabetes among policyholders with even a single diabetic person (at any age) in the family was nearly twice the average for all standard ordinary policyholders.

The mortality from suicide was several times the normal in insured persons who reported two or more cases of mental disease in the family before the age sixty (the age limitation serves to eliminate most cases of mental disease or degeneration due to cerebral arteriosclerosis).

Persons with a good family history of longevity, live longer on the average than those with a poor record of parental longevity.

All of which would make us amend the advice to choose a spouse with good health, intelligence, and a good sense of humor, by the addition of similar specifications for the spouse's parents.

H.G.M.

LIFE INSURANCE APPLICANT EXAMINATIONS

With more than 70 million Americans now owning life insurance and with hundreds of thousands of applicants being considered each year, the life insurance medical examiner has become a key figure in the largest American business enterprise outside the U. S. Government itself.

The wonder of it all is that in such a tremendous mass operation—involving printed forms, special rules and routines, and a multitude of personalities—the job of qualifying applicants for life insurance goes as smoothly as it does, with irritation and inconvenience to the examining doctor occurring only in relatively rare cases.

Consider some of the elements involved. There are forms to be filled out—fairly complex forms because the information desired usually relates to what will be a lifetime transaction, often involving many thousands of dollars. There are routines to follow which may on occasion seem a nuisance to the examiner unfamiliar with life insurance home office procedures. And lastly, there are personalities to be dealt with, not the least of which may be the applicant who, because he presumes himself to be healthy, does not always feel the same compulsion to co-operate as he would if he were seeking medical counsel in the usual doctor-patient relationship.

The fact, then, that these vast numbers of examinations are conducted so smoothly and successfully is, to a considerable extent, a tribute to the American physician's co-operation in expediting a social service which is of the broadest significance to modern-day Americans—doctors included.

The purpose of the life insurance examination is to obtain objective criteria which can be applied to the laws of actuarial and underwriting science. It is by no means a complete evaluation of the applicant's physical condition; this obviously would be too costly. But by establishing a minimum number of objective criteria such as the level of blood pressure, heart rate, height, weight, et cetera, definite objective or dollar and cents evaluation can be placed on the prospects for the individual's longevity. Actuarial and underwriting science is amazingly precise, even using as facts such variables as physical findings.

Relatively meager though these facts are, the industry has been able to set up a system of evaluating longevity which has proven very accurate. And since these facts are the whole basis for action, they must be accurately determined. The examinations in many cases have uncovered medical impairments of which the applicant was unaware, and have prompted him to seek medical help for these impairments.

The questionnaire which the physican is asked to fill out covers not only results of the examination, but the medical history of the applicant and his family, as certified to by the applicant. Some of these questions may strike the physician as reaching far afield, but each is designed to bring out some aspect of the record which the company, on the basis of its experience, deems relevant. An active committee of medical directors has as its sole purpose the elimination of unnecessary questions from the medical blank, and from the standpoint of the industry, these blanks have been materially shortened in recent years.

Unfortunately, because of the size of the industry, both applicant and physician must conform to a set routine. However, because of the importance of the examinations, and recognizing that the facts the doctors are attempting to establish are much better obtained in the doctors' offices, the companies prefer and do all in their power to see that these examinations are made at the doctors' offices.

It would be impossible to act impartially on the large number of cases handled each year if the company solicited the personal judgment of the physician. Medical underwriting depends on impartial facts, and cannot take into consideration the most interesting part of medicine—the *art* of medicine. The physician cannot be expected to decline or rate the applicant. It is his function to furnish the facts, and let the underwriters use these facts to determine the rating. This impersonal relationship has proven to be the only practical and equitable way of determining the necessary facts.

With hundreds of thousands of applicants reviewed each year, insurance "medicals" are a source of significant income to the medical profession. Currently, the life insurance companies are paying the doctors of the country about 17.5 millions a year for these examinations, with the level at which fees are fixed closely related to usual charges for comparable work.

As a result of this continuous co-operation by the medical profession, the insured mortality of the companies has been progressively improving, and this in turn has allowed the life insurance companies to hold the cost of life insurance protection to the American public at a far lower level than would otherwise be possible.

Karl W. Anderson, M.D.

RADIO-ISOTOPES IN MEDICINE

Although the advent of artificial radio-isotopes has had its principal impact on research, certain applications are emerging which are useful in the practice of medicine, both diagnostically and therapeutically. So far, there are four isotopes which stand out in this respect; namely, I-131, P-32, Au-198, and Co-60.

Diagnostically, the radioisotopes are employed as "tracers." For example, the avidity of the thyroid gland for iodine, a property directly related to thyroid function, is determined by administering orally a tiny quantity of radio-active iodine and then measuring with a Geiger counter after a certain number of hours the percentage of this I-131 which has been taken up by the thyroid gland. Similarly, the blood volume can be determined by injecting a small quantity of an appropriate radio-isotope into the blood stream, allowing time for mixing, and then withdrawing and measuring a sample to determine the dilution factor. Radio-isotope tracer techniques have the advantage of simplicity and increased accuracy over some of the alternative chemical and colorimetric methods.

The usefulness of thyroid uptake studies with I-131 is not limited to large institutions and city practice. This method is applicable to small scale practice also, and various rural hospitals and clinics are profiting from its contribution to the accurate diagnoses of thyroid aberration.

Therapeutically, radio-isotopes fall in the field of radiation therapy, and permit a wide range of applications, sometimes involving an intimate role in metabolic processes, as when P-32 is used to treat polycythemia vera, and I-131 to treat Grave's disease. Radioactive gold (Au-198) holds promise in the palliation of selected cases of carcinomatous effusions in the abdominal and pleural cavities. Co-60 has many of the properties of radium as well as certain advantages over it and is being used as needle sources as well as in large radiation units for the treatment of malignancy in thick portions of the body.

Thanks to the pioneering experience of the last sixty years in the hazards of radiation, numerous safeguards have now being instituted so that work with radio-isotopes can be a completely safe procedure.

Elmer C. Paulson, M.D.

THE SAVINGS AND LOAN INVESTMENT

What is the value to a doctor's estate of an investment in savings and loan associations? The answer is quite simple. No other investment of equal safety and liquidity pays such a high return. Let's look at those three key points in order: safety, liquidity and high return.

From a safety standpoint, you're on solid ground with an investment in insured savings and loan associations. You are investing in families and homes, the very backbone of America. Basically, the business of the savings and loan industry is that of making mortgage loans on single-family, owner-occupied homes. Financial history proves that those are blue-chip risks. They meet the most important safety requirement of a good investment program—diversification. The incomes of families buying homes are derived from hundreds of different businesses, services and professions. Your investment in savings and loan associations, in other words, is backed by all or most of the business activity in an entire metropolitan area.

In addition to the safety factors inherent in a savings and loan investment, your account at any federally chartered association is insured for safety up to $10,000 by the Federal Savings and Loan Insurance Corporation. This insurance is automatic without charge to you.

Turning to the factor of liquidity, or availability of your money, it has long been the custom of savings and loan associations in metropolitan areas to pay withdrawals promptly on request. Larger associations, in particular, ordinarily maintain a high degree of liquidity with a strong percentage of their assets in cash and government bonds. In addition, there is a constant flow of new cash into each association as a result of new savings accounts, monthly interest payments on loans and monthly repayments of the principal on mortgages.

This brings us to the important point of high return. Look back through the years and you'll find that savings and loan associations have always paid generous dividends. In the Twin Cities, they currently are paying investors an annual rate of 3 per cent compounded semi-annually. To earn a higher rate of return, you'd have to move into an investment area that may sacrifice safety.

Let us look briefly now at two other advantages of investing in savings and loan associations. It requiries no management expense, worry or bother, such as is involved in owning stocks or bonds and many other investments. And your savings and loan investment is not subject to fluctuations in principal value as is the bond and stock.

Basically, the savings and loan investment is valuable to your estate because it pays a higher return than is available on other investments of equal safety, liquidity and convenience.

Roy W. Larson

President, Twin City Federal Savings and Loan Association

CORRECTION

In the October, 1954, issue of Minnesota Medicine, an error occurred in the paper by Matthew A. Kiess, O.S.B., Ph.D., Robert P. Koenig, M.D., and Clifford R. Myre, M.D., entitled "Development of a Simplified Filter Paper Electrophoresis with a Study of Human Serum Proteins." Line eight, column two, page 709, should read ". . . and *15 grams* of sodium diethyl barbiturate . . ."

A. O. Swenson, M.D.
President, Minnesota State Medical Association
1955

President's Letter

THE ANNUAL CHALLENGE

The beginning of a new year, and all that traditionally goes with it, has turned out to be a challenge of major proportions in the year 1955.

Every year we find ourselves confronted with the dilemma of feeling obliged to make a certain number of New Year's resolutions—which we immediately break with equally resolute rationalization. What we need is the fortitude and determination to either keep every resolution we make, or to so live that it will be unnecessary to make resolutions in the first place.

But seriously, New Year's resolutions have a proper place in our lives today. We take a backward and a forward look at this time of year—we analyze where we stumbled in the past year, and decide firmly not to stumble over the same things in the coming year. And thus, we grow in stature, in experience and in better living.

We as physicians, however, find that January 1 is not the only time we must firmly resolve. Throughout every month of every year we are constantly challenged by some new problem which requires a quick, and correct, decision on our part. With every new patient, with every new medical organization problem, with every new medical-economic concept, we meet a challenge to maintain the tradition of public service which has been our American heritage.

Because the above tasks seem never ending, there is a tendency to become discouraged and lackadaisical in our efforts. Let us realize that it is a privilege to serve, and the simplest and most effective way to attain our ends is for each of us to strive diligently to make our lives as close to the ideal of medical practice as possible.

As your new president, I hope to take over the duties with a humble and dedicated heart. I pledge to work for Medicine and its high ideals; with the good will and willing co-operation of all conscientious members of the Association, I feel that we can continue to maintain and improve the high plane of practice we have known in Minnesota through the years.

To say that I am proud of our organization is somewhat of an understatement, for this group has been a strong and valiant one in the state. It has constantly stood for right; it has fought hard for its position of leadership; it has been headed by some of the finest people in the profession. With such standards to follow and uphold, it will be a great challenge to your president. But I hereby resolve to serve my term as president to the best of my ability; and I pray that, with your valuable assistance, I shall not break that resolution.

Arnold O. Swenson

President, Minnesota State Medical Association

The Dean's Page

I would like to take this opportunity to say a few words to the physicians of Minnesota concerning the requests which the Regents of the University will present to the 1955 Legislature for the support of the Medical School and University Hospitals.

The basic support of the Medical School—that is, faculty and technical staff salaries, supplies and expenses, library, heat, light, custodial service and maintenance of Medical School buildings—is included in the general University maintenance request for $17,262,846 for each year of the 1955-57 biennium. This includes $771,423 for promotions and for a 7.5 per cent increase in faculty salaries to partially meet increases in living costs which have occurred over the past two years: $391,647 for increases in Civil Service salaries, 6.9 per cent, in accord with the State Civil Service pattern; and $707,719 for new faculty positions to take care of increasing enrollments and other service responsibilities.

The University Hospital request is a "special" of $1,100,000 for the first year of the biennium and $1,125,000 for the second year of the biennium. This really represents an authorization to the state treasurer to reimburse the University for one half of the bills of "county patients" up to a maximum of this amount. Incidentally, the annual appropriation requested is $155,736 less for the first year and $130,736 less for the second year than for the year 1954-55.

Other "specials" for each year of the biennium include $75,000 for general medical research, $15,000 for the human aspects of brucellosis, $15,000 for continuation of the Multiple Sclerosis Clinic, $469,966 for the Psychopathic Hospital, $166,000 for Child Psychiatry, and $278,345 for the Rehabilitation Center. There is no matching of state funds by counties for patients in psychiatry, child psychiatry, or rehabilitation.

For building the Regents are requesting three appropriations that are vitally important to the Medical School. These are as follows: for remodelling of Millard Hall $330,000; for the Medical-Biological Library $875,000; for the remodelling of the University Hospitals and for needed equipment of the Hospital and Mayo Memorial $1,000,000.

The remodelling of Millard Hall, which was constructed more than forty years ago, has been long overdue but has been postponed until some of the departments that were previously housed in that building could be moved into the Mayo Memorial. This remodelling is a "must" if the laboratories in this building are to be used by the Departments of Physiology, Physiological Chemistry, and Pharmacology that will continue to occupy this building.

The Medical-Biological Library was listed almost twenty years ago by the students and faculty as the most critical need of the Medical School. The University of Minnesota has a splendid medical library, but since it has been housed in the Main Library Building, three or four blocks from the Medical School and Hospital, its use has been minimal. In the plans for the Mayo Memorial two floors were to be devoted to the Medical Library. However, the inflation and increased building costs which occurred as a result of the war in Korea necessitated the elimination of eight floors, including the library, from the tower portion of this building. The Medical Library was then placed on the University's list of buildings requested from the 1953 Legislature, but with appropriations at this session for a new law library building and for a library building on the Duluth Campus, the Medical Library was passed over.

A most important aspect of modern medical education is to develop in medical students the habit of intelligent and discriminating reading of medical literature as a basis for keeping abreast of medical developments throughout their years of

(Continued on Page 66)

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

FEDERAL HEALTH BUDGET TO REACH OVER $2 BILLION

The federal government this fiscal year (July 1954-June 1955) is spending an estimated $2,141,681,661 for its health, medical and related activities, which range from construction of multi-million dollar hospitals to maintenance of clinics, according to a recent issue of the AMA Washington letter.

The programs are spread among more than a score of departments, independent agencies, and commissions. This total figure means:

1. It is 8 per cent more than $1,775,882,000 budgeted by the federal government last year for the same purpose. (The 8 per cent overall increase does not include that part—70 per cent—of the Defense medical budget increase that the department estimates is chargeable to operating and construction costs, items not available last year under the accounting procedure then in use.)

2. It is equivalent to 10 per cent of the $22,000,000,000 that represents the entire U. S. budget with defense security costs eliminated.

3. It is approximately one-sixth of all the money spent in any way for medical and health items in this country annually, including the cost of drugs, dental care, pharmaceutical items, and drug store sundries. (Department of Commerce estimates the total U. S. health bill at $12,000,000,000.)

Departmental Breakdown

Breaking down the total figure by departments and their individual health and medical budgets, the following figures come to light:

Department of Defense—this year: $845,487,500; last year: $533,311,000. Department of Health, Education and Welfare—this year: $395,754,000; last year: $340,553,000. Federal Civil Defense Administration—this year: $28,755,000; last year: $26,650,000. Department of Interior—this year: $28,023,498; last year: $27,258,600. Atomic Energy Commission—this year: $27,000,000; last year: $26,565,000. Department of Labor—this year: $6,811,000; last year: $8,960,000. Federal Employes Health Program—this year: approximately $6,000,000; last year: approximately $6,000,000. Federal Trade Commission—this year: approximately $1,000,000; last year: approximately $1,000,000. Commission on Intergovernmental Relations—this year: $414,000; last year: $500,000. Civil Service Commission—this year: approximately $358,000; last year: new item. Department of Commerce—this year: $278,133; last year: $621,000. Veterans Administration—this year: $748,738,563; last year: $747,415,264. Department of State—this year: $12,607,667; last year: $14,127,733. Foreign Operations Administration—this year: $25,574,300; last year: $24,500,000. Panama Canal Zone—this year: $5,600,000; last year: $5,448,600. National Science Foundation—this year: $4,795,000; last year: $8,000,000. Department of Treasury—this year: $2,770,000; last year: $2,790,000. Department of Justice—this year: $1,300,000; last year: $1,326,000. National Advisory Committee to Selective Service—this year: $190,000; last year: $265,000. Commission on Organization of Executive Branch of Government—this year: approximately $130,000; last year: $500,000. Health Resources Advisory Committee—this year: approximately $95,000; last year: $91,000.

JAMA ASKS TAX-SUPPORTED MEDICAL SCHOOLS RELAX RESTRICTIONS

A recent issue of *The Journal of the American Medical Association* has recommended that state and city medical schools should relax their restrictions on admitting students from limited geographical areas.

An editorial in *The Journal* states that such limitations represent "a shortsighted policy that is a disservice to both the state and the school." The article goes on:

> "With roughly one-third as many applicants per vacancy to choose from as the other schools, the tax-supported schools are often placed in the unfortunate situation of having to select inferior applicants or fail to fill their quota."

With continually improved methods of screening candidates, *The Journal* says, the nation's medical schools have sharply cut the rate of students who drop out—largely because of academic failure—before graduation. This attrition rate dropped from 9.4 per cent in the freshman year 15 years ago to 5.1 per cent in 1952-53. Senior year attrition was only 0.2 per cent that year.

The article concludes: "As a result of being forced into unwise selections these (city and state) schools have an attrition rate twice that of schools that are free to select students without reference to their state residence. . . . If state and city schools would relax their restrictions even to the point of choosing a certain percentage of their students from other areas (at a slightly higher rate of tuition and with a certain amount of reciprocity) this would have a salutary effect."

AMEF REPORTS STATE STANDINGS

A recent report from the American Medical Education Foundation reveals the individual state standings in regard to reaching annual contribution goals to the Foundation.

The Foundation reports:

> "In over-all income and contributors, the Foundation is ahead of last year's totals. However, we are still short of the estimated $1,200,000 income figure that was projected earlier this year. We have received a total of $1,051,000 from more than 17,000 contributors. Since we did not pass the million dollar mark until late in November last year, we are ahead in our income record of last year by about 30 days. In short, we have an estimated 30 days to surpass our 1953 record."

The above statement was made early in November, and the bulletin was issued in an effort to pass the goal before the end of 1954.

State Contributions

Summarizing individual state achievements in this vital field of aiding medical education, the Foundation stated that during the first ten months of 1954, six states exceeded their 1953 marks in both dollars raised and contributors. Four other states increased their dollar income over 1953 but have not exceeded their total number of contributors. Five states exceeded totals in the number of contributors but not their dollar income over the previous year. Two states remained even with their 1953 records.

It is disappointing to note that Minnesota is not on any of these lists. However, Minnesota physicians have an opportunity to begin 1955 with a new and better record. The importance of making contributions to this fund cannot be overemphasized, for it has been one of American medicine's greatest weapons against federal subsidies for medical schools, which threatens federal control. It is also noted that considerable tax saving is realized by contributing to this fund.

PRESIDENT CITED FOR MEDICAL EDUCATION WORK

For his demonstrated interest in medical schools, and his efforts to bring them financial support from non-governmental sources, President Eisenhower has been honored with the first Frank H. Lahey award. While he was president of Columbia, Mr. Eisenhower was largely responsible for getting the National Fund for Medical Education under way. This fund collects money from non-government sources for distribution to medical schools "with no strings attached."

The Lahey award is a memorial to the late Dr. Lahey of Boston, past president of the AMA, who was a leader in the movement to keep medical schools solvent without federal aid. The presentation was made at the White House by S. Sloan Colt of New York, president of the National Fund for Medical Education. It was sponsored by the American Medical Association and the Association of American Medical Colleges, as well as the National Fund Organization.

In accepting the award, the President said he believes medicine "is one profession we don't want to get under the dead hand of bureaucracy." He said:

> "Quite naturally, I am very proud to receive an award from such an Association, from such a group. There is

an added distinction because it bears the name of one of our greatest professionals and our greatest citizens. On the other hand, Mr. Colt, I rarely felt quite so unworthy of receiving an award because my part in the organization of this National Fund was really getting someone else to do the work. It would be far more fitting this morning if I were presenting this to you, because you have been President of the Association from the beginning. But I can say this: I don't know of any group that is doing more necessary and worthwhile work than making certain that our medical schools have ample funds from private sources to keep running, because this is one profession we don't want to get under the dead hand of bureaucracy."

REINSURANCE TO BE REINTRODUCED

President Eisenhower has announced that he definitely plans to resubmit his health reinsurance plan to the next Congress, in spite of widespread opposition, not only from the AMA, but from much of the insurance industry, the U. S. Chamber of Commerce and many other groups.

In announcing his intentions not too long ago, the President said it would be "an important part of the health program in the great gaps in the field of health," adding that "we are years behind our potential achievement in the availability and adequacy of health services."

Mrs. Hobby, secretary of HEW, when reiterating the President's plan to resubmit the program, was asked about AMA's opposition and stated, "I very much hope that the AMA will see its way clear to support health reinsurance. We have the same objectives, the difference is only one of method." Incidentally, no additional pronouncement of policy on this matter was forthcoming from the Clinical Session of the AMA in Miami in December. Speculation was abroad whether or not the AMA may possibly have changed its mind on its opposition to reinsurance.

LABOR SECRETARY URGES EXPANSION OF HEALTH INSURANCE

Secretary of Labor Joseph P. Mitchell, joining in the public discussion of the administration's health and medical goals, advocates extension of health plans and continued development of medical education. According to a recent issue of the AMA Washington Letter, Mr. Mitchell made his statements in an address to the anniversary dinner of the Health Insurance Plan of Greater New York (HIP).

The Secretary said that great strides have been made in voluntary health insurance, providing "some degree of protection" to American families, but "much less has been done in terms of comprehensive health programs which provide complete medical and surgical care in the hospital and home."

Mr. Mitchell went on to say that there is no doubt in his mind that the American people are determined "to proceed on all fronts to establish adequate systems of health insurance."

Praises HIP

He also described HIP as "a good example of the development of a comprehensive system of medical care and hospitalization through a form of pre-payment which can be met by the average family." And, in addition to the widest possible medical care at the lowest possible cost, the country must have an adequate number of physicians, nurses and technicians as well as adequate hospitals, laboratories and medical research centers, Mr. Mitchell noted.

LARGE DOCTOR DRAFT CALL

More than twice the number of men taken in the last Doctor Draft call will be tapped for April induction, the Defense Department announces. This is presumably the last medical officer call under the present Doctor Draft Act, which is scheduled to expire on June 30, 1955. The Defense Department has asked Selective Service for 1,275 physicians—825 for the Army, 200 for the Navy, and 250 for the Air Force—and 459 dentists for the three-month period starting next April. The last quarterly Doctor Draft call, issued for December, 1954, took 550 physicians and 150 dentists. Priority III men (doctors not educated at government expense who have served no time on active duty) will comprise the majority of those taken in April.

Public Health

TRIBUTE TO TWO PUBLIC HEALTH WORKERS

Public health in Minnesota experienced an irreparable loss in the recent death of two women who spent their lives in devoted service to the people of this state. The Minnesota Department of Health desires to pay tribute in this section of MINNESOTA MEDICINE to the memory of Mrs. Kathryne R. Pearce and Miss Ann S. Nyquist. Mrs. Pearce, executive secretary of the Hennepin County Tuberculosis Association, died November 27, 1954, at the age of fifty-six years. Miss Nyquist, chief of the Section of Public Health Nursing for the Minnesota Department of Health, died December 8, 1954, at the age of sixty-six.

Mrs. Pearce was the wife of Naboth O. Pearce, M.D., until recently director of the Chronic Diseases and Geriatrics program of the State Health Department. Her work with the Tuberculosis Association began in 1918, when she was graduated from the University of Minnesota School of Social Work. She served as executive secretary of the Association for thirty-two years, from 1922 until her death in 1954. She was an officer or member of all Hennepin County organizations concerned with tuberculosis control, including the health council, the Tuberculosis Administrators, and Trudeau Society, and the Minnesota Rehabilitation Association. For twenty years, she served as member or chairman of the Committee on Rehabilitation of the National Tuberculosis Association, and in 1932 was president of the National Conference of Tuberculosis Workers.

The Board of Directors of the Association said of Mrs. Pearce:

> "Under her sincere and able direction, the Association's work has not only been responsible for saving many lives in Hennepin County, but it has served as a pattern, in many instances, for tuberculosis control measures throughout the nation. She was deeply concerned with the return of the tuberculosis patient to normal living and self-support. She helped to establish the national pattern for the rehabilitation of the tuberculous, making training for a future job a part of the treatment. She was alert to the many special problems of the tuberculous patient and his family—emotional and economic—and, to help solve these problems, worked with many individuals and groups in the community. The nation's fight against tuberculosis is proof of what consistent effort and hard work can accomplish. She was such a worker."

Ann Nyquist, too, spent her entire adult life in public service. Born in Renville County, Minnesota, she took her nurses training at St. Barnabas Hospital, Minneapolis. She was graduated in 1915 and served for the next three years there as anesthetist. During World War I, she served in a similar capacity at Base Hospital 26 in France. On her return, she studied public health nursing at the University of Minnesota, completing her course in 1920. Her first public health assignment was with the Red Cross in Sherburne County. Later, she served as public health nurse in Renville County.

In 1925, Miss Nyquist joined the Minnesota Department of Health as advisory nurse. In this position, her duties consisted of field supervision of local nurses and promotional work in the development of public health nursing services in Minnesota counties. In 1941, she was apointed director of public health nursing, a position she held until her death.

During World War II, she served as chairman of the Minnesota Procurement and Assignment Service for Nurses. In 1946, she received a citation signed by President Harry S. Truman "in recognition of uncompensated service to the United States of America during World War II." The citation reads: "She served with diligence and distinction in the interests of meeting both military and civilian needs . . . for the successful solution of complex and imperative wartime problems." In the same year she received a similar citation from the American National Red Cross.

In 1949, the Minnesota Department of Health, with the co-operation of the Hennepin County Tuberculosis Association and the Minnesota Division, American Cancer Society, produced the film, *Your Friend in Blue*. This film, portraying the work of a public health nurse, was dedicated "To a pioneer Minnesota public health nurse, Ann S. Nyquist." The film has been widely shown throughout the United States and has been used by the Voice of America.

Miss Nyquist had many public health interests. Among the chief were the Indian health program, rehabilitation of the disabled, meeting the needs of retarded children, and advancing the professional status of public health nurses. She was active in both the national and state League for Nursing, and during the last week of her life organized a fund drive for that group.

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Current Cardiac Concepts

DIAGNOSIS OF CORONARY HEART DISEASE

With the increasing recognition of the prevalence of coronary arteriosclerosis, doctors have become increasingly cognizant of the shortcomings of currently used diagnostic procedures. Obvious cases are readily recognized by their characteristic symptomatology and/or their specific electrocardiographic abnormalities. The electrocardiogram need not, however, be abnormal, and many cases show normal electrocardiographic pattern. Careful physical examination may disclose no abnormalities. Asymptomatic cases are, therefore, seldom recognized unless special diagnostic tests are made routinely or are indicated by certain non-specific abnormalities detected on routine physical examination.

There remain, nevertheless, many cases in which both clinical history and special diagnostic procedures are normal, and consequently, the clinical diagnosis of arteriosclerotic heart disease cannot possibly be made. It is this silent case that defies all known methods of detection which is an enigma to the physician. How often, unfortunately, is a patient given a "clean bill of health" only to have, subsequently, an acute myocardial infarction or, worse yet, to die suddenly from coronary heart disease which obviously was present at the time of the "negative" examination? It is because of this that one must be cautious to carefully evaluate vague, but suggestive, symptoms when they appear to plague one's diagnostic acumen.

On the other hand, it is equally important that the physician not be misled by a patient's vague chest symptoms into making a diagnosis of heart disease which does not exist. Important as it may be to make the cardiac patient aware of his disease and to teach him how to best protect himself, insofar as is possible, from its complications, so is it equally important to assure the patient who has no such disease that his symptoms are not cardiac in origin. No greater injustice can be done the individual with a normal heart than to mistakenly tell him he has heart disease, thereby coloring all his future actions and thoughts with the fear of imminent and unpredictable happenings. Few among us could accept with equanimity the diagnosis of coronary heart disease with all its implications; yet few among us would want *not* to be informed of its presence so that any prophylactic measures of value could be instituted. It is therefore of extreme urgency that the diagnosis, negative or positive, be as accurate as possible.

It is the presence of pain in any of the locations wherein heart pain may occur which usually raises the question of the possible presence of coronary heart disease. The clinical picture and electrocardiographic evolution seen in typical acute myocardial infarction *usually* make this diagnosis relatively simple, and other diseases with similar clinical symptoms are excluded with relative ease. On the other hand, with the shorter and generally milder pain of transitory coronary insufficiency (angina pectoris) the proper evaluation may be more difficult. *No* laboratory aid is as important in making this diagnosis as is a carefully elicited history. The location, character and radiation of the pain are often of help, but most important is the fact that this pain is resultant upon exertion (physical or emotional) and that it subsides promptly with rest or with nitroglycerine (although the latter occasionally relieves non-cardiac pains of smooth muscle origin). This pain does not generally occur at rest (except with myocardial infarction), and the fact that it is more easily precipitated after eating or on a cold or windy day is important diagnostically.

The reproduction of the pain or the production of electrocardiographic changes by the inhalation of oxygen poor air mixtures or the Master Two Step Test is of inestimable aid diagnostically if the result is positive. A negative test certainly does not exclude arteriosclerotic heart disease.

In recent years much study has been devoted to understanding the relationship of coronary heart disease to serum lipids in general and to serum cholesterol more specifically. Although research workers agree that the two are intimately related, this relationship is not yet clarified, and the level of serum lipids cannot be used diagnostically. Tests based on the relationship of the amounts of one type of lipid to another or on lipid patterns as determined by ultracentrifugation have not proved uniformly reliable. Authenticated cases of coronary heart disease with normal lipid amounts and proportions occur commonly.

It is entirely conceivable that someday a chemical or electrocardiographic test will be devised to detect the cases of "silent" coronary heart disease. There is also an increasing awareness of an apparent hormonal factor, and a diagnostic test along such lines may be forthcoming. In any event, no single consistently accurate test is now available, and it is on the carefully elicited and clearly evaluated clinical history that one must

(Continued on Page 76)

Contribution from the Minnesota Heart Association.

AMERICAN MEDICAL ASSOCIATION

Proceedings of the House of Delegates

EIGHTH CLINICAL MEETING

November 29 to December 2, 1954

Miami, Florida

Geriatrics, medical ethics, internships, grievance committees, hospital accreditation, osteopathy, the doctor draft law, state-subsidized medicine and malpractice insurance problems were among the major subjects of discussion and action by the House of Delegates at the American Medical Association's Eighth Clinical Meeting held November 29-December 2 in Miami.

During the meeting the AMA Board of Trustees also announced the appointment of a thirteen-member Commission to make a comprehensive survey of the various types of plans through which the American people receive medical services. The Commission, headed by Dr. Leonard W. Larson of Bismarck, North Dakota, member of the Board of Trustees, will begin work immediately and will require at least a year to complete its survey.

Named as the 1954 General Practitioner of the Year was Dr. Karl B. Pace of Greenville, North Carolina, whose selection by a special committee of the Board of Trustees was announced at the opening session of the House of Delegates on Monday by Dr. Dwight H. Murray of Napa, California, Board Chairman. Dr. Pace received the medal and citation, presented annually for community service by a family doctor, from Dr. Walter B. Martin of Norfolk, Virginia, President of the American Medical Association, immediately after the announcement.

Other highlights of the opening session were addresses by Dr. Martin; Seaborn P. Collins, National Commander of the American Legion; Mrs. Oveta Culp Hobby, Secretary of Health, Education and Welfare; Edwin J. Faulkner, President of the Woodmen Accident and Life Company of Lincoln, Nebraska.

Mr. Collins told the House that he is willing to appoint qualified Legion representatives on a committee to take part in joint Legion-AMA study of veterans' hospitalization. Later during the meeting the Board of Trustees announced appointment of a three-man committee to meet with the Legion on the issue of veterans' medical care. The members of the AMA committee are Dr. Elmer Hess, Dr. David Allman and Dr. Louis Orr.

Registration toward the end of the third day of the Clinical Meeting included 3,167 physicians; 3,441 guests including residents, interns, nurses and others, and approximately 900 exhibitors and exhibitors' guests—for a grand total of more than 7,500. Final total registration at the 1953 Clinical Meeting in St. Louis was 7,716.

New AMA Geriatrics Unit

The House of Delegates passed a Pennsylvania resolution which directed that the AMA Board of Trustees "consider the creation of an organization on geriatrics within the present structure of the American Medical Association, the purpose of which shall be (1) to develop and assist committees on geriatrics and gerontology originating from constituent state associations and component county societies of the American Medical Association; (2) to act as a liaison between such state and county committees so there shall be a free flow of information between all levels of organized medicine on the subject of geriatrics; (3) to make available to the American people such facts, data and opinions concerning the subject of geriatrics as may be considered of value in alleviating social and medical problems created by the increasing population of older age groups; and (4) to perform such other duties as will improve and advance the medical care rendered to people of the older age group."

Medical Ethics

Accepting a recommendation in a report of the Council on Constitution and Bylaws, the House amended Section 7 of Chapter I of the Principles of Medical Ethics so that it now reads as follows on the subject of patents and copyrights:

> "A physician may patent surgical instruments, appliances and medicines or copyright publications, methods and procedures. The use of such patents or copyrights or the receipt of remuneration from them which retards or inhibits research or restricts the benefits derivable therefrom is unethical."

In another action involving medical ethics, the House rejected a Kansas resolution which would have removed Section 8 of Chapter I from the Principles of Medical Ethics. The Reference Committee on Miscellaneous Business, in recommending disapproval of the resolution, said:

> "The American Medical Association would fail to assume a vital responsibility if no provision is included in the Principles of Medical Ethics regarding the problem of ownership of drug stores and dispensing of drugs by physicians. . . . It is possible that some phases of this principle are susceptible of amendment or change, but certainly the entire principle should not be discarded."

Report on Internships

Acting on the report of the Ad Hoc Committee on Internships, the House accepted a recommendation of the Reference Committee on Medical Education and Hospitals that "the data and judgments of the Ad Hoc Committee on Internships will provide valuable guidance to the Council on Medical Education and Hospitals and with this in view it is recommended that the report be referred to the latter for their further study and guidance." Following are a few excerpts from the report of the Ad Hoc Committee on Internships:

"It is our opinion that graduates of foreign medical schools should be considered for intern appointment in approved hospitals only when there is satisfactory evidence that:

"1. Language difficulties will not seriously impair the program.

"2. The same educational standards are applied to graduates of foreign schools as to graduates of approved American medical colleges.

"3. The appropriate state licensing board approves. . . .

"The Committee believes that the present standards detailing only the number of annual admissions, autopsy rate, number of beds and assignment of an intern to from 15 to 25 beds, are without significant meaning unless and until every local situation is reviewed 'on the grounds' and with full opportunity for discussion between the representative of the accrediting body and representatives of the hospital's governing board and its medical staff. . . .

"Had the 'two-thirds rule' remained a requirement and been rigidly applied to the two consecutive intern years 1952-3 in combination with 1953-4 it would have removed 448 hospitals, cancelled 4,205 internships to which 784 students were matched in those years and reduced the number of internships available to 6,766. . . .

"The committee suggests consideration of some requirement based on filling a percentage of approved internships and a time limit to eliminate some of the unhealthy aspects of the present situation. The following requirement is recommended: Any internship program which in two successive years does not obtain one-fourth of its stated intern complement be disapproved for internship training.

"As applied to the figures for 1952-3 in combination with 1953-4, this requirement would have removed 277 hospitals, cancelled 2,139 internships to which 80 students were matched in those years and reduced the number of internships available to 8,832."

Grievance Committees

In order to improve efficiency and maintain high standards in the operation of grievance or mediation committees, the House endorsed the principles of two similar resolutions introduced by the Colorado and Mississippi delegations and asked the Board of Trustees to appoint a committee to study and report on recommended standards for the operation of such services. Both resolutions had emphasized the valuable public service aspects of grievance committees and had suggested that the committee appointed by the Board of Trustees be composed of representatives from constituent societies in which grievance committees have been effective and useful.

Hospital Accreditation

In place of an Indiana resolution protesting certain situations arising in connection with hospital inspections, the House adopted the following substitute resolution to resolve the problems in question:

"Resolved, that the Secretary of the American Medical Association be directed to request that the Joint Commission on the Accreditation of Hospitals supply a copy of the letter of notification regarding the results of the survey of each hospital to the Hospital Administrator, to the Chief of the Professional Staff and to the Chairman of the Governing Board of the hospital."

Osteopathy

The House concurred in the following supplementary report of the Board of Trustees on the osteopathic situation:

"Contingent on the receipt of the report from the Committee to Study the Relations Between Osteopathy and Medicine of its 'on campus' observations of osteopathic schools, the House of Delegates in June, 1954, agreed to hold in abeyance any action on this important subject until this meeting.

"The Committee, after meetings and extensive negotiations with the American Osteopathic Association, has now made final arrangements for visiting five of the six schools of osteopathy, and these plans have been approved by the Board of Trustees.

"It is the recommendation of the Board, therefore, that consideration of this matter be held in abeyance by the House of Delegates until the June, 1955, meeting, at which time the Committee expects to have a complete report of its findings concerning the nature, scope and quality of education in schools of osteopathy."

The Doctor Draft Law

The Reference Committee on Medical Military Affairs considered several reports and resolutions involving the doctor draft law, and then proposed the following policy statement which was adopted by the House of Delegates:

"(A) That on the basis of current information the House of Delegates commend and express itself as being in complete accord with the Board of Trustees and its Council on National Defense that the 'Doctor Draft Law' should not be extended after June 30, 1955, and that the House of Delegates further express its confidence in the ability of the Board of Trustees and its Council on National Defense to properly handle any new situation which may develop in regard to this highly complex and involved problem.

"(B) That the Board of Trustees and its Council on National Defense continue to study the problem of providing the best possible medical service for members of the armed forces and that they make recommendations to the Department of Defense at the earliest possible time for a more permanent solution to the problem, giving special attention to the further development of a career medical corps with adequate compensation therefor."

State-Subsidized Medicine

Most controversial issue at the Miami meeting was a resolution on "Policy on Medical Practice by Tax Supported Medical Schools," introduced by the Mississippi State Medical Association. This resolution provided that:

> "The American Medical Association reaffirm its unalterable opposition to socialized and state subsidized medicine regardless of the form which it may assume, and
>
> "The House of Delegates of the American Medical Association is of the opinion that these principles should be considered by constituent and component medical societies together with all other facts pertinent to the local situation in all controversies arising in the employment of medical faculty by state (tax) supported medical schools and be fully considered in effecting action within the framework of this policy."

The Reference Committee on Medical Education and Hospitals agreed with that portion of the resolution regarding "unalterable opposition to socialized medicine" but recommended that the resolution be referred, without approval or disapproval at this time, to the Council on Medical Service which currently is studying the various aspects of this subject. The House adopted the reference committee's recommendation.

Eisenhower Reinsurance Plan

Time is not running out for those who believe in the voluntary way toward insurance protection, Edwin J. Faulkner, president of the Woodmen Accident and Life Company of Lincoln, Nebraska, told the delegates. Public opinion surveys show a substantial decline in the number of people who favor compulsory health insurance, he said.

Mr Faulkner also pointed out that a federal reinsurance program will not help private companies to sell unwilling buyers. Nor will it make insurance any cheaper or more easily available to persons or areas which cannot now be reached by voluntary insurance companies. What it will do, in his opinion, is this:

1. To the extent that it encourages extending health insurance to insurable risks, it will result in excessive losses leading to pressure for direct federal subsidies.

2. It will involve a very broad delegation of powers which some later administration might abuse.

3. Participating insurance companies will be subject to government regulation of premium rates on reinsured business. Because of the rapidly changing pattern of health care costs insurers need a maximum flexibility to keep abreast of developments.

Malpractice Insurance

Two resolutions and a Board of Trustees supplementary report—all dealing with the problems and difficulties in obtaining satisfactory professional liability insurance—were considered together by the Reference Committee on Insurance and Medical Service. The House of Delegates accepted the reference committee report which said: "Inasmuch as the Board of Trustees has reported that there is in progress a study on the subject, we feel that we can well await the recommendations that the Board is planning to make at the next session. Due to the apparent emergency aspect of the problem, the Board of Trustees is urged to report to the membership as soon as possible, through its component societies, on the progress of this urgent study."

Opening Session

Walter B. Martin, M.D., AMA President, declared at the opening session that "medicine belongs to the people" and physicians are "merely the purveyors" of medical care. Dr. Martin stressed that physicians have an obligation to the people that "goes beyond our own private practice and into the community," and he also emphasized the importance of "continued effort to meet the medical needs of the low-income and other non-insurable groups."

Mr. Collins, the American Legion National Commander, said that "we are citizens first and doctors and veterans second," as he urged removal of the veterans' medical care issue "from the area of name-calling and propaganda." The American Legion, he declared, neither expects nor wants the Government to give carte blanche entitlement to medical care to all veterans.

Mrs. Hobby, presenting the case for the Eisenhower Administration's health reinsurance proposal, said: "The health reinsurance proposal represents what we believe to be a necessity. It offers opportunity for self-help without subsidy." Mr. Faulkner, however, expressed the opinion that the reinsurance program, "would be foredoomed to disappoint its proponents," and he declared that voluntary health insurance can bring satisfactory protection "to practically all of our people" without a Federal reinsurance program.

Awards and Contributions

At the closing session of the House of Delegates the American Medical Association received a citation for pioneering in helping to bring educational television to the American public. James Keller, chairman of the Miami Citizens Committee for Educational Television, presented the award on behalf of the National Citizens Committee for Educational Television. Dr. Martin accepted the citation for the AMA.

At the same session the Utah State Medical Society, represented by George M. Fister, M.D., of Ogden, presented a check for $10,355 to the American Medical Education Foundation to aid in relieving the financial plight of the nation's

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History of Medicine in Minnesota

THE PIONEER DOCTORS OF CHIPPEWA COUNTY PRIOR TO THE YEAR 1900

LEON G. SMITH, M.D.

Montevideo, Minnesota

(Continued from October, 1954, issue)

It was upon Frink's farm, homesteaded in 1867, that much of Montevideo was built. What finally determined the future of the two villages, Chippewa City and Montevideo, was the location of the post-office, and that was brought to Montevideo by the superior foresight of its citizens. The mail was brought up the old Military Trail on the south bank of the Minnesota; and, since Montevideo was the first to build a ferry across the Minnesota below the mouth of the Chippewa, the mail was then brought up along the east bank of the Chippewa, with the natural result that they moved the post-office to the Montevideo side rather than transport the mail further across the river to Chippewa City.

The village was platted in 1870, the year in which the post-office, a schoolhouse and a hotel were built. The sawed lumber for the post-office was hauled 100 miles, but the roof was made from bark, stripped from elm trees and then covered with dirt. The floor was of dirt, with a thick carpet of hay, necessitating rather high stepping. Counters in stores consisted of posts driven into the ground with boards laid across. On February 28, the same year, Montevideo was designated the county seat by action of the Territorial Legislature, and this spelled "finis" to Chippewa City.

Only two years later we come across the name of Montevideo's first physician, Dr. Murphy.

Dr. Lea Murphy was born in Wisconsin, November 27, 1849. His family consisted of his parents, three brothers and nine sisters. When he was seven years old, the family moved to St. Charles, Minnesota, where he attended public school and became interested in medicine, reading the medical books of his Grandfather Sedduth. He entered Rush Medical College in Chicago in 1871. On October 8 to 9 of that year the famous Chicago fire occurred, and he had to walk for miles hauling a little cart, on which was his trunk, containing his books and other student equipment.

In the summer of 1872, he drove to Montevideo by horse and sulky with what little medical equipment he owned, a pair of saddle-bags, and twenty-five dollars in cash. At that time, his nearest competitor was located at Redwood Falls, and his territory extended from Benson to the Dakota line, west of Canby. Two years later, Dr. Stratton located at Granite Falls.

Requirements for medical practice were not at all rigid at the time, and he was able to practice without a license. In fact, he was probably better equipped for his work, by his year in medical school, than many other doctors who had located in the new country. Many of them had picked up the rudiments of medicine and surgery, working as surgeon's aids, or hospital attendants during the Civil War, and then had simply set themselves up as physicians and surgeons.

Apparently his practice was a lucrative one, because within a year he had made enough money to return to medical school and complete his studies, graduating from Rush Medical College in 1874, and returning to Montevideo.

In 1875 he was married to Ruth Helen Lawrence, and three children were born to this marriage: son, Lloyd L., in 1876; daughter, Nelbert, in 1878; and, son, Lea M., in 1885.

A law passed in 1883, required practitioners of medicine to obtain a license. It exempted those who had practiced in the state for five years. (Four years later in 1887, the legislature passed a more stringent law, repealing the law of 1883 and prohibiting anyone from practicing medicine without a license under penalty of fine, or imprisonment, or both).

Evidently, Dr. Murphy practiced for nine years without one, none being required or, presumably, even obtainable. But October 11, 1883, he obtained his license, Number 45, making him one of the earliest licensees in the state.

In 1878, he developed a very severe case of rheumatic fever which incapacitated him for many months. Probably, as the result of this illness, and the necessity for avoiding some of the more strenuous duties of medical practice, he entered the drug business with O. A. Griffis. His brother, Will, who worked as a clerk in the drug store for several years, eventually bought out Mr. Griffis, bringing the drug store under the ownership of the two Murphys, and under the name of the Murphy Drug Store, it continued in active business until 1921.

Old files of the *Montevideo Leader* (December 9, 1882) record his performance of a difficult operation upon J. J. Skonard, working with Dr. Rogers. "After the patient was chloroformed, an extensive canal was found, leading from an opening on the left side of the neck as far as the angle of the jaw. The dissection was made in the region of important blood vessels and nerves, which had to be carefully avoided, as a little slip might have proved a very serious matter. Three hours of skillful manipulation brought out the sac entire, known among surgeons as a dermoid cyst. We understand the young man is doing well and the doctors think he will have no further trouble." December 31, 1886, mention was made of a new phone connecting his house and office, which must have been one of the first instruments in the county. On May 13, 1887, there was an account of his performing a difficult operation, assisted by Drs. Rogers and Moyer, upon Magnus Quamen of Milan, wherein "a large, fistulous tract, draining a necrotic area of the coracoid, as well as the acromion, process of the scapula, leading across the brachial plexus, and discharging upon the skin of the upper arm, was opened, the necrotic bone chiselled out, and the wound dressed. At present account the patient is doing well."

He spent the winter of 1889 on the Pacific Coast, and in 1890, he "packed his household goods and shipped them to Minneapolis, where he moved with his family, and assumed his new duties as Medical Examiner and Manager of the Mount Benevolent Insurance Association. The community regrets their leaving after almost twenty years among us."

March 16, 1894, he was in Montevideo forming the Globe Fraternal Accident Association, which insured the I.O.O.F. (Independent Order of Odd Fellows) only.

He must have shuttled back and forth between Minneapolis and Montevideo two or three times in the next few years, because the *Montevideo Advance* of

June 5, 1896, announced that "Dr. Murphy's goods and family have arrived, and they are looking for a place in which to live."

Shortly thereafter, he bought a sanatorium, for tuberculosis and other respiratory diseases, in Minneapolis. The *Montevideo Commercial* of November 19, 1897, has an article as follows: "There was a bad fire in Dr. Murphy's Sanatorium a few days ago, caused by a burning nebulizer. Dr. Murphy, in his heroic efforts to prevent the fire's spread, and in throwing the nebulizer out of doors, sustained severe burns of the hands and arms. Twenty-two patients were moved to Dr. Rogers' Sanatorium." (Dr. Rogers was a former confrere of Dr. Murphy in Montevideo, and at the time, was running a similar institution in St. Paul.)

In 1899, he had again returned to Montevideo and, in 1900, he took several months of post-graduate work at Rush Medical College, following which, he moved his family to Badger, Roseau County, Minnesota, practicing medicine and conducting a drug-store there for a few years. He moved back to Montevideo in 1906, and again assumed an interest with his brother in the Murphy Drug Store, which he retained until 1919.

It was during this period that I became acquainted with him. He was a delightful man to know, full of wit and humor, and with a gift for anecdote. I greatly enjoyed his stories of his pioneer days. The one that especially impressed upon me the arduous work he had formerly done concerned a prolonged typhoid epidemic one fall. He had so many cases, extending all the way to Benson (over thirty miles), that it would take him all of a day, driving across the prairies and around the sloughs to visit half of them. After spending the night in Benson, the next day, on his return trip, he would visit the remainder. Far into the night he did his work in Montevideo, starting out again the following morning upon a repetition of his two day trip. This epidemic lasted for several weeks. Small wonder he was ready to turn to the less strenuous work of running a drug store.

He moved to Pasadena, California, in 1919 and died there in 1923. His older son, Lloyd, and his daughter, Nelbert Murphy Chouinard, still live in the Los Angeles area.

I have attempted to assemble the biographical sketches of the doctors, who succeeded Dr. Murphy, as nearly as possible in the chronological order in which they located in the county. There were apparently several who settled here for a short time in the 1870's and early 1880's, but information concerning them is meager.

Probably the first to locate after Dr. Murphy, was **Dr. Charles E. Daniels.**

In the *Montevideo Commercial* of August 7, 1887 there appeared, copied from the *Milbank Review,* a story concerning Dr. Daniels, at that time located in Milbank, South Dakota: "Dr. Daniels was born about 1840, and while still only a boy, enlisted in the U. S. Army, February 17, 1865. He was in Company D of Bracketts' Indian Battalion, serving under General Sully. During the winter of 1865-66, he acted as scout, chasing Indians, trapping beaver and hunting buffalo all through the region extending from what is now Sioux Falls to Milbank. After his discharge in May, 1866, he returned to Hastings, Minnesota, to finish school, and then went on to college at Hamline University in St. Paul and at Bennett Medical College in Chicago. After graduating from the latter, he located in Goodhue County in 1876, moved to Montevideo, Minnesota in 1878, and to Milbank, South Dakota in 1881." (A professional card in the *Montevideo Leader* of January 14, 1882, but gone by the issue of July 1, 1882, would indicate he moved from Montevideo to Milbank during early 1882.) "He is now 40 years old, is president of the Milbank Board of Health, and has enjoyed a lucrative practice for several years."

Evidently Dr. Daniels lived and practiced in Milbank the remainder of his life.

Another doctor who practiced in Montevideo for a short time during this period was **Dr. George E. Dennis.** He was born in Livonia, Michigan, November 27, 1839, and grew up there. He served in the First Michigan Cavalry during the Civil War. In 1868, he moved to Hastings, Minnesota, where he engaged in farming until 1879. He had begun the study of Medicine at home and later worked under the preceptorship of Dr. Hawes of Hastings.

There is no record of any acquaintanceship between him and Dr. Daniels, but both living in a small town the size of Hastings, it is reasonable to assume they were friends and that, when Dr. Daniels decided to leave Montevideo, he induced Dr. Dennis to come here and take his place. At any rate, Dr. Dennis came to Montevideo in 1880 and practiced here until he made enough money to complete his medical education as a homeopathic physician, graduating from Hahnemann Medical College of Chicago in 1883.

His Minnesota license, number 44, dated October 11, 1883, was a very early one.

The *Montevideo Leader* of October, 1882, stated: "Dr. G. E. Dennis leaves for the winter at Hahnemann Medical College, expecting to return in the spring. He has been here for two and a half years and has built up a fine practice, and we regret his temporary absence."

After graduation, he returned to Montevideo, but for only a short time, as he moved to Minneapolis in 1884 and practiced there until his death May 22, 1907. He was one of the better known doctors of Minneapolis during that time, and there are several biographical sketches of him. The best is in a "History of the First Presbyterian Church of Minneapolis," by A. B. Marshall.

Dr. Joseph B. Bacon located in Montevideo at the same time as Drs. Murphy, Daniels and Dennis. He remained for a somewhat longer period, but is not to be confused with **Dr. R. S. Bacon,** who came to the village about the time Dr. J. B. Bacon left, staying here for many years.

Dr. J. B. Bacon was born in Illinois in 1854. He attended Northwestern College of Chicago until 1876, Chicago Medical College until 1878, and the next year graduated from Texas Medical College. He practiced one year at Elliott, Illinois, and was then appointed assistant physician in the Cook County Hospital. Apparently, he did more medical studying during that time, as he is again listed as graduating from Chicago Medical College in 1881. He did not obtain his Minnesota license until December 21, 1883, so he, also, was one of those doctors who practiced without a license prior to the License Law of that year.

He was mentioned in several news items of the Montevideo papers up to the late 1880's. The *Montevideo Commercial* of May 9, 1892, had the following item: "Dr. J. B. Bacon, once a practicing physician here, is now a resident of Chicago. He has associated himself with several other physicians to start a medical school and hospital. Instead of lectures and a dissecting room, students will be given actual work upon patients under competent direction. All demonstrative work will be done at the bedside and classes will consist of but six students."

Dr. Bacon was listed in Polk's Medical Directory as being in Chicago in 1895 and 1898, but in 1903, he was listed from Macomb, Illinois, his name reappearing from there in each issue until 1927. The American Medical Association recorded his death July 21, 1936, with place of residence or death not given.

The listing of the "Causes of Death" for Chippewa County for the year 1881 makes interesting reading, by comparison with the present, with eighteen deaths under one year of age, ten between the ages of one and five, and only six over the age of sixty. This forcefully points up the change that has taken place in our mortality statistics as our country has grown older, showing an extremely high rate among infants and young children and a very low rate among older people in this report.

Causes of Death	*Number of Cases*
Diphtheria	13
Consumption	6
Typhoid fever	4
Cholera infantum	2
General debility	2
Spasms	2
Heart disease	2
Accidents	2
Various diseases	10
Not known	15

Accidents seem to have occurred at this time as frequently as at present. Accounts of injuries sustained by run-away horses, or of farmers being gored by bulls appeared in place of our present-day automobile smash-ups or corn-picker tragedies. And, of course, such terms as "black smallpox," "black diptheria," "typhlitis," "membranous croup" and "pest-house" sound strange to us now.

Dr. Ottul Klaranus Lindboe was located in Chippewa County for only a short time, but he was such an interesting person and he lived for so many years near the village of Lac qui Parle, that he was close enough for Montevideo to claim him as a member of our community. He was born November 16, 1851, in Konsberg, Norway, the son of Andere and Else Lindboe. He there lived until after his confirmation, when he moved to Christiania and worked by day and attended school at night.

After his father's death, his mother, who was a doctor, came with him and his brother to America in April, 1867. The trip took eight weeks and whatever food they needed on the journey they had to prepare themselves. They landed in Quebec and traveled from there to Sarnia, Ontario, overland by ox-cart. Crossing into the United States at Detroit, they took a boat to Chicago, where he worked as a painter during the day, attending night school long enough to learn to speak English.

In July, 1867, he set out with his uncle for the gold fields of Colorado, where he stayed a year and was fortunate enough to pan out about $5,000. On his return trip, however, he was not so fortunate, because, carrying his money and belongings in a knapsack and sleeping out doors, he awoke one morning to find himself robbed. Reaching Chicago penniless, he found his mother and brother Andrew had bought a drugstore. There he worked, and did painting and helped clean up the debris after the Chicago fire. He wanted enough money to secure an education, so he joined a party and set out upon a second trip to the gold fields. Again misfortune struck. The party was attacked by Indians for nine days and they finally had nothing to eat but raw mule meat. Lindboe was wounded, and for the rest of his life carried the scars of an Indian arrow in his leg and a bullet in his side.

Upon his return to Chicago, he studied pharmacy, and graduated in 1873. But he at once went on to study medicine at Northwestern University for one year, then at Rush Medical College, graduating in 1877. His brother, Andrew, graduated from the same school shortly thereafter, and the two started practice together, but Andrew soon contracted tuberculosis and died. After that, Ottul and his mother practiced a short time in Ironwood, Michigan. He came to Minnesota, practicing a short time in the vicinity of Red Wing and Cannon Falls, and, in 1879, was married to Anna Christina Johnson, of Beloit, Wisconsin.

The following year, he and his bride came to Montevideo, but remained only a short time before moving to Lac qui Parle Village, which, at that time, was one of the largest settlements in western Minnesota and reputed to have a population of over nine hundred.

During the winter of 1881, known as the "year of the big snow," Dr. Lindboe had to make his professional calls on snow shoes and skis, the snow being four feet deep on level ground. In later years, Dr. Lindboe liked to reminisce about that first winter, when people lived in sod houses with snow piled so high over them, that the only sign of life was smoke rising from the chimneys, which barely protruded above the drifts. Tunnels were dug between house and barn; slanting shafts were dug down to the house door in order to get in and out; and holes were cut in the barn roof to enter and care for the stock. On several occasions he came home from his calls with frost-nipped hands or feet or face.

After some years of practice with his office in Lac qui Parle Village, Dr. Lindboe began to suffer a great deal from rheumatism. Because of this he bought a farm a short distance north of the village. This he worked as a farmer, but he also administered to the medical needs of his neighbors for many years, and, in the absence of a dentist in the area, extracted many aching teeth. It is interesting to mention that he brought the late Governor Theodore Christianson into the world.

During his life in the community, he served for thirty years on the school board, and many terms on the township and health boards. He was for many years a member of the board of directors of the four-county Riverside Tuberculosis Sanatorium located near Granite Falls.

He was taken sick in January of 1942 and after a three weeks illness passed away February 7, 1942, at the age of ninety.

Dr. and Mrs. Lindboe had five children: one son, Andrew; and, four daughters, Ella (Lund), Clara (Anthony) and twins Elsie and Laura (Dahl). Mrs. Lindboe and Ella had preceded him in death, but his remaining four children, with many grandchildren and great-grandchildren, are living, many of them still in the Lac qui Parle area.

Prior to the year 1881, the only doctors who had located in Chippewa County had settled in Montevideo, but in that year **Dr. Nels Amherst Nelson** began practice in the village of Watson.

Dr. Nelson's parents were born in Norway: his father, Nels Bakken in Narvik, and his mother in Christiania. Nels was born in Lake Mills, Iowa, in 1858, one of a family of six children. He attended elementary school at Lake Mills and college at a small institution in Wisconsin, which was probably Ripon College. His medical education was obtained under the Eclectic School of Medicine in Eclectic Colleges in St. Louis, Missouri, and in Minneapolis, Minnesota. He also attended a post-graduate course at Rush Medical College in Chicago, Illinois, and graduated in 1880.

He moved to Watson, Minnesota, and during the next two years practiced in Chippewa County at Watson and Milan. Later he located in Appleton, Minnesota.

In 1880, he was married to Jennie Boe, a girl who lived a few miles east of Milan. She was born in Lier, Norway, in 1858, and had moved to this country with her parents when she was thirteen years of age, and was living near Milan when she met Dr. Nelson. Two children, a son and daughter, were born to their marriage.

Polk's Medical Directory states that Dr. Nelson graduated from the Eclectic Medical College of Des Moines, Iowa, in 1884, but this may be an error, because the *Montevideo Leader,* of September 16, 1882, recorded, "Dr. Nelson was called from Appleton to perform the amputation of an arm. He was assisted by Dr. J. B. Bacon and Nels Bergstrom." There is, of course, the possibility that, like many another physician of his day, he practiced at intervals, interspersed with periods of study in medical school. In any event, graduate or not at the time, he must have attained a considerable degree of skill to be able to do an amputation.

He received his Minnesota license June 25, 1885, and, at about that time, moved from Appleton to Dawson, Minnesota, where he practiced until his death in August, 1929.

He was one of the earliest settlers in that village, moving there at the time the Minneapolis and St. Louis Railroad was extended through Dawson. His residence was the third house built in the town.

For most of his life, he practiced alone. He was a life-long member of the Lutheran Church, and for many years a member of the Masonic Lodge. He also was a charter member of the Camp Release District Medical Society, and attended its meetings regularly and did a great deal of community welfare work. For many years he was village Health Officer. He had a wonderful bedside manner with his patients.

His outside interests were in hunting and fishing, and he was especially fond of violin music, of playing the violin himself, and of singing.

One sister and two brothers survive him, as well as his son and daughter. His son, Dr. Melvin S. Nelson, has been practicing medicine in Granite Falls, Minnesota most of his professional life, and is one of the oldest and best known doctors in western Minnesota.

Dr. Hans J. G. Koren is another physician who appears to have been practicing in Montevideo at the time of his licensure, December 28, 1883. He was born in Norway, and received his medical education at King Frederick University, graduating in 1869. He must have practiced in Norway for several years, because his son, Finn, later to become a Chippewa County doctor, was listed as having been born in Norway in 1873. Some time between 1873 and 1883, Dr. Koren moved his family to this country and settled in Montevideo, where he practiced, probably until Finn graduated from Windom Institute in the class of 1891. He and Mrs. Koren must then have returned to Norway for several years, because in the *Montevideo Commercial* of November 12, 1897, we find this item: "Dr. and Mrs. H. J. Koren, formerly of this village, who have been living in Norway for several years, are returning to the U. S. They will locate in Lamberton, Minnesota."

The *Montevideo Leader* of November 3, 1899, had the following: "Dr. H. J. Koren, formerly of this village, but later of Lamberton, died October 31, 1899, at St. Barnabas Hospital, Minneapolis, Minnesota, of appendicitis and resulting peritonitis, after six days' sickness. Pallbearers will be classmates from the University of Norway. Mrs. Koren will reside with her son, Finn, who is studying medicine at the University of Minnesota."

We next come to one of the most interesting personalities we will encounter in the history of the county, **Dr. Charles E. Rogers.** All the evidence seems to indicate that he was a man of strong convictions, boundless ambition and unlimited energy. There were no half-way measures with him. People were either his very good friends or his enemies. He lived and practiced off and on in Montevideo for thirty years, and during that time, exerted a powerful influence upon the community.

Charles Emory Rogers was born in Jefferson, Ohio, October 1, 1850, the son of Henry and Melinda Hoyt Rogers. Both of his parents were born in New York. When a lad, his family moved to Mauston, Wisconsin, where he obtained his elementary schooling. He followed this, by attending business college, teaching country school for a while, and later becoming part owner and teacher in a business college in La Crosse, Wisconsin. He was married to Rose Turner, of Mauston, at Wisconsin Dells in 1870.

He remained at La Crosse several years, and during that interval took up the study of medicine under the tutelage of Dr. Knut Hoegh, at that time, one of the best known surgeons west of Chicago. This was followed by a regular course of medical study at the Bellevue Hospital Medical College in New York City. After graduating in 1880, he returned to La Crosse and became associated with Dr. Hoegh. During this time, he was made a surgeon in the Third Regiment of the Wisconsin National Guard, with the rank of major.

An old boyhood friend from Mauston, H. E. Hoard, in the meantime, had settled in Montevideo, and had bought the *Montevideo Leader,* of which he was manager and editor. Later he was elected to several terms in the State Legislature.

Undoubtedly, as the result of this friendship, Dr. Rogers was induced to locate in Montevideo, which he did in the late fall of 1882.

Almost immediately he became a leader in the community. He was appointed surgeon of the C. M. and St. Paul (Milwaukee) Railroad, in which capacity he served until his death. He was a member of the State Medical Board for many years and for a number of years, was a teacher of anatomy, physiology and hygiene in Windom Institute. At various times he was a member of the village council and a director of the Citizen's State Bank.

(To be continued)

THE DEAN'S PAGE

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medical practice. The return of the Medical Library to the Medical Campus will make this possible. We most earnestly hope, therefore, that the 1955 Legislature will make the appropriation requested for this purpose.

Additional funds for remodelling and equipment of the University Hospitals and Mayo Memorial are needed because large areas, such as the operating rooms, x-ray department, clinical and research laboratories and departmental and administrative offices were removed from the hospital into the new building. The areas thus vacated need to be remodelled into rooms and wards for patient care. In addition, much of the plumbing and electrical work in this old building needs replacement, and considerable areas of the Outpatient Clinic need reconditioning and modernization.

The medical faculty will appreciate any assistance in the support of these appropriations which the physicians of the state are able to give.

Harold S. Diehl

In Memoriam

WILLIAM H. GOECKERMAN

Dr. William Goeckerman, member of the Mayo Clinic staff from 1921 to 1932, died November 8, 1954, in Los Angeles. He was seventy years old.

Dr. Goeckerman was born in Hanover, Germany, and received his medical degree from the Wisconsin College of Physicians and Surgeons in Milwaukee. He entered the Mayo Clinic as an assistant in dermatology and syphilology in 1917 and became an associate in 1921. He was also associate professor of dermatology in the Mayo Foundation. He practiced in Los Angeles upon leaving Rochester and was consultant at St. Vincent's Hospital and the California Hospital and clinical professor of medicine at the University of Southern California.

During his stay in Minnesota, Dr. Goeckerman was a member of his county medical society and of the Minnesota State Medical Association and the American Medical Association. He was also a member of the American Dermatological Association and many other scientific associations.

Surviving are his wife, the former Magdelana S. Moore, and several brothers.

JOHN FRANCIS NORMAN

Dr. J. F. Norman, of Crookston, died at the age of seventy-four in his home at Crookston on November 29, 1954. Dr. Norman was founder of the Crookston Clinic and was still actively associated with it. He died suddenly after a cerebral hemorrhage which occurred while he was putting on his overshoes to return to the clinic at noon.

Dr. Norman had been a leader in medical affairs in Minnesota for many years. He served as president of the Minnesota State Medical Association in 1951 and had been elected to all of the offices that his local and district medical organizations could bestow.

He was a member of the board which established the Blue Shield program in Minnesota and was a member of the American Medical Association as well as his local Red River Valley Medical Society and the state association. He was also a long-time member of the American College of Surgeons and a member of Rotary.

Born in Henderson, Dr. Norman taught school for five years in order to earn funds to go to college. He was graduated from Hamline University Medical School shortly before the school became part of the University of Minnesota Medical School in 1905. He served his internship at St. John's Hospital in Fargo, North Dakota, and later did some postgraduate work in Europe.

He is survived by his wife, the former Loretta Malone; two sons, Dr. David D. Norman, 1883 Goodrich Avenue, St. Paul, and Nelson F. Norman, Ph.D., Fresno State College, California; a daughter, Mrs. H. R. Perry, Santa Fe, New Mexico; a sister, Mrs. Mae Working; and seven grandchildren.

WILLIAM HENRY ROWE

Dr. William H. Rowe, seventy, died December 2, 1954, in Fairmont where he had practiced eye, ear, nose and throat work since 1935.

Dr. Rowe was the son of a physician who practiced in St. James, where he was born in 1884. He went to school in St. James and obtained a bachelor of arts degree at the University of Minnesota and a medical degree from Rush Medical College of Chicago in 1909. He served his internship at St. Mark's Hospital in Salt Lake City and took a year of post-graduate work at Knapp Memorial Hospital and Roosevelt Hospital in New York City. He practiced at St. James from 1910 to 1922 and in Minneapolis from 1922 to 1934.

In 1935 Dr. Rowe moved to Fairmont and remained there until his death.

AMERICAN MEDICAL ASSOCIATION

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medical schools. The contribution was received by Louis H. Bauer, M.D., president of the foundation, who also announced that a check for $1,000 had been contributed by the Southern Medical Association.

1957 Clinical Meeting

Philadelphia was chosen as the place for the 1957 Clinical Meeting, the dates of which will be announced later. Invitations also had been received from Denver, Detroit, Mexico City and Washington, D. C. The Clinical Meeting will be held in Boston in 1955 and in Seattle in 1956.

Health Fair

As the AMA Clinical Meeting came to a close on Thursday, December 2, a health fair for the public opened in Miami's Bayfront Auditorium under the auspices of the Dade County Medical Society. The fair, to be open through Sunday with more than eighty exhibits featured, marks the first time that such an event has been held in connection with the AMA Clinical Meeting.

J. Arnold Bargen, M.D., Rochester
O. J. Campbell, M.D., Minneapolis
George Earl, M.D., Saint Paul
F. J. Elias, M.D., Duluth
(Delegates to the American Medical Association)

Woman's Auxiliary

WORKING TOGETHER FOR HEALTH AND SAFETY

MRS. DAVID J. HALPERN
Brewster, Minnesota

First, I would like to congratulate Martin county for its splendid hospital auxiliary. You are the group who volunteer your services to the hospital. You are the ones on whom the administrator can call to raise funds for your hospital. You—who are well informed about the needs and facilities of your hospital—can go out among your friends and organizations and give them the correct information and thus create good will toward your hospital and an appreciation of the hospital as a community enterprise. Through education, information and service you are working for better hospital care for the people of your community. The greatest gift an individual can give to her community is a "little bit of herself."

The purpose of the medical auxiliary is health education and service. Hence, it seems very fitting that these two groups should have a joint meeting.

The United States has the world's highest standards of health and medical care. It is the world center of medical education and research. This has been accomplished by physicians with the co-operation and help of allied professions, branches of science, nurses, hospitals, etc. In fact, through the help of many branches of American society.

We know that many dread diseases that once were killers (typhoid, diphtheria, small pox, pneumonia and many others) have been brought under control. Yet, we do very little about the *unnecessary* deaths—those killed by accidents. Just this last weekend there were fourteen highway deaths in our own state of Minnesota. There were 38,000 highway deaths in our nation last year. This is more than the number killed in the Korean war. I haven't the figures, but if we were to add to those the number killed in farm, home and industry accidents, I am sure the number would be startling.

Think, too, of the millions who are injured—to say nothing of the economic loss. It makes us realize that safety, as a health measure, should be stressed. It is only a short step from carelessness to danger. The medical auxiliary is placing its accent on home and playground accidents. There is a definite place for each of you in this community action. Saving *healthy* lives is as important as saving *diseased* ones.

All of us are interested in well-trained personnel for our hospitals. You might interest the young people in your own family in the nursing profession. The shortage includes, besides registered nurses, practical nurses, medical record librarians, x-ray and laboratory technicians, dietitians, physical and occupational therapists, health educators, etc. Boys, as well as girls, should play an important part in filling these positions. You might interest high school students in these professions through teas, a panel discussion made up of nurses and those in the allied professions. You might tour your local hospital. You might see that nursing and allied professions are included if you have high school career days. You might sponsor essay or poster contests.

Another field in which we should all co-operate and work together on is civil defense. Our Auxiliary bulletin says: "Effective mass public education is the key to national survival—if war should come, women know that "a stitch in time saves nine." Are we, as women, delaying in taking the needed stitch? Many skilled and willing hands are needed. Points we are asked to stress are these:

"1. Home protection exercises. Every woman's first duty in the civil defense program is to educate her family in self protection against modern weapons and to make her home as safe as possible against both the dangers of enemy attack and disasters such as fire, flood, tornado.

"2. Home nursing classes.

"3. Education in the areas of sanitation, mass feeding, and temporary housing.

"4. Co-operation with the medical profession in preparedness."

We can each continue to render our valuable assistance to the Red Cross blood program. As you know, this program not only provides gamma globulin for polio, measles and hepatitis, but also furnishes free blood to our hospitals for community use.

The American Cancer Society, Heart association and other such agencies deserve the support of all Americans.

You can volunteer your services for the various types of testing in schools such as Mantoux test for immunization programs. You can help at the clinics for crippled children, at diabetic surveys.

These are a few of the fields in which you can help as volunteers. Someone said, "It is hard to imagine American life *minus its volunteers.* Let's hope they never go on 'strike'."

We, who are members of the Hospital Auxiliary and the Medical Auxiliary, should all *work* together for the health and safety of our community. I would like to close with an expression from India: "He who would leave footprints in the sands of time must wear *work* shoes."

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Presented at a combined meeting of the Martin County Hospital Auxiliary and the Auxiliary of the Minnesota State Medical Association, Fairmont, Minnesota, October 13, 1954.

Mrs. Halpern is recording secretary, Woman's Auxiliary to the Minnesota State Medical Association.

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

National

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American College of Surgeons, sectional meeting, The Fort Harry, Winnipeg, Manitoba, Canada, April 25-26, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

Conference on Reserpine in the treatment of neuropsychiatric, neurological and related clinical problems. Barbizon-Plaza Hotel, New York City, February 3-4, 1955.

International

International College of Surgeons, United States Section, Southwestern Division regional meeting, Shamrock Hotel, Houston, February 28-March 1, 1955.

Inter-American Congress of Radiology, Shoreham Hotel, Washington, D. C., April 24-29, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

International Symposium on Cardiovascular Surgery, Henry Ford Hospital, Detroit, March 17-19, 1955.

AMERICAN ACADEMY OF FORENSIC SCIENCES

The Seventh Annual Meeting of the American Academy of Forensic Sciences will be held in the Biltmore Hotel in Los Angeles on February 17-19, 1955. The president of the Academy this year is Dr. A. W. Freireich, Malverne, New York, and the chairman of the Program Committee is Dr. Milton Helpern, Chief Medical Examiner of New York City. The law department of the American Medical Association has long urged that the profession take an increasing interest in medicolegal problems and the programs of the Academy meetings are a definite step in that direction. Further information may be obtained by writing Dr. W. J. R. Camp, University of Illinois College of Medicine, 1853 W. Polk Street, Chicago, Illinois, secretary, or Dr. Frederick D. Newbarr, 109 Hall of Justice, Los Angeles 12, California, chairman of local committee on arrangements.

FAMILY DOCTORS' DAYS
at the
University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

January 19—Hospital Administration
February 16—Division of Anesthesiology
March 16—Division of Urology
April 20—Division of Neurosurgery

Activities begin with luncheon at 12:15 noon with the staff of the Department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, University of Minnesota Hospitals.

AMA COUNCIL ON EDUCATION AND HOSPITALS

The Council on Medical Education and Hospitals of the AMA is planning a program on the subject of "The Potential Use of Television in Postgraduate Medical Education" to be presented as a full-day working conference on February 5, 1955, in the ballroom of the Palmer House, Chicago. This is expected to be the first of a series of annual "workshop" type conferences on one particular aspect of postgraduate medical education. Television is the subject of the first meeting because of the extreme interest in this medium that has been shown recently, as well as its pertinence to the future of postgraduate education.

The program is planned in such a way as to present both the educational and technical aspects of the subject, so that medical educators and medical society, hospital and specialty society representatives at the meeting will be able to obtain a broad picture of the medium and help them to determine whether or not it is something they might use in their own programs, and if so the problems involved in its use.

Following a keynote address by Dr. John Cline, the morning session will be devoted to considerations of the purely educational aspects of the medium. The afternoon session will deal with technical considerations and financing. The participants will be drawn from the fields of general education, television, industry, medicine, medical education, pertinent government agencies and others. It is planned to have a number of demonstrations in the afternoon session using actual camera chains and receiving equipment. Following the session it will be possible for the audience to examine these and observe some of them in further action, and visit a local television station in action.

POSTGRADUATE COURSE ON DISEASES OF THE CHEST

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in co-operation with the respective state chapter of the College as well as the staffs and faculties of the local hospitals and medical schools of Philadelphia, will sponsor the Eighth Annual Postgraduate Course on Diseases of the Chest, to be held at the Bellevue-Stratford Hotel, Philadelphia, Pennsylvania, March 7-11, 1955.

Our postgraduate courses endeavor to bring physicians up to date on recent advancements in the diagnosis and treatment of heart and lung disease. Tuition is $75.

Further information may be secured by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

ESSAY AWARD CONTEST

The Council on Undergraduate Medical Education of the American College of Chest Physicians offers three cash awards to be given annually for the best contribution, prepared by any undergraduate medical student studying for a degree in medicine, on any phase of the diagnosis and treatment of chest diseases (heart and/or lungs).

The first prize will consist of a cash award of $250. Second prize will be $100 and third prize, $50. The three winners will also receive a certificate of merit.

The winning contributions will be selected by a committee of well-known chest specialists and will be announced at the 21st annual meeting of the American College of Chest Physicians, to be held in Atlantic City, New Jersey, June 2-5, 1955. All manuscripts become the property of the American College of Chest Physicians.

Applicants are requested to study the format of DISEASES OF THE CHEST as to length, form, and arrangement of illustrations to guide them in the preparation of the manuscript. The following conditions must be observed:

1. Five copies of the manuscript typewritten in English (double spaced) should be submitted to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, U.S.A., not later than April 10, 1955.

2. The only means of identification of the author shall be a motto or other device on the title page and a sealed envelope bearing the same motto on the outside enclosing the name and address of the author.

3. A letter from the Dean or Chairman of the Department of Medicine of the medical school certifying that the author is a medical student studying for his degree in medicine.

LECTURES AND DISCUSSIONS ON CURRENT MEDICAL PROBLEMS

The Mayo Clinic and Mayo Foundation announce a four-day program, April 19 to 22, inclusive, of lectures and discussions on problems of current interest in the general fields of medicine and surgery. The number of physicians and surgeons who can be accommodated is necessarily limited. Those wishing to attend should communicate with Dr. N. W. Barker, Mayo Clinic, Rochester, Minnesota, before March 1, 1955. Applications will be honored in the order in which they are received. There is no registration fee.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

A regular meeting of the Minnesota Society of Neurology and Psychiatry was held January 11 at the Town and Country Club. Dinner at 6:30 p.m. followed a social hour. During the evening, Dr. James H. Lyons and Dr. Ian A. Brown were voted into the Society as active members. The scientific program included two topics, "Modern Treatment of Brain Abscess" by Harold F. Buchstein, M.D., and "Children's Inpatient Psychiatry Service" by Reynold A. Jensen, M.D.

MINNESOTA TUBERCULOSIS AND HEALTH ASSOCIATION

The changing problem in tuberculosis and new measures of tuberculosis control were the topics discussed by James J. Waring, M.D., professor of medicine, emeritus, University of Colorado, Denver, Colorado, and Howard M. Payne, M.D., professor in medicine, Howard University College of Medicine, Washington, D. C., at the annual meeting of the Minnesota Tuberculosis and Health Association, October 11, 1954.

The meeting emphasized the importance of the Christmas Seal sale, which opened November 22, in supporting an expanded program in the fight against tuberculosis. The officers of the Minnesota Tuberculosis and Health Association who were re-elected are as follows:

President—Ray M. Amberg, Saint Paul
First Vice-President—Montreville J. Brown, Saint Paul
Second Vice-President—Mrs. H. E. Johnson, Stillwater
Secretary—W. H. Valentine, M.D., Tracy
Treasurer—T. C. Macoubrey, Saint Paul
Executive Secretary—E. A. Meyerding, M.D., Saint Paul

CONTINUATION COURSES

The University of Minnesota announces a continuation course in *Clinical Dietetics for Dietitians* which will be held at the Center for Continuation Study next March 3 to 5, 1955. Recent advances in medicine and their relationship to the field of dietetics will be taken up. The course will be presented under the direction of Gertrude Thomas, director of Nutrition and professor of Dietetics, University of Minnesota.

* * *

The University of Minnesota announces a continuation course in *recent advances in internal medicine for internists* which will be presented at the Center for Continuation Study from February 14-16, 1955. This year's course will deal with various aspects of hematology, cardiology, endocrinology, and respiratory physiology. The course will be presented under the direction of Dr. C. J. Watson, professor and director, Department of Medicine.

* * *

A continuation course in *clinical hematology for physicians* will be presented by the University of Minnesota at the Center for Continuation Study from February 28 to March 2, 1955. Registrants will be provided with slides for study during the course showing examples of various blood cell types and of the more frequent hematological diseases. Lectures will include discussions of the anemias, the leukemias, and coagulation defects. Registration will be limited.

RANGE MEDICAL SOCIETY ELECTS

Dr. T. R. Schweiger of Hibbing was elected president of the Range Medical Society when the group met last month in Hibbing. Dr. John S. Siegel of Virginia was named vice president and Dr. Kenneth Ahola of Hibbing was chosen secretary. Dr. M. J. McKenna of Grand Rapids is retiring president. Speaker for the scientific session was Dr. Erick Leppo of Duluth, who talked on "Toxoplasmosis."

SAFETY CLINIC HELD IN SAUK CENTRE

Farm and highway accidents, their cause and prevention, were studied at a six-county safety clinic held in December at Sauk Centre. Speaking at the clinic, the first such area-wide program, were Dr. H. H. Young of the Mayo Clinic, Rochester, who spoke on "The Seriousness of Farm Accidents"; Earl M. Larimer, director of highway safety with the State department of highways, who talked on the topic, "Your Responsibility in Traffic." The program was sponsored by the Sauk Centre Safety Council and the Citizens Advisory committee.

ST. LOUIS COUNTY SOCIETY MEETS

Dr. A. J. Bianco, Sr., of Duluth, was named president-elect of the St. Louis County Medical Society at the December meeting of the group. Dr. Bianco will serve in 1956. Installed as president for 1955 was Dr. K. R. Fawcett, Duluth, who succeeds Dr. Sam Boyer, Jr., Duluth. Elected vice president was Dr. Paul Reed of Virginia. Dr. J. E. Haavik, Duluth, was named secretary-treasurer. Dr. K. E. Johnson, Duluth, was elected to the society's Advisory Committee, where he will serve with Drs. M. J. Fellows and J. J. Coll, both of Duluth. On the State Advisory Committee will be Drs. Boyer, C. M. Bagley and P. F. Eckman, all of Duluth. New members of the Judiciary Committee are Drs. A. G. Athens, G. C. MacRae, and D. W. Wheeler, all of Duluth, who were elected for 1957. Serving in 1955 will be Drs. Richard Bardon, F. H. Magney and L. W. Morsman, Duluth, and serving in 1956 will be Drs. A. J. Bianco and F. J. Hirschboeck, of Duluth, and F. R. Kotchevar of Eveleth. On the Economics Committee new members are Drs. M. G. Fredricks, H. O. Hoff and O. E. Sarff, all of Duluth. The 1955 committee is made up of Drs. E. C. Bagley and J. J. Coll, Duluth, and L. W. Johnsrud, Hibbing, with Dr. C. M. Bagley and P. G. Boman, Duluth, and Paul Reed, Virginia, serving in 1956.

Grievance Committee members are: Drs. J. C. Feuling, Duluth, and M. J. McKenna, Grand Rapids, 1957; Drs. L. L. Merriman, Duluth, and R. H. Puumala, Cloquet, 1955; and Drs. M. G. Gillespie, Duluth, and P. S. Rudie, Duluth, 1956.

The society elected Dr. R. P. Buckley, Duluth, as 1957 delegate, with Dr. Josiah Fuller, Duluth, as alternate. Delegates in 1955 will be Drs. Gillespie and MacRae, with Drs. P. G. Boman and R. C. Pedersen, Duluth, as alternates. In 1956 Drs. Clarence Jacobson, Chisholm, and W. S. Neff, Virginia, will be delegates, with Drs. J. K. Butler, Cloquet, and G. M. Erskine, Grand Rapids, as alternates.

TRI-COUNTY MEDICAL SOCIETY ELECTION

Dr. Donald E. Dille of Litchfield was elected new president of the Kandiyohi-Swift-Meeker County Medical Society at a recent meeting in Willmar. He succeeds Dr. R. P. Griffin of Benson. Dr. Harold Wilmot and Dr. Karl Danielson, both of Litchfield, were named as delegate and alternate, respectively, to the Minnesota State Medical Association meeting.

WOMAN'S AUXILIARY

(Continued from Page 68)

RAMSEY COUNTY REPORTS ACTIVITIES

Chairman of Medical and Surgical Relief, Mrs. H. F. Schroeckenstein, reports the packing and sending of twenty-six cartons of medical supplies in November to the Central Conference of the United Lutheran church. In December the same committee sent 22 cartons to the Maryknoll Missions.

Mrs. Harold F. Flanagan represented the Auxiliary at a meeting in November of the Woman's Auxiliary to the Jewish Home for the Aged for the Northwest.

On December 7, Mrs. J. E. Teisberg and other auxiliary members staffed Christmas seal booths in the downtown stores and public buildings. Judging of Tuberculosis Essays for high school students took place on November 17, and Radio Project awards were presented to Judy Lewis, local junior winner, and to Joan Lachenmayer, local senior winner, both of St. Agnes High School. State winners were Judy Lewis, first place in the junior division, and Barbara Sitzman, first place in the senior division. Miss Sitzman is also from St. Agnes High School. The essays were read at the January meeting of the Auxiliary. A film on rehabilitation was shown. The auxiliary also elected a nominating committee at this meeting.

Mrs. L. T. Simons

Of General Interest

Dr. Elizabeth B. Jerome and **Dr. Henry P. Staub,** both of Minneapolis, have been elected to fellowships in the American Academy of Pediatrics.

* * *

A display of old medical instruments used by pioneer physicians of Itasca County has been prepared for a historical room in Grand Rapids by **Dr. M. J. Schirber**, Grand Rapids. The historical collection is sponsored by the Itasca County Historical Society and the Old Settlers organization. Medical instruments used by **Dr. Thomas Russell** and **Dr. H. E. Binet** are among those displayed.

* * *

A number of Minnesota physicians attended sessions of the 40th annual clinical congress of the American College of Surgeons in Atlantic City, New Jersey, in November. Rochester doctors who were on the program included **Drs. J. M. Waugh, O. S. Culp, H. K. Gray, J. W. Henderson, H. J. Moersch, J. L. Bollman, J. B. Erich, G. A. Hallenbeck, K. A. Lofgren, C. A. Owen, Jr., J. C. Cain, O. H. Beahrs, B. Marden Black, W. H. Hollinshead, R. K. Ghormley, J. C. Ivins, E. H. Soule** and **H. H. Young.** Also in attendance were **Drs. E. A. Banner, J. H. Grindlay** and **J. W. Kirklin**, all of Rochester, **Dr. David J. Sanderson** of Fergus Falls, **Dr. Robert R. Cooper** of Minneapolis, and **Dr. R. F. Hedin** of Red Wing.

* * *

Representing the Minnesota State Medical Council at sessions of the North Central Medical Conference at St. Paul in November was **Dr. C. L. Oppegaard** of Crookston. The conference is made up of membership from Iowa, Minnesota, Nebraska, North and South Dakota and Wisconsin. Dr. Oppegaard, with Mrs. Oppegaard, also attended the interim meeting of the American Medical Association in Miami, Florida.

* * *

Dr. Paul M. Brickley, formerly a consultant in the Department of Ophthalmology, Mayo Clinic, has joined the staff of the Sansum Clinic, Santa Barbara, California.

* * *

The American Academy of Pediatrics, Chicago, has announced the election to membership of **Dr. Robert O. Bergan** of the Duluth Clinic.

* * *

Capt. I. L. V. Norman, former Willmar resident, has been selected for promotion to rear admiral. Admiral Norman commands the United States naval hospital at Great Lakes, Ill. He is a graduate of the University of Minnesota, and has been on active duty since receiving his medical degree in 1927.

* * *

Dr. A. E. Ritt, has been elected president-elect for 1956 of the Ramsey County Medical Society. President for 1955 is **Dr. Harold F. Flanagan. Dr. E. C. Gibbs** has been named secretary-treasurer for 1955. Retiring president is **Dr. Wallace P. Ritchie.**

* * *

Three Northfield physicians have been reappointed to the Rice County nursing board by the county board of commissioners. Reappointed members include **Dr. Robert F. Mears, Dr. Donald H. Petersen** and **Dr. Leonard Aamodt.**

* * *

Speaker at the interim scientific session of the American Trudeau Society recently in New York City was **Dr. R. D. Miller** of Rochester.

* * *

Seven Minnesota medical men have been made Fellows of the American College of Surgeons. The induction ceremony took place at the group's five-day clinical congress in Atlantic City. Nearly 1,000 surgeons were made Fellows in the Academy, which is the world's largest organization of surgeons. Minnesotans inducted include **Dr. William C. Bernstein,** St. Paul; **Dr. Manuel R. Binder, Dr. Llewellyn E. Christensen, Dr. Robert R. Cooper** and **Dr. Owen F. Robbins,** all of Minneapolis; **Dr. Josiah Fuller** of Duluth, and **Dr. David J. Sanderson** of Fergus Falls.

* * *

Dr. C. S. Bossert, Mora physician, was honored at a dinner last month sponsored by the Mora Commercial and Civic clubs. Dr. Bossert has completed more than 40 years of service in Kanabec county and the surrounding area.

* * *

Dr. Daniel H. Bessesen, Buffalo Lake, formerly Minneapolis physician, has been elected chief of the medical staff of Renville County Hospital at Olivia.

* * *

A member of a panel discussing cardio-pulmonary laboratory work at the eighth Postgraduate Assembly in Anesthesiology at the Hotel New Yorker last month was **Dr. H. F. Helmholz, Jr.** of Rochester, associate professor of physiology in the Mayo Foundation.

* * *

At home on Crystal Bay, Lake Minnetonka, are **Dr. and Mrs. Winston Rudolph Lindberg.** Mrs. Lindberg is the former Larey Evans, daughter of **Mr. and Mrs. Walter Sale Evans** of Mendenhall, Mississippi. Dr. Lindberg's parents are **Dr. and Mrs. Arvid C. Lindberg** of Minneapolis. Dr. Lindberg has practiced in Minneapolis since his graduation from the University of Minnesota Medical School.

* * *

Dr. H. J. Moersch presented a paper at the interim session of the College of Chest Physicians. The meeting was held in conjunction with the semiannual meeting of the board of regents of the College in Miami Beach in December. Dr. Moersch also gave a paper before the Cuban chapter of the American College of Chest Physicians meeting in Havana.

* * *

Dr. Grant L. Garlock has announced the opening of offices for the general practice of medicine and surgery at 509½ Beltrami Avenue in Bemidji.

* * *

Dr. Willard White of Minneapolis addressed the Scott-Carver County Medical Society at its December meeting at Shakopee. The subject of Dr. White's talk was "Intramedullary Fixation of Fractures."

* * *

Dr. W. F. Maertz, formerly of St. Paul, has been a staff member of St. Peter State Hospital at St. Peter, Minnesota, since June, 1954.

* * *

Dr. C. H. Mead and **Dr. E. I. Parson,** who have been associated for a number of years in the practice of general surgery in Duluth, have announced the formation of a partnership for continuation of their practice.

* * *

At the annual meeting of the Park Region District Medical Society held at the River Inn, Fergus Falls, in December, 1954, the guest speaker was **Dr. Merrill D. Chesler** of Minneapolis. His topic was "Skin Grafts and Flaps in Reconstructive Surgery."

* * *

Recently certified as a specialist in orthopedic surgery by the Royal College of Surgeons of Canada is **Dr. A. Ross Lerner** of Minneapolis. Dr. Lerner recently passed the oral and written examinations held in Canada.

* * *

Among Minnesota physicians attending an interim session of the American Medical Association in Miami, Florida, recently were **Dr. Jack Delmore** of Roseau, **Dr. R. R. Wright** and **Dr. F. H. Rosenthal** of Austin and **Dr. E. R. Hudec** of Echo.

* * *

Dr. John A. Anderson, a University of Minnesota graduate and professor of pediatrics at Stanford University, has been named head of the University of Minnesota pediatrics department to succeed **Dr. Irvine McQuarrie.** Dr. McQuarrie, who taught the new department head his specialty, will devote his time to medical training and research. Dr. Anderson is a native of Sioux Falls, South Dakota, and is a member of nearly a dozen medical and professional organizations, a diplomate of the national medical board and of the American Board of Pediatrics and a writer and editor in the pediatric field.

* * *

Captain Raymond J. Lindeman, Air Force medical officer and commanding officer of the 615th Infirmary at Ent Air Force Base, has received his flight surgeon's wings. Captain Lindeman is a graduate of the University of Tennessee, and took specialized training at the School of Aviation Medicine at Randolph Field, Texas.

Dr. James Thomson of Austin recently attended a reunion at Cook County Hospital in Chicago. Dr. Thomson took four years of study and training in obstetrics and gynecology at the University of Chicago Lying-in-Hospital and Cook County Hospital.

* * *

Featured speaker at Minnesota Farm and Home Week activities this month at the University of Minnesota was **Dr. Walter C. Alvarez,** former Mayo Clinic staff member and medical writer and lecturer. Other prominent speakers included Governor-elect **Orville L. Freeman** and **Ezra Taft Benson,** secretary of agriculture.

* * *

Dr. R. B. Skogerboe of Karlstad has made plans to take a year of graduate work in surgery at Abbott Hospital, Minneapolis. Dr. Skogerboe will continue his work with **Dr. B. R. McHardy** at the Karlstad Clinic.

* * *

A number of Mayo Clinic staff members presented papers before various groups in December. **Dr. L. M. Eaton** participated in the National Conference of the Myasthenia Gravis Foundation at the University of Pennsylvania School of Medicine in Philadelphia and **Drs. R. G. Bickford** and **H. M. Keith** presented a combined paper at the meeting of the American League Against Epilepsy in New York City. **Dr. A. G. Karlson** gave a paper as part of the short course on diseases of laboratory animals, which was given by the Armed Forces Institute of Pathology in Washington, D. C.

* * *

A Minneapolis surgeon and former president of the Minnesota State Medical Association, **Dr. O. J. Campbell,** has been named to succeed **Dr. Arthur A. Zierold** as chief of the surgical staff at Minneapolis General Hospital. Dr. Zierold resigned in November.

* * *

Dr. C. A. McNeely, who recently opened private practice in Herman, has closed her offices. She has announced no future plans.

* * *

A Waseca physician since 1951, **Dr. S. T. Normann,** has purchased the medical offices of **Dr. H. M. McIntire** of Waseca, who retired last summer. Dr. Normann has had his offices with **Dr. B. J. Gallagher** and **Dr. R. D. Davis.**

* * *

Ten members of the Mayo Clinic staff were among those attending the joint meeting of the American Academy of Obstetrics and Gynecology and the Sixth Annual American Congress of Obstetrics and Gynecology in Chicago in December. **Dr. L. M. Randall,** program chairman, led a conference and **Drs. E. A. Banner, J. E. Faber, M. E. Giffin, J. S. Hunter, Jr.** and **J. H. Pratt** conducted conferences and led round-table discussions. Drs. Faber, Giffin and Hunter presented papers and **Dr. A. B. Hunt** attended a meeting of the section officers. Others attending were **Drs. C. E. Johnson, M. E. Mussey** and **R. A. Smith.**

Dr. T. S. McClanahan has been named chief of staff at St. John's Hospital for 1955. **Dr. R. W. Emmons** was elected first vice chief and **Dr. P. G. Polski**, second vice chief. **Dr. J. W. Strand** was named secretary-treasurer of the staff. Also named to the executive committee for a three-year term at the December staff meeting were Dr. Polski and **Dr. F. J. Plondke.**

* * *

Some 1,000 friends honored **Dr.** and **Mrs. M. A. Burns** of Milan on their fiftieth wedding anniversary December 21. The open house was held at the Kviteside Lutheran church, Milan. Dr. Burns, now over eighty years old, is still in active practice in Milan, where he and his wife have resided for over fifty years. For the past several years Dr. Burns has been associated with his son, **Dr. Floyd M. Burns.**

* * *

Five Mayo Clinic staff men attended the December session of the Radiological Society of North America in Los Angeles. Conducting courses were **Dr. D. S. Childs, Jr.** and **Dr. G. D. Davis. Dr. J. R. Hodgson** presented a paper and an exhibit. Also in attendance were **Dr. C. B. Holman** and **Dr. Martin VanHerik.**

* * *

Recently named to the board of governors of the Minnesota Society of Obstetrics and Gynecology was **Dr. Jack R. Pierce** of the Lenont-Peterson Clinic in Virginia. Dr. Pierce also presented a paper on infant mortality in Virginia at the December meeting of the Society.

* * *

Fourteen members of the Mayo Clinic staff attended the interim session of the American Medical Association in Miami last month. Among those attending meetings of the House of Delegates were **Drs. E. V. Allen, J. A. Bargen, F. H. Krusen, L. A. Buie** and **R. R. Kierland.** Dr. Buie is also a member of the Judicial Council and chairman of the Council on Constitution and By-laws. Presenting exhibits at the meeting were **Dr. T. T. Myers, R. L. Parker, H. F. Polley** and **E. W. Johnson Dr. J. L. Emmett** presented a paper. Also at the meeting were **Dr. Victor Johnson,** a member of the Council on Medical Education and Hospitals, and **Drs. W. McK. Craig, J. W. DuShane** and **N. O. Hanson.**

* * *

Dr. E. C. Sargent of the Austin Clinic attended a meeting of the American Congress of Maternal Welfare last month at the Palmer House in Chicago. Dr. Sargent was also formally inducted into the American Academy of Obstetrics and Gynecology.

* * *

Dr. Robert R. Kierland, dermatologic consultant at the Mayo Clinic, Rochester, has been elected assistant secretary-treasurer of the American Academy of Dermatology and Syphilology. The group met last month in Chicago. Dr. Kierland is also associate professor of dermatology in the Mayo Foundation at the University of Minnesota graduate school.

* * *

Convalescing at his home in Browerville is **Dr. A. J. Lenarz,** who was a patient in St. Paul since he had a heart attack several months ago. **Dr. Joseph Brennan,** resident at St. Luke's Hospital, St. Paul, has taken over Dr. Lenarz' practice temporarily.

* * *

At a meeting in December of the Hastings Memorial Hospital medical staff, **Dr. N. J. Kulzer** was named staff president and **Dr. H. T. Fasbender,** secretary.

* * *

Mayo Clinic staff physicians attending the meeting of the American Academy of Dermatology and Syphilology in Chicago last month were **Dr. L. A. Brunsting,** member of the Committee on Education, **Dr. H. Montgomery,** chairman of the Committee on Pathology, **Dr. B. L. Lewis, Dr. R. R. Kierland, Dr. W. F. Kvale, Dr. P. A. O'Leary** and **Dr. H. O. Perry.** Dr. Brunsting led an informal discussion group and conducted a symposium and Dr. Montgomery gave an instructional course and presented an exhibit prepared with Dr. Lewis. Others appearing on the program were Drs. Kierland, Kvale and O'Leary.

* * *

Dr. Adelaide M. Johnson was in New York last month to attend a meeting of the American Psychoanalytic Association. She served as chairman of a panel discussion on psychotherapy.

* * *

Dr. Leonard A. Lang of Minneapolis was elected president of the Minnesota Obstetrical Society at its meeting December 4. **Dr. Rodney F. Sturley** of Saint Paul was elected vice president, and **Dr. Edward A. Banner** of Rochester was named secretary-treasurer.

* * *

After a wedding trip to New Orleans, **Dr. and Mrs. Gordon J. Vosti** are at home in Saint Paul. The bride is the former Marcia Jean Williams of Saint Paul. Dr. Vosti, formerly of California, is on the staff of Minneapolis General Hospital.

* * *

Newly-elected chief of staff of Asbury Methodist Hospital is **Dr. Wayne S. Hagen.** Dr. Hagen succeeds **Dr. P. J. Preston. Dr. Einer Monson** was elected vice president, and **Dr. M. Elizabeth Craig** was chosen secretary-treasurer.

* * *

Dr. Robert Delmore left Roseau for Louisiana, December 27, to do postgraduate study in surgery in New Orleans.

* * *

Two Mayo Clinic staff members were recently named to posts on national medical boards. **Dr. D. A. Boyd** was re-elected secretary-treasurer of the American Board of Psychiatry and Neurology at its annual meeting in New York City. **Dr. F. P. Moersch** was appointed to represent the American Board of Psychiatry and Neurology on the advisory board for Medical Specialties.

* * *

Dr. Meyer Z. Goldner is the new president and chief of staff at Mount Sinai Hospital for 1955, succeeding **Dr. B. I. Saliterman.** Other new officers are: **Dr. Reuben Berman,** vice president, and **Dr. E. B. Cohen,** secretary-treasurer.

Dr. Earl Hill will be chief of the service for medicine; **Dr. Norman Bloom,** surgery; **Dr. Arnold Anderson,** pediatrics, and **Dr. Melvin Sinykin,** obstetrics and gynecology. **Dr. Manuel Binder** and **Dr. John LaBree** are newly-elected members of the executive committee.

* * *

Host to staff and board of directors of Union Hospital at New Ulm for their annual Christmas party was **Dr. F. H. Dubbe.**

* * *

Dr. Owen H. Wangensteen, chief of the Surgery Department and professor of surgery at the University of Minnesota, has been named one of ten recipients of the 1955 *Modern Medicine* Award for Distinguished Achievement. Announcement of his selection was made by Dr. Walter C. Alvarez, editor in chief of *Modern Medicine* magazine.

Dr. Wangensteen's award is the result of his research in technical surgery and his development of educational programs in the field of surgery. The cross-circulation operation, eliminating the need for heart-lung machines in heart surgery, was developed at the University of Minnesota under his direction.

The award is the only one that represents the opinions of members of the medical profession. The annual awards are made in recognition of significant contributions to medical science and the nation's health.

* * *

At the December meeting of the **Medical Staff of Glenwood Hills Hospital and Homewood Hospital,** the following officers were appointed for 1955: President and chief of staff, Irving C. Bernstein, M.D.; vice president, John Schumacher, M.D., and, secretary-treasurer, J. C. L. Peteler, M.D.

NEW LOCATIONS

Dr. J. A. Abbullarade, a former resident of San Salvador, has joined the staff of the Cokato hospital as an associate of **Dr. Theodore Greenfield.** Dr. Abbullarade previously was at Eitel, Fairview and Maternity hospitals in Minneapolis.

* * *

Dr. William D. Misbach of Fairmont has accepted a residency at the University of California at Los Angeles. He will train in pediatrics. Dr. Misbach was graduated from the University of Minnesota School of Medicine and interned at Wesley Memorial hospital in Chicago.

* * *

Dr. Allen W. Wittchow, who has practiced ophthalmology in St. Paul, has joined the staff of the Quain and Ramstad Clinic in Bismarck, North Dakota. A graduate of the University of Wisconsin Medical School, Dr. Wittchow interned at General hospital, Minneapolis, and completed a three-year fellowship at Mayo Clinic.

Dr. Thomas Kemp, a graduate of the University of Minnesota School of Medicine, has joined the medical offices of **Dr. G. A. Cooper** and **Dr. C. W. Stoops** in Madison, Wis. He will specialize in dermatology. Dr. Kemp interned at St. Luke's hospital, Duluth, and did postgraduate work at the University of Pennsylvania and at the Philadelphia skin and cancer hospital.

* * *

Dr. William J. Hultgen, who was located at 939 Lowry Medical Arts Building, St. Paul, is now engaged in the practice of ophthalmology at 7809 Herschel Avenue, La Jolla, California.

* * *

Dr. Warren H. Ruchie, a resident of Racine, Wisconsin, for the past two years, has become associated with **Dr. William Macklin, Jr.,** in the practice of radiology at Willmar. Dr. Ruchie, a graduate of the St. Louis University School of Medicine, interned at St. Mary's hospital, and is a Diplomate of the American Board of Radiology.

* * *

Dr. Kermit Stensgaard, who practiced medicine in Thief River Falls from 1946 to 1948, has returned to the city to become affiliated with the Falls Clinic. Dr. Stensgaard was a resident in surgery at Wayne University Hospital, Detroit, in 1948 and served in the Navy until recently.

MINNESOTA BLUE CROSS—BLUE SHIELD

The famous Blue Cross—symbol of the finest in hospital care protection to one out of every four Americans—has been assigned a new protective role. To protect you on the road, this emblem is covered in a highly-reflective material that can be seen for a distance of half a mile under head-light beams. Attached to the rear bumper of your car, it can mean life-saving protection if your tail light fails. When you are parked, the emblem's brilliant glow gives approaching motorists ample warning. Night or day, this Blue Cross emblem on your car gives you an added measure of safety and security.

Blue Cross will be happy to furnish you and your associates with emblems without cost or obligation. You can obtain an adequate number of free emblems by dropping a card or letter to the Blue Cross office.

Throughout the first month during which Blue Shield field representatives have been fully active in the Professional Relations programs, visits have been made to the offices of approximately 600 doctors in the Twin Cities and the surrounding area.

The principal purpose of this program is one of service to the doctors. Through the discussion of mutual problems which arise, more information about Blue Shield is furnished the doctors and their office personnel. In the reports submitted by the Blue Shield field representatives engaged in this work, questions have arisen about consultation services, diagnostic x-ray benefits when the x-rays are directly related to in-hospital medical care, surgery or obstetrical services, the meaning of the term "emergency medical care," which is defined in the contract as medical services required when the condition for which the patient is hospitalized places the patient's life in immediate and serious danger. Other inquiries have related to the information necessary in presenting a claim for payment by Blue Shield. In the latter, the result of this program of service may ultimately eliminate many problems which affect the prompt payment of claims.

Complete and detailed records are being maintained, including any recommendations made by doctors regarding the administration of Blue Shield and proper liaison between Blue Shield and the doctor or his staff. In view of the fact that the program has been so well received in the Twin Cities, it is being expanded to cover the doctors outside of the Twin City area. Having found the program to be beneficial both to the doctors and to Blue Shield, the field representatives are now going in the other areas of the state. With the co-operation of the members of the medical profession, a mutual understanding and resulting improved administration should result from this program.

DIAGNOSIS OF CORONARY HEART DISEASE

(Continued from Page 55)

put the greatest reliance. Extensive cardiovascular research is presently being done to ascertain the etiology of atherosclerosis. Ultimately the pieces of this puzzle will fall into place, and the completed picture will emerge. When the etiology becomes apparent, appropriate diagnostic as well as better prophylactic and therapeutic measures should also become available.

Milton M. Hurwitz, M.D.

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Original Contributions

Hypothermia in Cardiac and General Surgery

F. JOHN LEWIS, M.D.

Minneapolis, Minnesota

THE intricate control system which maintains body temperature within a narrow range is amazingly effective in a wide variety of environmental situations. Without this control, optimum function of the internal metabolism and long survival is impossible, yet for relatively short intervals, normal regulation may be purposely overcome and the body temperature may be significantly reduced without permanent harm. There is during this period of lowered temperature a striking reduction of the general metabolic rate which makes it possible to occlude the blood flow to all or any part of the body for a much longer period of time than is possible at normal temperatures. Thus, time is provided to do some types of surgery in an open, bloodless field—an advantage which has proven to be useful in intracardiac surgery and in surgery of the great vessels. The bloodless field made possible by hypothermia may also prove to be a worthwhile advantage in doing extensive operations for cancer. Furthermore, a moderate degree of hypothermia has been advocated for poor risk patients who must undergo surgery and it is possible that critically ill, hyperthermic patients may be benefited by a drastic reduction in their body temperature.

In this article the author will attempt to summarize, briefly, some of the provocative new uses of hypothermia, which are still mostly experimental, largely in the light of his own interest and experience.

Open Intracardiac Surgery

Bigelow[2] and independently, Boerema,[3] were the first to demonstrate experimentally that hypothermia makes it possible to operate within the open heart by means of a simple technique. With the body temperature reduced the total inflow to the heart may be occluded and then any chamber may be opened long enough to perform deliberate operations under direct vision. If this is attempted without lowering the temperature, the period of occlusion must be too short for practical use or else the central nervous system, and possibly the heart will be damaged. Hypothermia, by providing sufficient time, has made this uncomplicated way of working within the heart practical for patients as well as for the experimental laboratory.

Technique.—In patients who are to undergo intracardiac surgery for the repair of atrial septal defects[7], anesthetic induction with pentothal and curare precedes the actual cooling. The extremity leads of a cardioscope are fixed in position and a continuously recording rectal thermometer is adjusted. After this preparation, the cooling is started by wrapping the subject in a rubberized cover through which an alcohol solution kept at 25° F. is circulated. A period of one to two and a half hours is needed to lower the body temperature to 29.5 degrees C. (85 degrees F.). Then the temperature of the cooling blankets is raised, the chest is exposed, and the operation is begun, but the body temperature will continue to fall slowly for another hour reaching a low point of about 26 degrees C. (81 degrees F.).

The chest is opened, through an anterior incision with transverse division of the sternum and entry into both pleural cavities at the fourth interspaces. This provides excellent exposure for the exploration and repair. Exploration of the heart is carried out first before the open repair is done by inserting a finger through an opening in the right auricular appendage. By this means the surgeon obtains an understanding of the pathological anatomy which allows him to plan the precise type of repair that will be used.

From the Department of Surgery, University of Minnesota, Minneapolis.

This investigation was supported by Research Grant H-1374 of the National Heart Institute of the National Institutes of Health, Public Health Service, and by Grants from the University of Minnesota Graduate School, and the Minnesota Heart Association.

Just before the right atrium is opened, the channels to and from the heart are occluded. The superior and inferior vena cavae are closed first, then after a few beats, loops of tape are tightened around both pulmonary roots occluding the pulmonary veins, and finally a clamp through the transverse sinus of the heart is closed to shut off the aorta and pulmonary artery. The aorta is occluded to prevent air embolism while the heart is open.

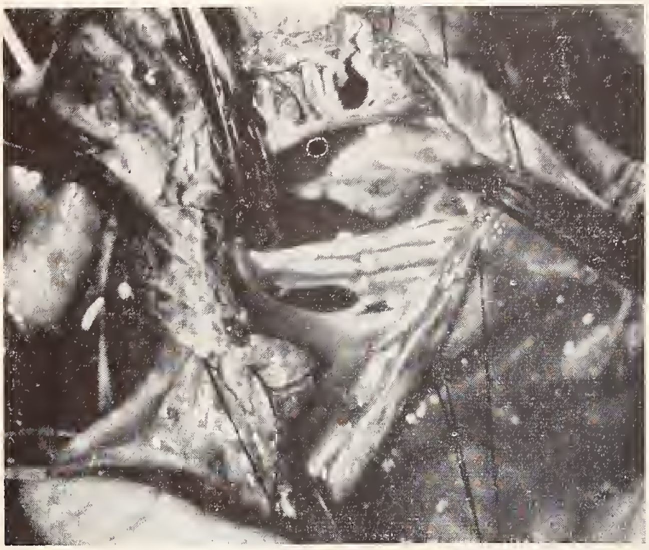

Fig. 1. Photograph of the open right atrium while the vascular inflow and outflow are occluded. The metal suction tip is placed within the atrial septal defect. Suture repair is about to begin. The head of the patient is to the left.

With the right atrium open, the septal defect is closed with continuous or interrupted silk stitches (Fig. 1). Just as the last stitch is tied the air is washed out of the left atrium by running saline into it through a small tube lying in the defect. This tube is removed as the repair is completed. Before closing the heart, air is evacuated from the right atrium by partially releasing the inferior vena cava thus allowing the atrium to fill with blood. The atrial wound is closed with a clamp and circulation is resumed. Finally, the atrial wound is sutured with a continuous silk stitch. In our human patients the circulation has been interrupted from four to eight minutes.

By the time the chest is being closed the rubberized blankets covering the patient have reached a temperature of 45 degrees C. and the body temperature may have started to rise. This warming-up process is accelerated, after the skin has been sutured, by immersing the patient in a bath of hot water at 45 degrees C. (114 degrees F.). In thirty minutes to one hour the body temperature will have reached 90 to 95 degrees F., and the patient is then removed from the water.

At the time we wrote about our early experience with this method,[7] eleven patients with atrial septal defects had been operated upon with two deaths. Now twenty-two patients with this type of defect have undergone surgery and there have been no additional deaths. The oldest, a survivor, was fifty-two.

This technique is a good one for repairing defects of the atrial septum, but it is not yet clear whether this or one of the other methods of closing these defects will prove to be the best. In any case, it is evident that this type of congenital heart disease is now curable.

Success in operating upon the atrial septum has been a stimulus to test the technique on other regions of the intracardiac anatomy. We have found that hypothermia can be used experimentally to make and repair ventricular septal defects, [11,12] and to operate upon the various cardiac valves under direct vision. It is likely that some of these experimental operations can be adapted to the treatment of human heart disease. Until much simpler and more efficient heart-lung machines are invented or until cross-circulation can be employed with no risk to the donor, hypothermia will play an important part in the new field of open, intracardiac surgery.

Cancer Surgery

More than a decade ago general body cooling was used in a memorable experiment to treat patients with advanced cancer.[5] Though most of the patients withstood the prolonged cooling well, there were, unfortunately, no cures, and there was probably no significant retardation in the onslaught of the disease. Hypothermia failed then as a treatment of cancer but it may now turn out to be an aid in doing some types of cancer surgery.

The usefulness of hypothermia for this type of surgery is due to the fact that it permits a relatively prolonged occlusion of the blood supply to any large segment or organ of the body. The bloodless operating field thus created makes resections, which would otherwise be bloody and difficult, quite dry and easy.

After preliminary experimental work,[10] we have used hypothermia this way to do total right hepatectomies for metastatic or primary liver cancers (Fig. 2). The patients have been cooled as the cardiac patients were and after cooling the liver was exposed through a right, combined, thoraco-abdominal incision. After a preliminary clearing of the porta hepatis the blood flow to and from the liver was interrupted, and we were then ready to resect the liver. Blood flow was occluded by clamping the thoracic aorta above the diaphragm, the portal vein and the hepatic artery below the liver and the inferior vena cava both above and below the liver. In this way the entire right lobe could be resected with minimal blood loss and adequate time was provided to repair holes in the vena cava and to suture the open vessels in the substance of the liver before the circulation was restarted. The blood flow has been occluded during these operations for forty-five minutes without producing anoxic injury.

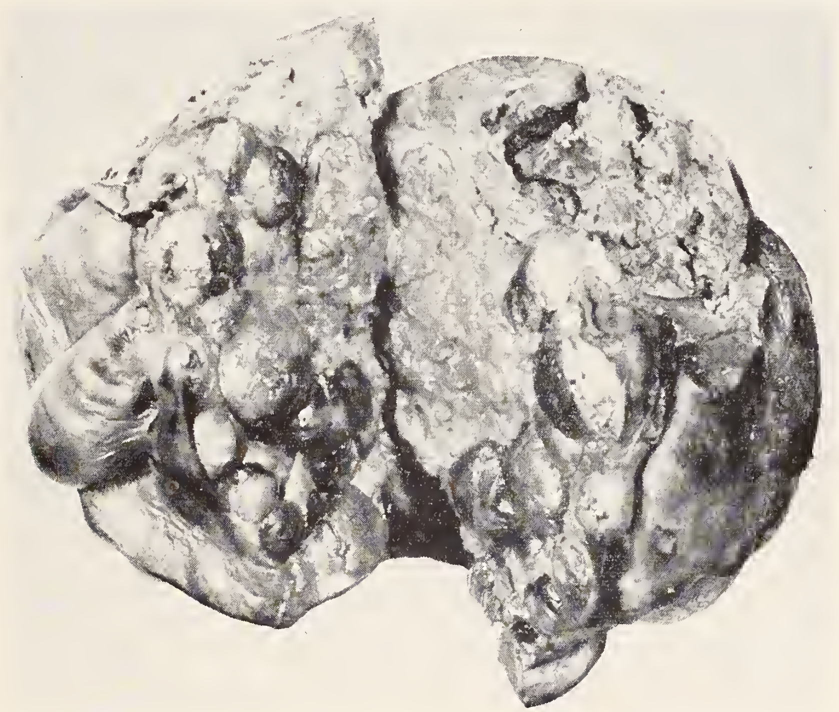

Fig. 2. Entire right lobe of the liver containing a large hepatoma which was removed during hypothermia. The patient was a nine-year-old boy. The gall bladder is seen attached to the surface at the left of the photograph.

Four right hepatectomies have been done this way. Two patients survived and there were two operative deaths, both of which were due to postoperative intra-abdominal hemorrhage which apparently started after the body temperature had returned to normal. One nine-month-old infant died a few hours after surgery of hemorrhage and a seventy-two-year-old man died three weeks postoperatively of an intra-abdominal abscess which appeared to be the late complication of hemorrhage.

The most recent patient who had a right hepatectomy during hypothermia was rewarmed before her abdomen was closed and post-operative hemorrhage did not occur. This seems to be the sensible way to avoid late hemorrhage. If it continues to be successful, hypothermia, plus high vascular occlusion, may prove to be a useful technique for doing extensive cancer operations, not only in the liver but in other areas of the body as well.

Surgery of the Aorta

General hypothermia has found a limited though distinct application for doing resections of the thoracic aorta.[4] Here again, as in intracardiac and cancer surgery, hypothermia allows a longer vascular interruption than is tolerated at normal body temperature. The abdominal aorta below the celiac and the renal arteries may be safely interrupted long enough at normal temperatures to remove an aneursym and stitch a graft in place, but in the thoracic aorta this may not be possible. A sufficient period of occlusion at the thoracic level to allow removal of a traumatic or syphilitic aneurysm and replace it with a graft, may lead to spinal cord damage. With the body temperature lowered, however, the period of occlusion that can be used safely is much longer. Cooling is not necessary when resecting a coarctation of the aorta and replacing it with a graft, for patients with this disease have an extensive collateral circulation which bypasses the obstruction.

Surgery in "Poor Risk" Patients

Bigler and McQuiston[1] in this country pioneered the use of hypothermia for "poor risk" children during heart surgery, but though the danger of an elevated temperature in these patients has been generally recognized, planned hypothermia as part of the anesthetic technique has not been widely adopted. Furthermore, at least in this country, moderate hypothermia has not often been tried for desperately ill patients who have to undergo other types of surgery. In Europe, on the other hand, Laborit[6] and other French surgeons, particularly, have advocated moderate degrees of hypothermia or "artificial hibernation" for weak, aged, or critically ill patients who must undergo major surgery. A combination of drugs called a "lytic cocktail" has been used with limited surface cooling to produce a slight reduction in body temperature prior to surgery.

Our own experience with this use of hypothermia has been limited but we have been impressed, nevertheless, with the ability of ill and aged patients to withstand surgery while their temperature was intentionally reduced. Frankly, however, it is not yet certain that hypothermia will help in bringing "poor risk" patients through major operations. To reach such a conclusion concerning this use of hypothermia or "artificial hibernation" a statistically controlled clinical study may be necessary and none, as yet, has been forthcoming.

Patients with Fever

Some effort to reduce the temperature of patients with high fever through the use of alcohol sponge baths, wet towels, and fans has long been a common practice. Such methods plus the use of antipyretics have been employed when the temperature, for any cause, has risen above 104 to 105 degrees F., and they have been abandoned again when the temperature has fallen to 102 degrees F. or lower. This treatment is often helpful but it may not have been carried far enough. There has not, for example, generally been a persistent systematic effort to reduce the temperature in these patients to normal or even subnormal levels and keep it there, yet such an effort may, we think, occasionally be strikingly beneficial. Certainly, the exhausting effect of a continuous fever with the racing metabolic rate that accompanies it may be crucially harmful and probably in no sense helpful.

This line of thinking has led us to use cooling in eleven seriously ill, feverish patients who have usually been suffering from postoperative complications. Chlorpromazine* plus sodium luminal was used initially for sedation and the patient was then placed on a large rubberized blanket containing a long rubber tube through which a cold alcohol solution was pumped (Fig. 3). In order to bring the temperature down it was usually necessary to cool the blankets to about 40 to 50 degrees F., but once the rectal temperature had reached a level of 95 to 97 degrees F. it could be kept there by maintaining the temperature of the blanket between 70 to 80 degrees F. Additional drugs were not always necessary after the temperature had fallen, and the patient could be kept at a normal or slightly subnormal temperature for days, if necessary, until, as cooling of the blanket was required no longer, it was evident that the tendency to fever had disappeared.

This is obviously merely palliative therapy and we have used it only for critically ill patients. It has, nevertheless, apparently been beneficial for several individuals. Of the eleven patients treated in this fashion five were apparently benefited and survived, three were helped temporarily but they finally died, and three succumbed without even temporary improvement.

Discussion

A few of the potential, clinical applications of hypothermia have been touched upon in this short report, but it would be highly presumptuous at this time, to conclude that hypothermia is going to be of lasting value in any of the ways described. Certainly, both regional and general hypothermia have turned out to be disappointing in the past after a calm evaluation stilled an early flurry of enthusiasm. It is possible that the uses of hypothermia listed here may also be superseded by other methods, or that they may for various reasons, never be accepted. It is true, even now, that many of the things that can be done with hypothermia can be done with other means, but possibly not so well nor so simply. Simplicity is clearly one of hypothermia's most attractive qualities.

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*"Thorazine" distributed by Smith, Kline and French.

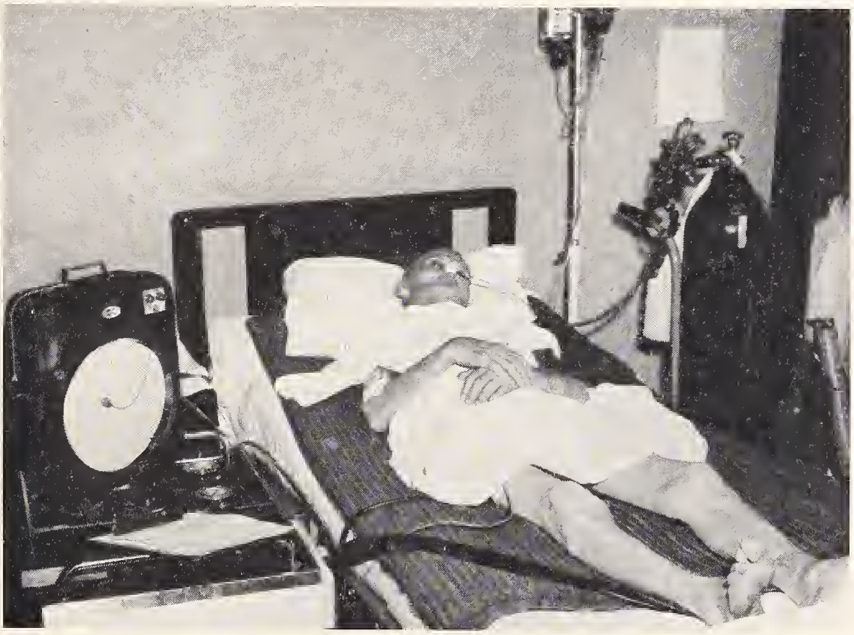

Fig. 3. This illustrates the technique used for cooling a hyperthermic patient. The rubberized blanket, cooled by the machine at the left, is seen beneath the patient. Usually this is covered with a single sheet. A rectal temperature recorder is placed on top of the refrigerating machine.

This quality of simplicity plus the other valuable characteristics of the technique have made it what is probably the best way at present to repair certain intracardiac defects under direct vision. For the repair of atrial septal defects and perhaps for other intracardiac anomalies the interval of blood flow occlusion allowed by moderate degrees of hypothermia is sufficient. Patients of a wide age range and various degrees of illness can successfully withstand this moderate degree of hypothermia.

For the future there is the challenge of trying to lower the level of safe, clinically practical, hypothermia. When temperatures significantly below those presently employed are possible, the method will doubtless have a wide new range of usefulness. This objective may soon be achieved for it is now possible to lower the temperature of rats and dogs down to a few degrees above freezing and revive at least some of them without sequelae.[8,9]

In this brief exposition there has been no space to try and describe the physiology of hypothermia, but this is just as well, for much that has been left unexplained is still unknown. A number of alluring pathways, each seemingly worthy of a long exploration, tempt the investigator of hypothermia; perhaps at least one of them will lead to a better understanding of this intriguing phenomenon.

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Heredity of Diseases of the Heart, Blood Vessels, and Blood

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DISEASES of the heart, blood vessels and blood constitute a large fraction of the organic ailments dealt with by the practicing physician. Usually of a serious nature, these diseases are frequently dependent in greater or lesser degree upon hereditary factors. Diseases having definite or possible genetic significance are shown in Tables I and II, with the more strongly hereditary conditions heading each list.

TABLE I. DISEASES OF THE HEART AND BLOOD VESSELS

Disease	Genetic Pattern
"Primary"	
Telangiectasia	Dominant
Congenital lymphedema	Usually dominant
Glycogen storage disease of heart	Probably recessive
Essential hypertension	Dominant factor (s)?
Rheumatic fever	Genetic susceptibility?
Congenital heart defects	Genetic factors?
Coronary heart disease	Genetic factors?
"Secondary"	
Arachnodactyly	Dominant
Friedreich's ataxia	Recessive
Myotonia dystrophica	Dominant
Muscular dystrophy	Several genetic types
Mongolism	Genetic factors?

"Primary" Types

Telangiectasia, an anomaly of the small blood vessels, is a well-known dominant trait whose presence is usually first heralded by nosebleeds. Not so widely known is the occasional occurrence of pulmonary arterio-venous fistulae as part of the clinical picture.

Congenital lymphedema, or Milroy's disease, is an uncommon anomaly of the lymph vessels which generally follows a dominant pattern of inheritance.

Glycogen storage disease of the heart is a rare and fatal abnormality of the heart in infants which is usually diagnosed only after death. Its occurrence in siblings has strongly suggested a recessive mode of inheritance. It is not to be confused with the hepatic type of glycogen storage disease, also probably a recessive genetic disease.

Essential or primary hypertension constitutes one of the most common chronic diseases in the adult population. Its familial occurrence has long been recognized, and a dominant mode of inheritance has been suggested by some workers in view of its frequent appearance in successive generations. However, its clinical features vary greatly, probably due to a variety of complicating environmental factors. Genetic analysis of this trait in populations is complicated by the variation in clinical picture, confusion with secondary types of hypertension, and the possibility of its not being a single entity.

Rheumatic fever, with its frequent heart involvement, is another of the common diseases with a well-recognized familial tendency. Innumerable genetic studies have been reported, with a variety of results and conclusions. Some of these appeared before, some after, the relatively recent finding that streptococcal infection is the immediate precipitating factor for an attack of rheumatic fever. Thus, the problem consists of determining whether the distribution of rheumatic fever cases is merely a random event (rheumatic fever is said to follow approximately 3 per cent of Group A

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beta hemolytic streptococcal infections), is determined by environmental factors, or whether it depends upon hereditary susceptibility of either a specific or general nature. The answer to this question awaits the development of biochemical studies, but present evidence suggests that susceptibility is at least partially dependent upon genetic factors.

TABLE II. DISEASES OF THE BLOOD

Disease	Genetic Pattern
Classical hemophilia	Sex-linked recessive
Afibrinogenemia	Recessive
Thalassemia	Recessive
Sickle-cell anemia	Recessive
Congenital hemolytic jaundice	Dominant
Hereditary nonspherocytic hemolytic anemia	Dominant
Ovalocytosis	Dominant
Pelger's nuclear anomaly	Dominant
Fanconi's anemia	Probably recessive
Pernicious anemia	Probable genetic factors
Polycythemia vera	Possible genetic factors
Familial neutropenia	Possible genetic factors
Familial eosinophilia	Possible genetic factors
Thrombocytopenia	Sometimes a genetic factor?
Leukemia	Sometimes a genetic factor?

Congenital heart malformations have recently been shown to have a fourfold increase in incidence in subsequent siblings of affected cases as compared to the general population. Other than this, and a known relationship to maternal Rubella in a small minority of cases, the etiology remains essentially unknown.

Coronary heart disease, like hypertension, is known to run in families, though to a less striking extent. Its occurrence is probably partially related to dietary factors, but the clarification of its exact etiology awaits further metabolic and chemical studies.

"Secondary" Types

Arachnodactyly, or Marfan's syndrome, is a dominant hereditary condition with a variety of striking clinical manifestations. The characteristic long slender limbs and digits have accounted for its name. Frequent eye involvement, with displacement of the lens, has made this condition well known to the ophthalmologist. Heart disease is also frequently associated. Formerly, the most common cardiac lesions were considered to be septal defects, but it is now agreed that medial degeneration of the aorta, often with aneurysm formation, is the typical cardiac manifestation of the disease. The syndrome illustrates beautifully the genetic phenomena of pleiotropy and variable penetrance.

Friedreich's ataxia has been viewed primarily as a disease of the central nervous system, even though some of the earliest case reports described myocardial changes. Cases simulating rheumatic and congenital heart disease are now being detected with increasing frequency by alert cardiologists familiar with its complete clinical picture. The disease generally shows a typical recessive type of inheritance.

Myotonia dystrophica, a dominant disease also familiar to the neurologist, sometimes shows myocardial changes. The same is true for the various genetic types of *muscular dystrophy* (dominant and sex-linked recessive forms).

Mongolism, one of the most common and best known types of mental deficiency (incidence of about one in 750 births), is considered by some to be partially dependent upon genetic factors, but its etiology remains essentially unknown. An association with increased maternal age has been reported by nearly all investigators. In 25 to 40 per cent of cases there are associated congenital heart defects, usually septal defects, but encompassing the whole spectrum of congenital heart anomalies.

Diseases of the Blood

Hemophilia still ranks as the best known of the hereditary diseases of the blood, having been the subject of countless articles in the lay press and continuing to be the classical example of sex-linked recessive inheritance in all textbooks of genetics. With the recent development of more precise tests for deficiencies in the clotting mechanism of blood there has been a critical review of cases which formerly would have been labeled merely as hemophilia or hemophilia-like disease. These studies have revealed a whole "family" of diseases. Classical hemophilia remains the most common type, yet clinical evidence suggests that this disease may represent a series of alleles, each producing disease of different severity. One of the proposed classifications, that by the group at the University of North Carolina,[1] is as shown in Table III. All of these diseases are bleeding disorders involving clotting abnormalities. Incidentally, the frequently asked question of whether hemophilia can occur in the human female can now be answered in the affirmative. A rather recent and remarkable finding has been the recognition of a disease in dogs simulating human hemophilia both in laboratory features and in genetic pattern.

TABLE III. HEMOPHILIA AND RELATED DISEASES

Disease	Genetic Pattern
Classical hemophilia (deficiency of antihemophilic globulin or AHG)	Sex-linked recessive allelic series
Hemophilioid state A (Parahemophilia or Owren's disease; deficiency of accelerator globulin or AcG	Probably dominant
Hemophilioid state B (deficiency of serum accelerator Factor or SAF)	Dominant
Hemophilioid state C (Christmas disease; deficiency of plasma thromboplastin component or PTC)	Sex-linked recessive
Hemophilioid state D (deficiency of plasma thromboplastin antecedent or PTA)	Probable genetic factor

Afibrinogenemia, another bleeding disorder which could well have been included in Table III, has proven to be as interesting as it is rare. Just as genetic theory requires of a very rare recessive trait, this condition involves cousin marriage in a high percentage of reported cases. Heterozygotes, or carriers of a single abnormal gene, show a reduced amount of fibrinogen, and can therefore be identified despite the absence of clinical symptoms.

Thalassemia, previously better known as Mediterranean anemia because of its higher incidence among Mediterranean peoples, is another recessive trait in which the heterozygote shows a lesser but definite effect. In fact, the homozygous condition has been termed thalassemia major, and the heterozygous condition has been called thalassemia minor. This situation raises the purely academic question of nomenclature, whether to designate the thalassemia gene as a recessive or a dominant.

Sickle-cell anemia is somewhat similar to the foregoing in its genetic features. The homozygote shows the full-blown disease, and the heterozygote ordinarily shows merely the sickling trait. Biochemical studies have shown the former to possess 100 per cent abnormal "s" hemoglobin usually, and the latter approximately 40 per cent. Sickle-cell anemia shows a marked racial predilection, being a disease almost exclusively of individuals with negro ancestry. Interaction between the sickle-cell gene and the thalassemia gene has been identified, resulting in a clinical condition resembling sickle-cell anemia. In addition, another abnormal hemoglobin, hemoglobin "c," has been identified, and has been found to have a similar genetic pattern. It appears that the current combined clinical-genetic-biochemical studies of the hemoglobins will reveal a whole series of genetically distinct but clinically related diseases.

Congenital (hereditary) hemolytic jaundice has been a favorite textbook example of dominant inheritance for many years. In addition, it has been used to illustrate the application of genetics to clinical medicine. Some bearers of the gene will suffer severe hemolytic crises, while others will remain asymptomatic. All cases can be identified by the presence of abnormal erythrocytes, spherocytes, with increased osmotic fragility as compared to normal erythrocytes. Since surgical removal of the spleen affects a clinical cure in this condition, splenectomy has been suggested as a prophylactic measure to be used in asymptomatic relatives who have been identified by family studies as carriers of the gene. In the past several years a new disease has been described, *hereditary nonspherocytic hemolytic anemia.* It resembles the previous disease in its genetic pattern, but it does not respond to splenectomy.

Ovalocytosis, or elliptocytosis, dominantly inherited, is a relatively uncommon anomaly of the red blood cells as implied by the name. The trait is of rather minimal clinical importance.

Pelger's nuclear anomaly, again a dominant trait, involves decreased segmentation of the nuclear material in the polymorphonuclear leukocytes.

Fanconi's anemia was first described several decades ago, but in recent years there has been a real influx of cases into the medical literature. The disease presents as a hypoplastic anemia together with a number of other varied signs, skeletal anomalies being among the more conspicuous. Its frequent occurrence in siblings suggests a recessive inheritance.

Pernicious anemia rates as one of the most celebrated of human ailments, having been the subject of some of the most brilliant of medical discoveries. Though its familial tendencies have long been recognized, its exact etiology and its basic or primary nature remain largely unknown.

Polycythemia vera, familial neutropenia, and familial eosinophilia have all been described as showing familial tendencies in some cases, just as the names suggest. However, the etiology of these conditions continues to be mainly a matter of speculation, and they may each be loose groupings of several entities.

Primary thrombocytopenic purpura has also been described as showing familial tendencies in some cases, but it is more interesting in its "pseudogenetic" features. Thus, women with this condition, whether splenectomized or not, can have infants who show a transient thrombocytopenia (low platelet count) during the first few weeks following birth.

Leukemia has been the subject of intensive investigation. As with the majority of malignancies, the etiology remains obscure. A few pedigrees suggest that hereditary factors may play a role in an exceptional case.

Many other disorders of the blood and vascular system have also been described as familial or hereditary, but usually the evidence rests on only a scattering of pedigrees. Moreover, the disorders themselves have often been rather poorly defined clinically. There is little doubt that new hereditary diseases of the blood will be identified, especially as progress is made in the field of blood proteins. Thus, pediatric cases of a recently recognized abnormality, *agammaglobulinemia,* may represent a sex-linked recessive gene. Even as it is, the currently known hereditary diseases will progressively attain greater clinical significance as those of environmental origin gradually come under medical control.

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MONIES FOR MEDICAL SCHOOLS

The first 1954 distribution of unrestricted funds to the nation's eighty medical schools was made in July by the National Fund for Medical Education. These 1954 grants totalled $2,176,904.71, including $1,101,000 from the medical profession through the American Medical Education Foundation.

Each of the seventy-four four-year medical schools received $15,000 plus $25 per undergraduate medical student enrolled in the school. Each of the six two-year schools received $7,500 plus $25 per student. Added to these grants were gifts of individual doctors to designated schools.

Since 1951, nearly seven million dollars has been awarded to the country's medical schools—half of this total contributed by the medical profession. Fund grants, are used by the schools primarily to fill teaching vacancies, to create new faculty posts and to initiate courses in areas of recent scientific advances.

Psychiatric Aspects of Ulcerative Colitis

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IT IS NOW generally accepted that emotional disturbances may produce functional and organic changes in various organs of the body. Repressed emotional conflicts, unfulfilled desires, long-standing deep frustrations, feelings of guilt and fears of one type or another are factors that frequently manifest themselves in the development of physiological and pathological alterations in the human body.

Emotionally conditioned illnesses are prevalent and widespread. Emotional factors frequently block the recovery of an illness that otherwise would respond to medical care more rapidly and more completely. The resolution of these emotional factors is necessary for the patient's recovery. Unless this is accomplished many patients may develop a chronicity of disabling, discomforting symptoms that produce a state of invalidism.

Psychiatric aspects implies behavioral aspects. Behavioral aspects includes all that has gone before in the way of life's experiences and the development and organization of man's defenses, with which to meet new situations, especially those that threaten his well-being. Behavior then, is a process of adaptation of the organism between physiological needs and external circumstances.

The dynamic approach to the understanding of human behavior and its relation to organic lesions, still meets with some resistance from physicians and students of medicine. This is somewhat understandable when one considers that psychiatry must, in a sense, compete with a more easily understood and more objective and tangible approach to disease, as demonstrated in those illnesses where known etiologic factors are present, laboratory data confirm a dysfunction and very real pathological tissue changes can be visualized. It is important, however, that the physician recognize that not only does the patient have a physical disease but that the patient is already possessed of behavior patterns to which the passing disease process has merely been added.

Physicians must not lose themselves in their consideration of the pathologic and bacteriological aspects of a disease, to the point where they lose sight of the meaningful data in the patient's life history. They must recognize that the patient's past experiences, his capacities and his motivation toward the future, all may serve as determining factors in the way in which he will react to any given organic process and also may be the deciding factor in the precipitation and continuation of a patho-physiological disturbance.

The importance of emotional factors in recovery from many physical diseases is now stressed by medical schools throughout the student's entire training. Most physicians are now thoroughly conditioned to the recognition of the inseparability of mind and body. The application of this knowledge has aided greatly in the treatment of psychosomatic disorders. To lose sight of the emotional factors means losing an important contribution to the patient's welfare.

Ulcerative colitis is one of many illnesses having emotional disturbances as a factor in their etiology. It is a disease that is notoriously resistant to treatment and is severely disabling. Unresolved emotional problems delay and may prevent recovery in this condition. There are three phases of treatment: medical, surgical and psychiatric. All three are important. All three must be given careful consideration in the treatment program.

The emotional factors may vary in degree from case to case and play a more important part in one patient than in another. It can be said, however, that there is an emotional disturbance in every case of "non-specific" colitis. Unless the emotional factor is solved, medical and surgical

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care will be greatly retarded and frequently complete cure will not be possible.

Careful investigation of the life experiences and emotional responses of patients suffering from ulcerative colitis, reveals a close relationship between the appearance of a painful psychological situation and the appearance of symptoms of the disease. The histories of these patients show that they fall into very similar categories. They show many infantile traits, among them an inability to control the bowel—a common infantile response to fear.

The ulcerative colitis personality according to Daniels,[3] is apt to be self-centered and dependent. The dependency is shown particularly toward the mother or a mother substitute, frequently an older sister. This dependency is often maintained by the involved parent or substitute, at the expense of the patient, especially through fostering illness under the guise of care. As would be expected in dependent persons, many ulcerative colitis patients are emotionally and sexually immature, and do not carry responsibility well. The Yale group called them the "giver-uppers." In this they are the opposite of the peptic ulcer patients, who are aggressive as a rule and seek responsibility. The males are inclined to be of the passive type, pathologically attached to the mothers and frequently unmarried. Of Murray's[8] original series of fifteen cases, seven were men and unmarried. The women, who are more frequently married, are apt to be of the fussy type of housewife, with a marked sense of neatness.

Wittkower[9] found that psychiatric disorders were far more frequent in the ulcerative colitis patients than in the average population. These manifestations antedated the colitis and were so gross that a special controlled group was not deemed necessary. He found no uniform personality make-up, but his cases fell into three groups, with common characteristics within the group; a fourth group consisted of the miscellaneous personality types. The first group was characterized by obsessional traits; the second, which was made up entirely of women, by hysterical traits; and the third by depressive or schizoid trends. The personalities in childhood coincided with those in adult life, except that there was often an exaggeration of characteristics after maturity.

Comparative studies by Grace, Pinsky and Wolff[6] of carefully matched groups of patients suffering from ulcerative colitis have shown that the length of illness, rate of serious complications and mortality rate were all considerably less in a group that was managed by the psychiatric approach, than in a controlled group in which the importance of the emotional factors was either not recognized or was neglected.

To theorize about the dynamics whereby psycho-biological forces result in a demonstrable organic pathology and physiological malfunction is indeed intriguing. Many hypotheses have been postulated but all leave much unanswered. Perhaps it will never fall within the scope of man to understand all about himself, either as a physical being or as a thinking and feeling organism; nevertheless, we must continue to strive to solve these problems in order to further man's welfare.

In discussing the role of the hypothalamus in relation to psychosomatic disorders, Grinker[7] states:

> "The hypothalamus developed from the basal portion of the diencephalon under the influence of the medial forebrain bundle is in a position favorable for its role as a central integrator of several important functions It receives afferent fibers from the ascending tracts by way of the thalamus and subthalamus, and sends efferent fibers which directly or indirectly affect the activity of the entire autonomic nervous system. The hypothalamus integrates individual activities of viscera, endocrine glands, and smooth muscles with reference to the economical needs of the entire organism. Such regulation and integration of the body's internal milieu serves to maintain what Cannon termed 'homeostasis.'
>
> "If the hypothalamus is an autonomic center concerned with maintaining a constant internal environment, it probably has two general systems, each with its own afferent and efferent connections and acting reciprocally to each other. There is considerable evidence that these dual centers exist. The posterior hypothalamus is an orthosympathetic and the anterior a parasympathetic center. The hypothalamus represents visceral activity in more special highly organized combinations, a regrouping from the lower levels of the nervous system from such centers as the spinal, bulbar, pontine, et cetera. Its orthosympathetic areas control and integrate all visceral activity, its parasympathetic areas are more highly specialized and discreet."

According to Fulton[5]:

> "The autonomic division of the nervous system can no longer be regarded as a purely peripheral system, but rather as an elaborately organized division of the

> central nervous system with representation at all levels. At each level, moreover, the somatic and autonomic systems dovetail with one another, and while the somatic may predominate in a given region—and thus obscure the autonomic component—it is generally impossible to evoke a somatic reflex that does not have an autonomic concomitant of central origin."

Alkan[2] first clearly stated that organic disease may be profitably studied by psychological methods. He indicated that psychogenic disturbances within the field of the autonomic nervous system may result finally in organic changes, the morphological mechanisms of which form only the last link of an intricate causal chain. He stated that spasms of smooth muscle, which secondarily lead to anemia of an organ, stasis, muscular atrophy or infection may be the expression of emotional conflicts. As a result of these secondary factors, organic changes result in the visceral tissues or somatic structures, which of themselves, as terminal events, are irreversible and constitute the so-called organic disease.

Alexander,[1] through his psychoanalytic researches, called attention to the role of psychic conflicts in the etiology of gastrointestinal disorders. He stated, however, that no disturbance of innervation of an organ could symbolically express a specific phantasy or a repressed idea. On the other hand, an organ's specific function could be used to express a tendency or psychic direction which by a process of conflict would not normally be expressed through the voluntary nervous system and its organs of expression.

Fulton[4] has stated:

> "Newer disclosures concerning the relation of the cerebral cortex to the autonomic nervous system give an adequate physiological basis for the relationship long recognized between mental states and visceral processes. To be a little more specific, it is clear that one now has adequate reason for believing that the load which is placed upon the heart and circulation may be determined in large measure by the mental status of the individual, and be thus quite independent of the more obvious factors of over-exercise and exertion. The heart and circulation may be worked just as hard, to just as much of a detriment to the body as a whole, from the swivel chair as from the rower's seat. Many disturbances of gastro-intestinal functions undoubtedly have a similar basis and one might add that instead of referring to them as "psychogenic disturbances" one may more appropriately designate them as purely physiological aberration and explicable in terms of recognized physiological mechanisms. The more precise analyses of these psychosomatic relationships will come from the study of man, and hence can come only through active collaboration between clinician and physiologist."

In anxiety, danger is not real; it is unknown, internal, and results from an increase in instinctual tension demanding expression. Freud called it a condition signal within the ego, conscious within the personality as a warning of impending danger. Anxiety produces specific unpleasurable feelings, efferent discharges and their perception. Anxiety often results in a paralysis of certain higher ego functions. It is, then, the first sign of an autonomic influence on the cortex which has been learned by previous testing to indicate danger for the organism. It is a feeling which accompanies autonomic forces sufficiently strong to overcome cortical inhibition and force the cortex into activity, movement resulting, in an attempt at solving the situation which has evoked an internal tension. The organism attempts to solve the conflict by psychological measures equivalent to fright or flight. Compromises at times are attempted. Successful solutions are frequently not possible and the cortex will allow regression and infantile modes of emotional expression for the sake of avoiding prolonged or greater emotional feeling. The result is visceral expression or organ dysfunction, that constitutes the first step toward organic disease.

Psychiatric consultation is not always necessary in the treatment of ulcerative colitis. Psychotherapy can, in many instances, be successfully carried out by the attending physician, provided he is conditioned to the patient's needs, shows a real interest in the emotional aspects of the illness and has adequate understanding of what psychotherapy entails. When one considers the personality make-up of most patients with ulcerative colitis, it becomes evident that a supportive type of treatment will usually be the most successful. A supportive approach is a necessity during the acute phases of this disease. Some therapists might argue this point and claim that supportive type of treatment weakens the patients by making them too dependent upon the physician, thus making it difficult for them to build up their own resources to a point where, upon recovery, they can again take their place in society. There is a degree of danger in supportive treatment unless it is done adeptly. There is a definite place for more direct and intensive psychiatric therapy when the patient has improved to a point where the uncovering of painful emotional experiences will not endanger a relapse of the disease.

Careful attention to the physical treatment of the patient, applied daily, with a goodly mixture of support and protection will be found to be the best treatment. During the course of therapy, the patient must be given ample opportunity to express fully his life's experiences in a longitudinal fashion, starting with his childhood experiences and on up through his school years, adolescence and college, his work adjustments, social adjustments, including marriage; in fact, an opportunity to express everything that has happened to him up to the time of the illness. The attitude of the therapist must be that of a sympathetic listener. There is no room for admonishment, lecturing or preaching to a patient of this kind. From time to time during the interview, acknowledgment of the nature and meaning of the life's experiences of the individual should be made in a fashion that shows the patient a sympathetic understanding and carefully avoids any manifestation of unfavorable or critical attitude toward the patient and the way in which he has lived.

It is well for the patient from time to time in the course of the interviews, to be reassured of his physician's attitude toward him. It is necessary to be quite emphatic at times in showing the patient that the physician desires to understand him, recognizes him as a worthy fellow and shows by word and manner, that in addition to being a medicine man, the doctor is a counselor and friend.

Since no patient lives in an isolated emotional environment, it is necessary for the physician to be thoroughly acquainted with all the contributing factors in the immediate life situation of the patient. This entails interviewing many other members of the family, especially the husband or wife. Oftentimes unpleasant relationships between immediate members of the patient's family or in-laws, have helped to create an unbearable situation for the patient, and thus contribute to his illness. In these instances every effort should be made to remove these disturbing factors. It is often necessary to exclude certain visitors, who the physician feels would be unwelcome to the patient. Isolation may be necessary and advantageous in some instances in order to avoid unfavorable reactions on the patient from incidents occurring in the immediate environment.

The nursing staff in charge of the patient should be adequately briefed relative to the particular significance of their part in the treatment program. In these days of nursing shortage and understaffing of competent, well-trained nurses, those in immediate charge of the nursing duties on a given section of the hospital are usually over-worked and since they are human, their dispositions do not always make for the best relationship with the patient. This is particularly true in a case requiring very close attention to the patient's physical needs, such as feeding, cleanliness and attention to exactness in regard to medication. Since the ulcerative colitis patient is usually emotionally ill-at-ease, he is quick to sense omissions in his care and interpret neglect as being willful, because of the particular nature of his illness. Every effort should be made to create, as far as possible, an element of smooth teamwork on the part of the attending physician and the nursing staff. Unless this is done, repeated incidents will happen during the course of the day that will largely undo what the physician is trying to accomplish.

Summary

1. The need for the consideration of emotional disturbances in the treatment of ulcerative colitis is pointed out.

2. The hypothalamus is discussed in the light of its being an integrating center for autonomic nervous function and its relation to emotional stresses is reviewed.

3. The nature of anxiety and its effect upon physiological states is described.

4. Team work in the treatment of ulcerative colitis is stressed. This team work calls for recognition and proper timing on the part of the surgeon, internist and psychiatrist in relation to their contribution to the treatment program.

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(Continued on Page 95)

Meconium Peritonitis

Report of a Case and Review of the Literature

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Report of Case

The patient was a newborn infant, whose birth weight was 6 pounds 14½ ounces, and length 20 inches. The child was born spontaneously by vertex presentation after normal labor. The mother's antenatal course was uneventful.

During the following two days, temperatures were taken on the infant four times a day; the readings being as follows:

Minimum reading 97.6 at birth.

Maximum reading 97.8 at 10:00 A.M., the second day of life.

The color at all times was good, and the baby was put to breast after the usual twenty-four-hour waiting period. The infant was alert, the chest was clear, and no evidence of abnormality was noted until the morning of the second day of life, at which time the mother called the physician to her bedside and said that she thought the abdomen of the child was somewhat distended. There was no evidence at this time of any fetal distress, and the doctor who saw the baby was unable to find any physical abnormality other than a mild distention of the abdomen. The liver and spleen were both palpable and the abdomen was soft. The latter was tympanitic over much of its surface.

Approximately seven hours after the abdominal distention was initially observed, the baby was again examined, the distention previously noted had somewhat increased. The physician suspected meconium ileus had occurred and gave a 2-ounce normal saline enema, which was returned along with a small amount of inspissated meconium. The baby at that time did not seem to have respiratory distress. Because of the result of the enema, in returning inspissated meconium, another 2-ounce saline enema was given. Within a few minutes respiratory distress was evident, the respirations becoming shallow and gasping. The enema was not returned, and the infant was placed in an incubator under oxygen. It was thought, at the time, some perforation of the rectum or sigmoid colon by the enema tip might have occurred.

An x-ray was taken, and evidence of some free air was seen under the left dome of the diaphragm. The consulting surgeon was of the opinion that immediate surgery should be undertaken to close the perforation of the bowel. Further x-rays were then taken in lateral and upright postures, and more free air was evidenced in the peritoneal cavity, confirming the diagnosis of a perforated viscus. While preparing the infant for surgery she suddenly expired approximately eight and a half hours after the abdominal distention was originally observed.

Autopsy Findings

The embalmed body was that of a two-day-old infant measuring 49.5 centimeters long and weighing 3,125 grams. The abdomen was distended. There were no other external abnormalities.

The skull and central nervous system were not examined.

Heart.—The heart was normally placed, but was enlarged on the right, primarily by an enlarged right auricle. The pericardium was smooth. The sac contained no fluid. The heart chambers were opened and no abnormality other than right auricular dilatation was noted. The valves appeared competent. The foramen ovale was patent and the ductus arteriosus was open. The great vessels of the heart were normally placed and showed no abnormality.

Lungs.—Pleural spaces were smooth and free of fluid. Both lungs floated when immersed in water. Cut surfaces showed no evidence of inflammation or infection. The trachea and bronchi were unobstructed. Hilar lymph nodes appeared normal.

Abdominal Cavity.—All peritoneal surfaces were inflamed and covered by a thick, yellow-green, purulent material, and a moderate amount of foul-smelling air was present in the peritoneal cavity (Figs. 1 and 2).

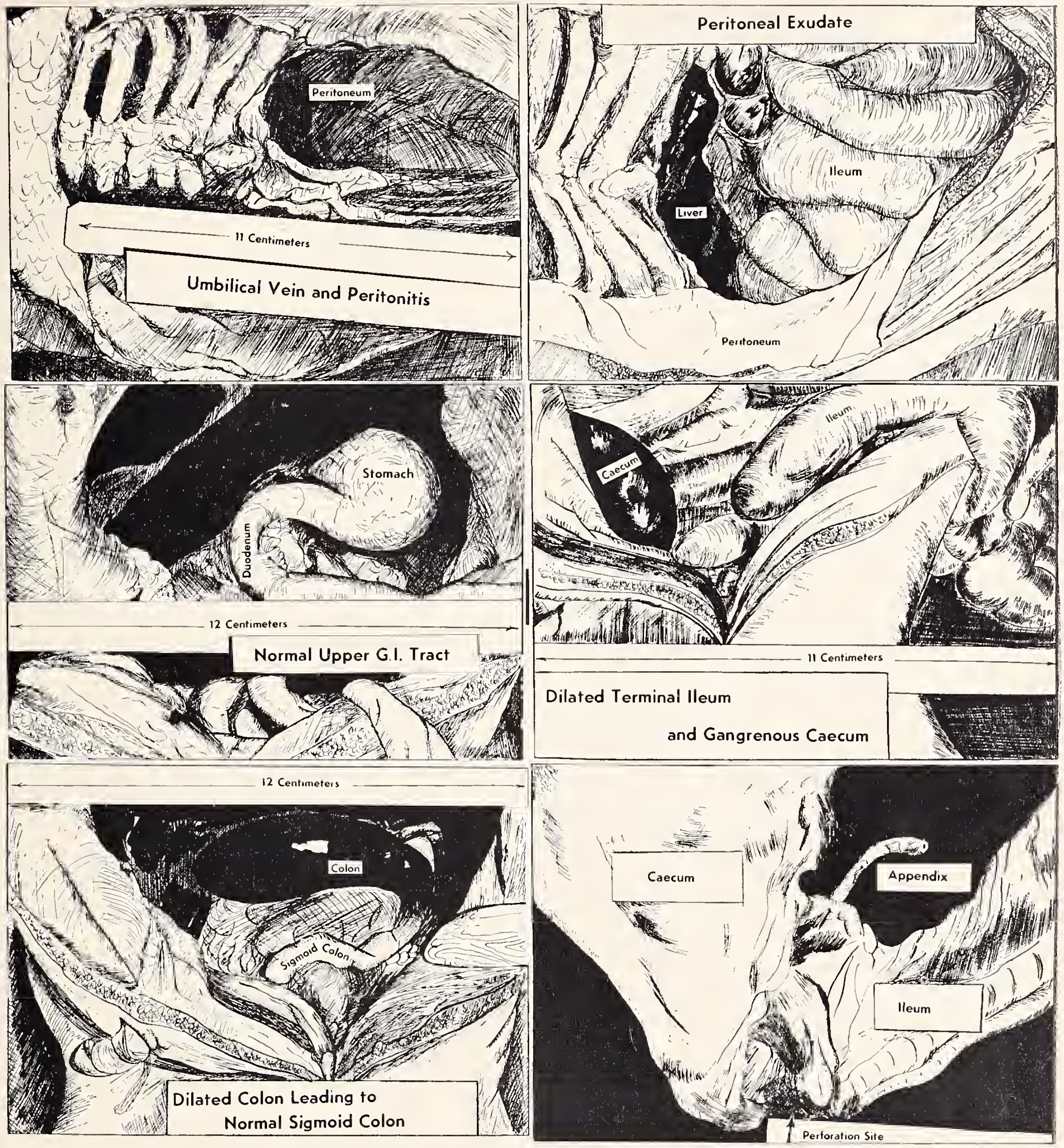
No gross lesions were noted in the stomach, duodenum, jejunum, or first portion of the ileum. The distal ileum, the ascending, transverse, and upper half of the descending colon all showed a green-brown discoloration, and a moderate to marked amount of distention. The distention was particularly noted in the caecum, where an area, 2 centimeters in diameter, on the anteromedial aspect, was black and perforated in its center by a 2 millimeter defect (Figs. 3, 4, 5, 6, and 7). A considerable amount of firm, inspissated meconium was removed from the caecum.

The colon below the junction of the descending colon and the sigmoid was completely normal, and there was no evidence of perforation from the enema tip. The mucosa of the rectum and sigmoid were not damaged.

The liver was covered by a thick, green, purulent exudate. Cut surfaces showed no recognizable gross lesion. Biliary tracts were patent.

Presented at the annual meeting of the Southern Minnesota Medical Association, Winona, Minnesota, September.

Dr. McNear is with the Mankato Clinic, Mankato.


Peritoneum
11 Centimeters
Umbilical Vein and Peritonitis
Peritoneal Exudate
Ileum
Liver
Peritoneum
Stomach
Duodenum
12 Centimeters
Normal Upper G.I. Tract
Ileum
Caecum
11 Centimeters
Dilated Terminal Ileum
and Gangrenous Caecum
12 Centimeters
Colon
Sigmoid Colon
Dilated Colon Leading to
Normal Sigmoid Colon
Caecum
Appendix
Ileum
Perforation Site

Figs. 1-6

Endocrine System.—The thymus was slightly enlarged but was not constricting the trachea or compressing the vessels of the anterior mediastinum. The thyroid was not enlarged. The adrenals were normally located and did not appear enlarged. The cut surface of both adrenals showed a thinning of the cortex, with some evidence of hemorrhage into it.

Diagnosis.—The diagnosis on the basis of gross autopsy findings was: (1) Peritonitis, severe, acute, due to (2) Perforation of the caecum—cause of caecal perforation unknown, and (3) Right heart failure.

The autopsy findings were difficult to understand,

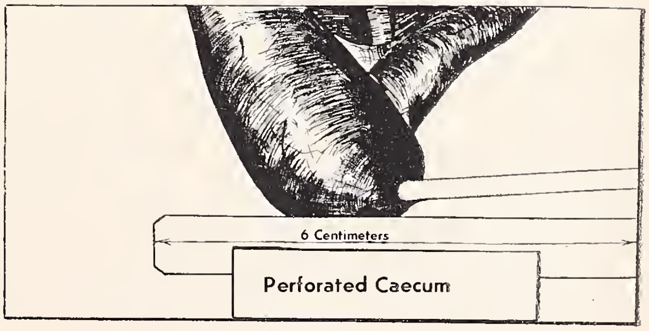

6 Centimeters
Perforated Caecum

Fig. 7

as we were unable to determine any way in which the caecum could have been perforated, and until the microscopic findings were returned, we remained in the dark.

Microscopic findings showed several things of significance. The first was hemorrhage into the adrenal cortex, and, second, gangrene around the site of perforation, apparently caused by compression of the blood vessels of the caecum by some foreign body. This finding of trophic gangrene led us to the diagnosis of meconium peritonitis.

It was felt by the pathologist who did the autopsy, that there was sufficient inspissated meconium in the lower gastrointestinal tract to account for the perforation. Therefore, the final diagnosis, based on gross and microscopic pathology, was peritonitis, due to perforation of the caecum by pressure necrosis from inspissated meconium.

Discussion

In 1838, Sir James Y. Simpson first described fetal peritonitis. At the time of the original description, twenty-five cases were discussed. Since that time several authors have broached the subject of meconium peritonitis, and occasional isolated case reports and reviews have appeared in the literature.[1]

Perhaps one of the best reviews of this condition is published in the *American Journal of Roentgenology and Radium Therapy,* by Dr. Edward B. D. Neuhauser; in this review the radiographic criteria for diagnosis are summarized and examples given. His article gives three additional cases, and all three of these showed one or more congenital anomalies of the bowel, the most common being atresia. In all of his cases one or more irregular plaques of calcification, visible on roentgenogram, were found, and these proved to be meconium, which had been extruded through a perforation and then undergone calcification. However, in no case was he able to demonstrate the site of perforation, either at operation or autopsy.[5]

According to Kneidel,[4] the first description of a case of congenital intestinal obstruction was given by Calder in 1733. Various incidences of this disease have been given; Kneidel's vary from one out of 20,000 births to one out of every 1,000 births by other authors. However, Davis and Poynter[3] indicate that of the 392 cases reported in the literature as of 1922, only 25 per cent (101 cases) occurred in the ileum and caecum. The mechanism of obstruction and perforation associated with meconium ileus has been thought to be related to fibrocystic disease of the pancreas. In this situation, the peristaltic waves passing through the intestine are apparently unable to force the thickened meconium through the lumen of the gut, and obstruction results.

It was pointed out by Neuhauser and Kneidel that the majority of cases of meconium ileus associated with obstruction or perforation were usually the result of one or another congenital lesion of the intestinal tract, associated with faulty development. Pyloric stenosis; ileal and jejunal atresia; diaphragmatic hernia causing gastric obstruction; imperforate anus; duodenal stenosis and/or atresia, were the commonest types of congenital anomalies noted.

In our case, no evidence of congenital malformation of the gastro-intestinal tract was seen. In addition, we were able to demonstrate the exact location of the point of intestinal perforation. Microscopically, we were able to see evidence indicating pressure necrosis from the inspissated meconium. We were not able, however, to find the classical radiological picture of meconium peritonitis, *viz:*

1. One or more irregular plaques of calcification visible in the peritoneal cavity, either lying within the bowel lumen, wall, or parietes.
2. Markedly dilated loops of small bowel with numerous air and fluid levels.

On the other hand, our case did demonstrate the classical x-ray findings of perforation of a hollow viscus, clearly demonstrating air beneath the diaphragm and beneath the abdominal wall.

Because of the failure of the autopsy to demonstrate calcification of the meconium spilled within the peritoneal cavity, it is our conclusion that the perforation must have been quite recent in its origin.

It has been pointed out[2] that it is possible for rupture of the bowel to occur any time after the fifth month of gestation, and that meconium can be spilled any time after perforation occurs. In the fifth month, intestinal peristalsis seems to start and prior to that time the gastro-intestinal tract is silent. Had rupture occurred very long prior to the death of our infant, some evidence of calcification, or fibrotic walling-off of the foreign body, would have been evidenced at autopsy. However, we found only thick, purulent meconium covering the peritoneal surfaces of the abdominal cavity, without any evidence,

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Medical Partnerships

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THERE appears to be a growing interest among physicians and surgeons, both in the field of general practice and in the various specialties, in combining the tangible and intangible resources of two or more doctors for practice as a group or firm. Since the medical profession in common with other professions is generally prohibited from practicing in or through a corporate body, the partnership form of organization seems to be the only practical means of combining resources. For that reason an article touching upon the various problems inherent in a partnership organization and some of the solutions of such problems seems timely.

It is perhaps only natural that most professional and businessmen contemplating the formation of a partnership (and it nearly always involves the combining of the resources and efforts of persons who are good friends, as well as persons with common professional or business interests), think only of three or four matters to be decided at the outset. The questions which occur to them immediately are: What shall we call our firm? What will be our office or business location? How much property by way of cash and equipment will each prospective partner contribute? How will we share in the profits? Strangely enough, there is almost never any thought of losses or future misunderstandings or disputes.

The legal profession has long since learned that misfortunes, misunderstandings and disputes will arise in some partnerships. Not only that, but sooner or later every partnership is going to be terminated with accompanying problems incident to the business aspects of dissolution. For that reason, lawyers generally urge prospective partners to devote great care and thought to their partnership agreement and to reduce it to writing for future certainty.

When Doctor "Jim" and Doctor "Joe" decide that they would like to form a partnership and pool their combined equipment, knowledge, experience and other resources, they should immediately make up their minds that they are undertaking a business relationship, the details of which should be worked out in advance so far as possible. They should sit down and lay aside the sensitivity that so frequently affects discussions between close friends and discuss all the aspects with complete candor.

Their discussions should include among others, the following problems:

1. The firm name under which the partnership will operate and its office location.
2. The date when the partnership shall commence and the term for which it shall continue (usually from year to year until such time as it is terminated as provided in the agreement).
3. The original acquisition of supplies, fixtures and equipment and the repair, maintenance and replacement during the life of the partnership.
4. The definition of just what receipts from particular sources of income shall constitute partnership property.
5. The definition of partnership expenses to be paid out of funds of the partnership.
6. Whether automobiles shall be purchased by the partnership or supplied by the individual partners, and the manner of obtaining and paying for liability and property damage insurance necessary to protect the partnership as distinguished from the individual.
7. The bank in which the partnership bank account shall be established and the responsibilities of the partners with respect to that account.
8. Nature and extent of the records (including books of account) to be kept by the partnership, the place of keeping such books and the rights of each partner with respect to access to all records.
9. Whether or not the firm's books of accounts are to be supervised or checked by in-

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dependent auditors; if so, how often and whether the independent audit report shall become conclusive as to each of the partners within some definite period after it is received.

10. Manner and times of distribution of net income.

11. Times and amount of current drawings by each partner from current income for living and other personal expenses.

12. Manner of termination of the partnership and the methods of liquidation of assets upon dissolution.

13. Treatment of accounts receivable owned by the individuals at the time the partnership is formed.

14. Restrictions, if any, upon each partner as concerns his activities outside the partnership.

15. Amount of vacation time or other free time to be allowed to each partner during the existence of the partnership.

16. The fixing of responsibility, if so desired, for particular types of practice, for example, obstetrics.

17. Any restrictions which are to be placed upon any partner with respect to his practice in the event of termination of the partnership.

The possible solutions to most of the questions or problems enumerated are quite obvious, and will not be the subject of further comment here. However, several of the matters which should be covered in every good partnership agreement seem worthy of brief comment.

Quite frequently medical partnerships comprise an older doctor who owns substantial equipment and controls a substantial practice, and a young doctor whose resources in that respect are small or almost nonexistent. In that situation the supplies, fixtures and equipment with which the partnership will start operation may be furnished by the older doctor either on the basis of an appraised or an agreed value, a prescribed percentage of which the younger man is to pay to the older partner out of future earnings, or the older doctor may contribute these supplies to the partnership with no provision for reimbursement but only a provision that upon termination of the partnership any of the original supplies, fixtures and equipment still in use shall revert to him. Where the physicians or surgeons forming the partnership are on a more equal footing, it is possible that each will have substantial holdings of supplies, fixtures and equipment to contribute, in which event the value can be appraised or agreed upon and any differential adjusted by cash contribution to the partnership account, or such difference in value can be taken into account in the fixing of participation by each partner in profits and losses. In any event, the agreement should further specify the manner of procuring and paying for repairs and maintenance and replacement equipment.

Meticulous care should be exercised in working out the terms of the agreement for distribution of net income (and in the case of misfortune, the sharing of losses) so that each partner will know from time to time exactly where he stands with reference to partnership earnings. In most instances, the partners do not start on an equal footing, and it is intended that the younger partner with less experience and less to offer the partnership participate in earnings (and share in costs and losses) on a basis which starts with a relatively small proportion and increases from year to year until he arrives at a point where his participation is perhaps equal to that of any other partner. It is highly desirable that the partnership agreement set forth precisely the participation of each partner from year to year and that subsequent years not be left to future agreement. If, in years following the commencement of the partnership, it becomes obvious that the provisions respecting distribution of income as originally set forth are inequitable, the agreement can always be amended to express any new arrangement.

An audit of the partnership accounts by an outside firm of accountants may seem like a small matter, but to those of us who have had extensive experience with business and professional firms, it is perhaps one of the most important items for consideration. If the audit is done by competent people, it can serve several purposes. In the first place, the auditor will consider and report any improvements which can be made in the practices of the firm's bookkeeper to make the accounting more efficient and informative. Secondly, the audit should disclose not only inefficiency, if any, in the handling of the business practices of the partnership, but also any possible dishonesty on the part of employes—a problem which exists somewhat more frequently than most persons are aware. In the third place, the audit, if made annually, and the report from the auditor informs each partner as to just what progress the partnership is making and avoids possible necessity for going back over a number of years to settle a dispute as to individual income received from the partnership or costs paid by the partnership and allocated to the individual partners. In the fourth place, the audit is the best possible supporting data for necessary income tax returns of the individual partners, insofar as those returns reflect income and deductions allocable to partnership operations.

It is desirable that the partnership agreement cover the question of termination (a) by notice given by a disgruntled partner a specific length of time before the end of a fiscal year, calendar year or month, (b) by entry into the military service and remaining in such service for a prescribed period of time and (c) by retirement of a partner. The death of a partner, of course, terminates a partnership as a matter of law. Equally important is a provision in the partnership agreement covering liquidation of assets upon dissolution. In the absence of some agreement all partnership assets would be subject to liquidation and the receipts therefrom would be distributed in the proportion of the partners' ownership of the assets and participation in income. Quite obviously this could result in a substantial loss and it is perfectly feasible to provide in the partnership agreement that upon termination the assets of the partnership shall be distributed in a particular manner. For instance, it may be provided that a particular partner shall take all or a specified portion of the equipment and fixtures and the office location. Likewise, it may be provided that the accounts receivable will be liquidated in a particular manner. Specific provisions may be inserted for liquidation in one fashion if the partnership is terminated by notice for military service and in another manner in the case of the death or retirement of a particular partner.

In those instances where a doctor with an established practice forms a partnership with a younger and to some extent untried man, it is particularly important to consider and agree upon any restriction upon the practice of one or another of the partners in the event of termination of the partnership. It is generally considered to be a valid provision provided that the restriction applies only to a reasonable area and extends only for a reasonable time. There would seem to be little doubt that a partnership agreement between two doctors practicing in a town of modest size might well provide that in the event of termination of the partnership, the younger man would be prohibited from practicing in that town or its immediate environs for a period of one, two or possibly three years.

In this article no attempt has been made to touch upon income tax or other tax aspects of the partnership. As a matter of fact, a partnership is not recognized as a legal entity in the same sense that a corporation is so recognized and no separate income tax is imposed upon it unless it elects to be subjected to a corporate income tax under the 1954 Revenue Code. The partnership as such makes an information return disclosing partnership income and partnership expense as well as the net distribution to the partners. The partnership operation does not, therefore, have the effect of imposing a double tax upon the income ultimately realized by the individual partners.

PSYCHIATRIC ASPECTS OF ULCERATIVE COLITIS

(Continued from Page 89)

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Serological Testing for Syphilis in Minnesota

A Recommendation for a More Productive Use of Routine Testing

ANNE C. KIMBALL, HENRY BAUER, AND R. N. BARR
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MINNESOTA has experienced a marked reduction in cases of syphilis since 1946, when 1,844 cases were reported. In 1953 only 327 cases were reported. During this period there has been no appreciable reduction in the number of specimens submitted to the Minnesota Department of Health Laboratories for serological tests for syphilis. These factors led the Division of Medical Laboratories to evaluate its serological testing program to determine how serological testing can best be used as a case finding tool.

TABLE I. COMPARISON OF ENTIRE YEAR AND 4.4 PER CENT SAMPLE

	Entire Year		4.4% Sample	
	No.	%	No.	%
Kline Test Result				
Negative	262,195	95.18	11,550	95.19
Doubtful ± or 1+	5,551	2.02	241	1.98
Positive 2+, 3+ or 4+	7,717	2.80	343	2.83
	275,463	100.00	12,134	100.00
Number of Tests				
Screen test only	254,201	92.28	11,217	92.44
Three tests	17,667	6.41	785	6.47
Five tests	3,595	1.31	132	1.09
	275,463	100.00	12,134	100.00
Overall Testing Result				
Kline or VDRL & Kolmer negative	266,968	96.92	11,762	96.93
VDRL positive & Kolmer positive 3+ or 4+	4,217	1.55	196	1.45
Other combinations of VDRL & Kolmer test results	4,278	1.53	176	1.62
	275,463	100.00	12,134	100.00

A comparative study was made of blood specimens submitted and reported cases of syphilis. To determine from whom specimens were collected, by whom submitted, and for what reasons, data were studied with reference to such factors as age, sex, marital status, whites and non-whites. Findings from the specimen study were correlated with similar data from reported cases. When appropriate, data were compared with the total Minnesota population, using 1950 census figures.[2,3]

Method

Source of the Data.—The study was based on a 4.4 per cent sample of all blood specimens tested in 1952 in the health department laboratories and on all reported cases in which the diagnostic serology was done in the department laboratories. The year 1952 was selected for study as the most recent year available.

A total of 278,357 blood specimens was received in 1952. This included 2,496 (0.9 per cent) untested specimens, which were unsatisfactory due to insufficient serum or hemolysis, and 398 specimens for which the results were not reported because the patient and/or physician were not identified on the data card and could not be subsequently identified. The sample was drawn from the remaining 275,463 specimens which were tested and on which results were reported.

The sample of 12,134 (4.4 per cent) was selected by an appropriate random sampling method to provide a statistically satisfactory representation of the total group of 275,463. The close agreement between the entire year and the 4.4 per cent sample shown in nine comparisons in Table I indicates that the sample error in this study was small. The distribution of the sample in successive thousands of the numerical specimen number series was within expected theoret-

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From the Minnesota Department of Health.

ical sampling limits to give further assurance that the sample error was small.

There were 479 new cases reported in 1952; 104 cases (21.7 per cent) were eliminated from the study because the diagnostic serology was done in laboratories other than those of the Minnesota Department of Health. The study included all of the remaining 375 cases. Age, sex, marital status, color, and patient residence on cases were correlated under similar data from the specimen sample.

TABLE II. AGE DISTRIBUTION

10,674 Specimens in Sample—372 Reported Cases of Syphilis*

	1950 Minnesota Population %	Specimens in Sample %	Percentage of Total Population Tested %	Cases Reported %	Specimens Tested Per Case Found No.
Age 0- 4	11.1	0.9	0.6	0.8	671
5- 9	9.0	0.6	0.5	0.0	—
10-14	7.5	1.0	1.0	0.0	—
15-19	7.0	9.7	11.4	4.6	1382
20-24	7.2	16.5	18.7	8.9	1210
25-29	7.4	14.5	15.9	7.8	1212
30-34	7.1	12.5	14.3	4.0	2027
35-39	6.9	9.8	11.5	3.5	1823
40-44	6.4	7.7	9.9	6.5	779
45-49	5.9	6.3	8.7	9.9	414
50-54	5.7	5.5	7.8	11.0	324
55-59	5.3	4.3	6.6	11.8	238
60-64	4.5	4.0	7.1	9.9	249
65-69	3.5	3.0	6.9	10.8	182
70-74	2.5	1.8	5.9	5.7	209
75+	3.0	1.9	5.2	4.8	261
All Ages	100.0	100.0	8.1	100.0	651

*Age not given on 1460 specimen data cards (12.0%) and on three case report cards (0.8%).

Reported case data came from two sources, case report cards and the data card accompanying the first specimen submitted for laboratory diagnosis. Specimen data came from specimen cards.

Limitations of the Data.—Accuracy of specific data (age, marital status, diagnosis, etc.) for each individual tested and each reported case was limited by the accuracy of the information given on the cards submitted and by the fact that many cards were incomplete.

The specimens tested cannot be considered as representative of the same number of persons because of repeated testings on some individuals and there was no way to determine accurately the amount of repeat testing. For all calculations of the percentage of population tested there has been a correction introduced to allow for repeat testing. In each table, the number on which there was no information in the sample (10 to 15 per cent) is used to represent the extent of repeat testing. It is probable that 10 to 15 per cent is an overcorrection.

Results

Geographic distribution of the testing by patient residence varied from less than two per cent of the population tested in some counties to more than 13 per cent in others.* Variation in testing did not correlate with density of population nor with the number of physicians per 1,000 of population in each county.

In the seventeen counties where less than two per cent of the population was tested, 3.1 reported cases of syphilis were found in 1952 per 100,000 persons. In nine counties where more than 10 per cent of the population was tested, the case rate was 10.7. This increase from 3.1 to 10.7 in cases found with increase in percentage of population tested is not a smooth progressive increase. A correlation study was made of the two variables (per cent of population tested and case rate) using the 87 counties and three large cities. The correlation coefficient was 0.48. It indicates that increasing the percentage of population tested was not accompanied by a proportionate increase in the number of cases found.

Age distribution in five year age groups is shown in Table II. The table excludes 1,460 specimens (12.0 per cent) and three cases (0.8 per cent), because patient age was not given.

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*Counties with more than 13 per cent of the population tested were Itasca, St. Louis, and Koochiching where employe testing is routine for some industries in those areas; but since the specimens were submitted by private physicians and carried no indications to distinguish the employe examination from the usual private practice specimens, this testing was not included in the industrial group shown in Table VIII.

TABLE III. AGE, SEX, WHITE AND NON-WHITE DISTRIBUTION

10,342 Specimens in Sample—372 Cases of Syphilis*

	1950 Minnesota Population		Specimens in Sample		Percentage of Total Population Tested		Cases Reported		Specimens Tested Per Case Found	
	Male %	Female %	Male %	Female %	Male %	Female %	Male %	Female %	Male No.	Female No.
White										
Age 0-14	13.9	13.3	1.2	1.0	0.7	0.6	0.0	0.8	—	772
15-34	14.0	14.4	22.1	30.1	12.4	16.5	7.8	6.5	1788	2940
35-54	12.4	12.3	15.6	13.1	9.9	8.1	12.1	14.8	814	543
55+	9.5	9.2	8.1	6.6	6.7	6.5	27.9	12.6	182	328
White All Ages	49.8	49.2	47.0	50.7	7.4	8.1	47.8	34.7	620	924
White Both Sexes	99.0		97.7		7.8		82.5		747	
Non-White										
Age 0-14	0.17	0.16	0.08	0.08	3.7	3.8	0.0	0.0	—	—
15-34	0.16	0.15	0.59	0.77	29.5	40.9	5.4	5.6	69	85
35-54	0.12	0.10	0.28	0.30	19.2	24.3	3.0	1.0	58	170
55+	0.08	0.06	0.17	0.03	18.2	3.8	2.4	0.0	43	—
Non-White All Ages	0.53	0.47	1.12	1.18	17.2	19.8	10.8	6.7	65	109
Non-White Both Sexes	1.0		2.3		18.5		17.5		82	

*Age, color, or age and color not given on 1792 (14.8%) specimen cards. Age not given on three case report cards.

Very little blood testing was done and only three cases of syphilis (0.8 per cent) were found in persons from birth to fourteen years of age. The largest amount of blood testing (43.5 per cent) was done in the three age groups twenty to thirty-four years, inclusive. From 14.3 to 18.7 per cent of the Minnesota population was tested in these three groups but they accounted for only 20.7 per cent of syphilis found. Most of the cases reported (54.0 per cent) came from persons aged fifty or above. The number of specimens tested per case found showed a marked change at age forty. Below age forty, 1,440 specimens (weighted average) were tested per case found; above age forty, there were 320 specimens (weighted average) tested per case found; in the age group sixty-five to sixty-nine years there were 182 specimens tested per case found.

Distribution by age and sex in the white and non-white groups is shown in Table III, with age distribution shown in four large age groups, birth to fourteen years, fifteen to thirty-four, thirty-five to fifty-four, and fifty-five and older. The table excludes the 1,792 specimens (14.8 per cent) and the three cases for whom data were not available. The population in Minnesota is 99.0 per cent white,[2,3] and 97.7 per cent of the testing was on this group. Whites accounted for 82.5 per cent of syphilis found. The state population of non-whites is 1.0 per cent,[2,3] and 2.3 per cent of the testing was done on this group. Non-whites accounted for 17.5 per cent of the syphilis reported.

The yield of reported cases in non-whites was one case per 82 specimens, as compared to one case per 747 specimens in whites. In both the whites and non-whites, the heaviest testing of the population was in the fifteen to thirty-four year age groups. They were the only age groups in which there was significantly more testing of females than males, with routine pregnancy examinations accounting for this increase. Of the non-white female population age fifty-five and above, 3.8 per cent were tested, with no cases found.

Table IV shows the various types of syphilis reported by age groups in the white and non-white groups. Primary and secondary syphilis were found largely in young adults; twenty-six of the thirty-seven cases were found in the age group fifteen to twenty-nine years. The asymptomatic types, including early latent, late latent, and asymptomatic central nervous system syphilis, were found in adults of all ages but predominantly above the age of forty. Congenital syphilis was reported in all age groups, but nine of the 18 cases were found in the fifteen to twenty-nine-year-old group. In the white group, 77.7 per cent of the cases were found in persons over age forty, and in the non-white group 33.3 per cent of the cases were found over age forty. Of the total cases in whites, 7.7 per cent were infectious; 20.0 per cent of all cases in non-whites were infectious.

Table V presents the distribution with reference to marital status. It excludes a total of 1,470 specimens, including 259 children and 1,211 (10.2 per cent) adults where marital status was not known, and three cases of congenital syphilis in children aged birth to four. The sample indicates

TABLE IV. CASES OF SYPHILIS AS REPORTED BY TYPE, AGE AND COLOR

	Infectious		Asymptomatic			Late Active			Congenital No.	Total No.	White No.	Non-White No.
	Primary No.	Secondary No.	Early Latent No.	Late Latent No.	Asympt. C.N.S. No.	Other C.N.S. No.	Cardio-Vascular No.	Other Late No.				
Age 0-14									3	3	3	0
15-29	14	12	29	11	3	1			9	79	42	37
30-39	4	2	5	13	2				2	28	21	7
40-49	4		3	41	2	8	1		2	61	52	9
50-59	1		5	42	14	14	6	1	2	85	81	4
60+			2	61	5	18	23	7		116	108	8
Age Unknown				1	1	1				3	3	
All Ages	23	14	44	169	27	42	30	8	18	375	310	65
White	17	7	24	149	25	37	29	8	14	310		
Non-White	6	7	20	20	2	5	1		4	65		

TABLE V. MARITAL STATUS AND SEX

10,664 Specimens in Sample, Age 15 Years and Older
372 Reported Cases of Syphilis*
1950 Minnesota Census Figures, Age 14 Years and Older

	1950 Minnesota Population %	Specimens in Sample %	Percentage of Total Population Tested %	Cases Reported %	Specimens Tested Per Case Found No.
Male					
Single	14.8	13.7	10.1	14.2	623
Married	32.5	31.1	10.6	34.4	589
Wid. & Div.	2.8	2.0	7.8	10.5	125
All Males	50.1	46.8	10.3	59.1	515
Females					
Single	11.3	10.1	9.9	5.4	1231
Married	32.4	39.3	13.3	25.3	1010
Wid. & Div.	6.2	3.8	6.8	10.2	244
All Females	49.9	53.2	11.7	40.9	847

*1470 specimens eliminated: 259 from children and 1211 (10%) with marital status not given. Three cases eliminated: age 0-4.

that 7.8 per cent of widowed and divorced males and 6.8 per cent of widowed and divorced females were tested; 10.5 per cent and 10.2 per cent of cases were found in these males and females, respectively. A new case was found by testing 125 specimens from widowed and divorced males and 244 specimens from widowed and divorced females.

Table VI shows the reason specimens were submitted and the number of cases reported. One-half per cent of the specimens were submitted for "Following Progress of Anti-luetic Treatment." Six cases of syphilis were found in this group.*

Only 0.6 per cent of the specimens indicated "Suspected Case of Syphilis"; fifty-five cases were found in this group. This was the most productive group tested with twenty-eight specimens tested per case found. The fifty-five cases provided 59.5 per cent (22) of the total number of thirty-seven cases of infectious syphilis, with fourteen primary and eight secondary.

Routine pregnancy examinations constituted 11.9 per cent of the testing, with a total of 32,915 specimens. (The sample represents an estimated 33 per cent of all pregnancies, based on a total of 80,380 live and still births resulting from an estimated 100,000 pregnancies.) Only eleven cases of syphilis were found by routine pregnancy examinations, to give a ratio of 2,992 specimens per case found. Of these eleven cases, two were infectious, seven asymptomatic, and two congenital.

"Routine in General Physical Examination" was the reason marked on 57.4 per cent of the specimen cards. This included a large proportion of specimens from hospital patients and therefore does not indicate that all these specimens were from well persons. The groups for which there was "No Reason Marked" constituted 29.6 per cent of the specimens. These two heterogeneous

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*Ideally one would not expect to find this reason on any first specimen prior to diagnosis. Possible explanations of this small bizarre group are: (1) the card was so marked by mistake, (2) the physician treated the case prior to testing, or (3) a test was done prior to diagnosis in other laboratories but was not included in the record of the case on file.

TABLE VI. REASON FOR SUBMITTING SPECIMENS

12,134 Specimens in Sample—375 Reported Cases of Syphilis

Reason	Specimens in Sample	Type of Syphilis Reported									Total Cases Reported		Specimens Tested Per Case Found
		Infectious		Asymptomatic			Late Active						
	%	Prim. No.	Sec. No.	E. Lat. No.	L. Lat. No.	Asym. C.N.S. No.	Other C.N.S. No.	Cardio-Vas. No.	Other Late No.	Cong. No.	No.	%	No.
Following Antiluetic Treatment	0.5				2		1	1	1	1	6	1.6	235
Suspected Case of Syphilis	0.6	14	8	10	12	1	4	5		1	55	14.7	28
Routine in Pregnancy	11.9	1	1	4	2	1				2	11	2.9	2992
Routine in General Physical Examination	57.4	3	5	21	80	16	16	11	4	3	159	42.4	995
No Reason Marked	29.6	5		9	73	9	21	13	3	11	144	38.4	566
All Reasons	100.0	23	14	44	169	27	42	30	8	18	375	100.0	735

TABLE VII. PHYSICIANS OR INSTITUTIONS SUBMITTING SPECIMENS

12,134 Specimens in Sample—375 Reported Cases of Syphilis

	Specimens in Sample	Type of Syphilis Reported									Total Cases Reported		Specimens Tested Per Case Found
		Infectious		Asymptomatic			Late Active						
	%	Prim. No.	Sec. No.	E. Lat. No.	L. Lat. No.	Asym. C.N.S. No.	Other C.N.S. No.	Cardio-Vas. No.	Other Late No.	Cong. No.	No.	%	No.
Private Physicians Offices													
Non-city	23.2	4	3	2	26	6	4	7	3	4	59	15.8	1081*
City	30.3	10	7	20	51	8	11	5	4	3	119	31.7	702
Total Private Physicians	53.5	14	10	22	77	14	15	12	7	7	178	47.5	828
Hospitals	24.7	7	3	14	61	9	24	15	1	8	142	37.9	483
Physicians Submitting Specimens from Industry or Corp.	14.7	1	1	4	15	1				1	23	6.1	1757
Colleges	3.7			2		1					3	0.8	3396
Miscellaneous**	3.4	1		2	16	2	3	3		2	29	7.7	324†
Total	100.0	23	14	44	169	27	42	30	8	18	375	100.0	735

*Includes an unidentifiable number of specimens from "employment" examinations.

**Public clinics, penal institutions, blood donor centers.

†324 specimens tested per case found can be changed to 3338 if we consider the screen testing of specimens done at the two large donor centers prior to sending selected specimens to the state laboratories.

groups accounted for 87.0 per cent of the specimens and 80.8 per cent of cases (303). All types of syphilis were found in these groups.

Table VII presents the data by the source of the specimen. Private physicians provided 53.5 per cent of the specimens tested; 47.5 per cent of all reported cases; and 64.9 per cent (24) of the thirty-seven infectious cases. Specimens tested per case found was 828.

Specimens from hospitals made up 24.7 per cent of the sample; 37.9 per cent of the reported cases; and 27.0 per cent of the infectious cases. Specimens tested per case found was 483.

Specimens submitted by physicians examining for industry or corporations made up 14.7 per cent of the testing and 6.1 per cent of the reported cases. Specimens tested per case found was 1,757.

Specimens from colleges made up 3.7 per cent of the sample and 0.8 per cent of the reported cases. Specimens tested per case found was 3,396. Two of the three cases were college employes. Specimens tested per case found among college students was about one in 10,000.

In regard to the types of syphilis, half of the eighty cases of late active syphilis were found in hospitalized patients, with twenty-four central nervous system, fifteen cardiovascular and one syphilitic cirrhosis. Syphilis cases found by testing specimens from industry, corporations, and colleges were largely of the asymptomatic types.

Because of the low case yield, the pregnancy group and specimens submitted by physicians examining for industry or corporations were further analyzed as to age, previous specimen information, and white and non-white distribution as shown in Table VIII.

In pregnancy testing, more cases were found in the fifteen to twenty-four-year-old group than in any other age group, with 1,646 specimens tested per case found. Above this age, only three cases were found, with 5,448 specimens (weighted average) tested per case found. On 30.7 per cent no previous test had been done; eight of the eleven cases were found in this group. A previous specimen had been tested on 10.1 per cent of the total group; no cases were found in this group. On 59.2 per cent of the sample, no information was given as to a previous test and three cases were found in this group. A marked difference was noted between the white and non-white groups; 6,315 specimens were tested per case found in the white group and only ninety-five specimens per case found in the non-white.

Study of the specimens from physicians examining for industry or corporations revealed that the least productive age group was fifteen to twenty-four years, with 4,938 specimens tested per case found. Progressively fewer specimens were tested per case found in older persons; in the fifty-five to sixty-four year old group, 322 specimens were tested per case found. Only 1.7 per cent indicated that a previous specimen had been tested; no cases were found in this group. A comparison of white and non-white groups reveals that 3,269 specimens were tested per case found in the white group and seventy-eight specimens tested per case found in non-whites.

Discussion

The marked reduction in the number of reported cases of syphilis in Minnesota suggests that the situation is well enough in hand at the present time to consider the possibility of making more selective use of serological testing, with effort directed toward those groups which are most productive in terms of case yield.

Indiscriminate Routine Testing.—Routine testing of many population groups, including persons applying for marriage licenses,* food handlers, applicants for employment, and all hospital admissions has been recommended[5] but it has not been a procedure that is applicable in Minnesota where syphilis is as well under control as it is. If all of these groups were routinely tested, it would involve an estimated total of one million specimens a year, or the equivalent of one-third of the total population, with much unnecessary and expensive repeat testing of non-syphilitic persons.

Aspects of this study relating to geographic distribution of testing by patient residence indicated that increasing the percentage of population tested does not show a proportionate increase in the number of cases found. A better procedure appears to be to limit routine testing to groups in which syphilis is more often found and to emphasize the need for a high level of suspicion of syphilis on the part of the physician in selecting other persons for testing. Data presented in this study may assist in that selection.

Limited Routine Testing.—In this study, the groups in which syphilis was frequently found were: suspected cases, widowed and divorced persons, non-whites, and whites over age forty. Specimens from the above groups comprised 40 per cent of the specimen load, and 331 or 88.3 per cent of the syphilis case load.

The yield of reported cases in suspected cases was one case per twenty-eight specimens tested; for widowed and divorced persons, one case per 184 specimens; for non-whites, one case per eighty-two specimens; for persons over age forty, one case per 320 specimens.

With reference to marital status, widowed and divorced adults represent the smallest population group in the state. The extent to which specimens were submitted represents an even smaller percentage of the blood testing done. (Table V). However, these groups, both male and female, made up 20.7 per cent of cases found. It appears that this group is not being tested to the extent that other marital groups are tested, but it is yielding more than its proportionate share of cases. The fact that 5.8 per cent of the testing revealed 20.7 per cent of the cases suggests the need for greater testing in this group.

Another group in which there appears to be inadequate testing is the non-white female population aged 55 and over where only 3.8 per cent were tested (Table III). The fact that no cases were found in this age group would seem to indicate not so much that there was less syphilis in this group but that there was inadequate testing of these women.

Only 7.6 per cent (weighted average) of the population over 40 years of age is being tested at the present time, but all data indicated that age

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*Premarital testing is a legal requirement in many states. At the present time, approximately one per cent of all specimens examined by the Minnesota Department of Health are to meet the premarital requirements of other states. It is estimated that the cost of processing a premarital specimen is from four to five times greater than the cost for processing the average specimen.

TABLE VIII. AGE, PREVIOUS SPECIMEN INFORMATION AND WHITE, NON-WHITE DISTRIBUTION IN PREGNANCY TESTING AND SPECIMENS FROM PHYSICIANS EXAMINING FOR INDUSTRIES AND CORPORATIONS

	Pregnancy Testing				From Industry and Corporations			
	Specimens in Sample		Cases Reported	Specimens Tested Per Case Found	Specimens in Sample		Cases Reported	Specimens Tested Per Case Found
	No.*	%	No.	No.	No.*	%	No.	No.
Age Distribution								
Age 15-24	580	44.6	8	1646	435	25.4	2	4938
25-34	578	44.5	2	6560	608	35.5	6	2300
35-44	142	10.9	1	3223	388	22.7	5	1762
45-54	0	0.0	0	—	200	11.7	5	908
55-64	0	0.0	0	—	71	4.1	5	322
65+	0	0.0	0	—	11	0.6	0	—
All Known Ages	1300	100.0	11	2683	1713	100.0	23	1691
Age Unknown	150		0	—	67		0	—
Entire Group	1450		11	2992	1780		23	1757
Previous Specimen Information								
No Previous Test	445	30.7	8	1263	749	42.1	4	4251
Previously Tested**	146	10.1	0	—	29	1.7	0	—
No Information	859	59.2	3	6500	1002	56.3	19	1197
Entire Group	1450	100.0	11	2992	1780	100.0	23	1757
Color								
White	1391	98.2	5	6315	1728	97.8	12	3269
Non-white	25	1.8	6	95	38	2.2	11	78
Color Known	1416	100.0	11	2922	1766	100.0	23	1743
Color Unknown	34		0	—	14		0	—
Entire Group	1450		11	2992	1780		23	1757

*$\frac{\text{No.} \times 22.7}{111 \text{ cases.}}$ =specimens tested per case found.

**In pregnancy group, 145 negative and one doubtful. In industrial group, 26 negative, one doubtful, two positive.

was a significant factor. The number of specimens tested per case found showed a marked change at age forty. (Table II). The same trend was found in specimens examined from industry or corporations (Table VIII), in which progressively fewer specimens were tested per case found in older persons. Only 34.5 per cent of the testing is from persons above the age of forty but 262 (70.4 per cent) of the 375 cases of syphilis were found in this group (Table IV).

Selected Testing.—Syphilis was not frequently found by routine pregnancy examinations; a total of 2,992 specimens were tested per case found (Table VI). Table VIII shows that few cases were found in the older pregnant women, no cases were found in the previously tested women, and few cases were found in the white group. Data available on routine pregnancy examination did not reveal which pregnancies were primipara and which were multipara, but it seems reasonable to presume that the majority of pregnant women being tested a second time were tested in a previous pregnancy.

With reference to reasons for submitting specimens, Table VI shows that the most productive group tested was "Suspected Case of Syphilis." This group provided 14.7 per cent of all cases and 59.5 per cent of the cases of infectious syphilis. In twenty-two of the thirty-seven primary and secondary cases, syphilis was suspected prior to the first blood test. Of the remaining fifteen infectious cases (40.5 per cent), seven would have been found by the suggested limited routine testing. Only eight would have been missed.

Any reduction in serological testing would be unwise from the point of view of the infected individual or from that of public health if primary or secondary cases were missed. With these cases, the lesions, symptoms, history of exposure, and darkfield examination carry more diagnostic weight than the blood test, which is primarily confirmatory. If a detailed history and physical examination did not reveal findings suggestive of syphilis and the suspicion of the disease was aroused only after the positive blood report was received, then we must continue to encourage widespread blood testing, even though it means testing several thousand bloods to find each case. On the other hand, if the suspicion of syphilis preceded the blood testing but was not so indicated on the card, we need not rely on routine testing to find 40.5 per cent of the cases of infectious syphilis, but on selection by the examining physician.

On only 1,521 specimens was the data card checked "suspected case" indicating syphilis was considered prior to drawing the specimen. Since syphilis is a protean disease, should it not be considered or suspected in taking a history or doing a physical especially in the ages over forty, rather than casually checking the data card "routine exam"?

The types of syphilis most dependent on serological testing for diagnosis are early latent, late latent, and asymptomatic central nervous system syphilis. During recent years, newly diagnosed cases of syphilis in Minnesota consist very largely of these types. In this study, 240 (64.0 per cent) of the 375 cases were of these asymptomatic types. Table IV shows that these types were found in adults of all ages, but most of them were aged forty and over. Only 34.5 per cent of the testing was from persons above the age of forty but 177 (73.8 per cent) of the 240 cases of asymptomatic syphilis were found in this group. Finding these cases is of great importance to the individual, since the disability of late active syphilis can often be prevented. Prevention of early death or permanent disability from cardiovascular or central nervous system syphilis has very tangible public benefit.*

In a sense, the eighty cases of late active syphilis in the total group of 375 cases may be considered public health failures since they were not found when the disease was in its infectious or asymptomatic state. In the same sense, congenital syphilis cases are also public health failures.

Although a single case of either does represent a public health failure, a low case rate for late active and congenital syphilis is a measure of the effectiveness of the syphilis control program not only for the present time, but also for the last ten or twenty years. It is encouraging to note the continued reduction in the failure rate (late active and congenital syphilis per 100,000) since 1940 when the failure rate was thirty-two. In 1947 the rate had fallen to 10.2. In 1952, the rate was only 3.5. These rates indicate that for more than a decade most cases of syphilis have been found in time to prevent late active syphilis and congenital syphilis. We can look forward to an even smaller proportion of such cases in the future if we can continue to find and treat infectious and asymptomatic cases.

Death certificates provide additional evidence of the completeness of coverage of the syphilis control program, with a trend toward more complete coverage strongly indicated. As recently as 1947, syphilis was the major or contributory cause of death on 135 death certificates, but of this group, only forty-four cases were unknown previous to the time of death, as checked against the Minnesota record of reported cases. In 1953, with eighty-four deaths in which syphilis was indicated as the major or contributory factor, only sixteen cases were unknown prior to death.

One of the reasons for the success of the syphilis control program in Minnesota is the excellent co-operation of physicians in private practice over a period of many years. The State Board of Health has always taken the position that a realistic approach to the control of all communicable disease, including syphilis, is to keep diagnosis and treatment in the hands of private physicians to the extent possible. With reference to syphilis, this has been the active policy of Dr. H. G. Irvine, administrator of the program since it was initiated in 1918. Data in this study reveals the effectiveness of this policy. Table 7 indicates that private physicians provided 53.5 per cent of the specimens tested; 47.5 per cent of all reported cases; and 64.9 per cent of the infectious cases. Hospitals made up 24.7 per cent of the sample; 37.9 per cent of the reported cases; and 27 per cent of the infectious cases. In combination, these two sources provided 78.2 per cent of specimens tested; 85.4 per cent of all reported cases; and 91.9 per cent of all infectious cases. Because many cases found in hospital patients and industrial examinations are later diagnosed and treated by private physicians, more than 70 per cent of actual case reports were from physicians in private practice.† The excellent record of most private physicians in case finding carries over into follow-up on source and contacts, and physician co-operation in this phase of the work continues to be invaluable to this department in its epidemiological work.

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*This is particularly true of general paresis which if not prevented may cost the public as high as $11,000 for the institutional care of an average case. From January 1, 1951, to May 1, 1954, 74 people died in state mental hospitals with syphilis as a cause or contributory cause of death. These people spent an average of 10.3 years in the state hospitals. The annual cost of care for one year in these institutions is $1,051 (weighted average for eight hospitals).[1]

†Minnesota physicians may be interested in knowing that this is not the usual ratio. For the fiscal year of 1953, case reports of syphilis from private physicians in the United States exceeded 70 per cent of the total case reports in only nine states; these nine states included Minnesota and only two other states with populations exceeding two million.[4]

Conclusions

Selection of persons to be serologically tested for syphilis can be improved. Most of the blood testing in this study was in the age groups twenty to thirty-four, with these groups accounting for 43.5 per cent of the blood testing but only 20.7 per cent of the cases. Most of the cases were found in the age groups forty and over, and in this study these persons accounted for 70.4 per cent of the cases. Testing in those groups in which syphilis was more frequently found (suspected cases, widowed and divorced, non-whites, and whites over age forty) accounted for 40 per cent of the specimen load and 88.3 per cent of the case load. It would seem that there is need to test a greater percentage of the population in those groups where more cases are found.

Recommendations

1. Indiscriminate routine testing should be discouraged, but routine testing of selected groups is strongly recommended.

2. It is urged that routine testing be increased in those groups in which syphilis was more frequently found (suspected cases, widowed and divorced persons, non-whites, and persons over age forty). It is recognized that repeat testing following a negative in the older age groups will usually be a relatively non-productive procedure.

3. Because of the complexities of syphilis in pregnancy it is advisable to continue to recommend routine prenatal testing in spite of the low case yield. The limited data indicated that pregnant women having had a previously negative test were still negative. A more comprehensive study of this group may give criteria to guide selective testing of pregnant women.

4. The physician should suspect syphilis at the slightest provocation. When clinically demonstrable syphilis is seen less frequently a higher level of awareness is required if early cases are not to be overlooked.

5. Continuing studies of this kind will aid all persons with a concern for the effective control of syphilis in focusing their efforts on those segments of the population in which more cases are found. Future studies can be improved by:

(a) A redesigned data card which will give more accurate and complete information.

(b) Co-operation of physicians in supplying all information requested on specimen and case report cards.

Acknowledgment

The authors are indebted to Dr. J. Bearman, Biostatistician, University of Minnesota School of Public Health, for advice on sample size, and selection, and to the Division of Biostatistics for the correlation studies that were a part of this research.

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SPECIALIZATION?

"When a workman is unceasingly and exclusively engaged in the fabrication of one thing, he ultimately does his work with singular dexterity; but at the same time he loses the general faculty of applying his mind to the direction of the work. He every day becomes more adroit and less industrious; so that it may be said of him that in proportion as the workman improves, the man is degraded.

What can be expected of a man who has spent twenty years of his life making heads for pins? And to what can that mighty human intelligence which has so often stirred the world be applied in him except it be to investigate the best method of making pins' heads? When a workman has spent a considerable portion of his existence in this manner, his thoughts are forever set upon the object of his daily toil; . . . he no longer belongs to himself, but to the calling he has chosen . . ."—de Tocqueville's *Democracy in America*.

Suffocation, A Killer of Children

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THIS article is one of a series on accidents and accidental deaths in children. The purpose is to inform the readers of MINNESOTA MEDICNE of the nature and magnitude of accidents in children and to encourage the development of "accident prophylactic-minded" doctors. I have been asked to discuss under the heading of suffocation the particular problems of crib deaths, hangings and ice-box deaths.

The wide publicity given to deaths of children in iceboxes has had a good effect. In many communities, laws have been made to prevent the abandonment of iceboxes in places where children can get at them. Clubs and groups have volunteered to dispose of old iceboxes. The doctor is in a good position to see that his community is free of the icebox menace. Minnesota has been free of icebox deaths so far. Similar type accidents, with suffocation occurring in cedar chests or closets, have happened in Minnesota.

Hanging deaths in children seem to fall into categories of bizarre accidents or suicides. The accidents are sometimes too realistic re-enactments of cowboy stories and the like. Children should be taught that ropes, like fire, can be dangerous. Suicide is occasionally a spur of the moment act of a hurt or angry child; more often there will be other evidences of psychiatric disturbance. Do not ignore the threats of a child who talks of suicide! He may fool you, and himself.

In 1952, twenty-seven infant deaths in Minnesota were attributed to suffocation in the crib. In only three instances were autopsies done. Evidence obtainable from other states where autopsies are done routinely would indicate that most of Minnesota's twenty-seven deaths were not due to suffocation. Werne and Garrow[3] from the office of the Chief Medical Examiner of the City of New York, reported on a series of 299 sudden deaths in infants under one year of age over a period of twenty years in one borough of the City of New York. These authors, probably more than anybody else, helped to clarify the causes of death in such instances. For many years, most of such deaths were believed to be caused by suffocation, aspiration, or an enlarged thymus. Nobody in authority any longer supports the concept of "status thymolymphaticus" or thymic deaths. Aspiration too has lost repute as a cause of death. It has been shown to be a common finding in instances of death due to other causes.[4] Suffocation undoubtedly occurs, but the evidence says that most crib deaths are due to infection. Gruenwald and Jacobi[2] found mononuclear pneumonia very commonly in infants who died suddenly after being in apparent good health or who were ill for only a short time. Werne and Garrow[5] had autopsy examinations in three different categories of infant deaths. The first group was thirty-one infants who died suddenly while in apparent good health. The second group was twenty-six infants who were actually observed while they died suddenly while in apparent good health; and the third group was twenty-six infants who died violent deaths. Many of the first group of thirty-one infants were thought to have died from suffocation. Careful microscopic examination of the tissues showed that all the infants had respiratory disease. In twenty-six infants dying violent deaths, evidence of respiratory infection was spotty and mild. In the group of infants who were observed while dying, ten had heart disease or gross bronchopneumonia and the remaining sixteen infants all showed microscopic respiratory infection. The significant points in these studies were:

1. Most sudden deaths in infancy and especially below the age of six months are due to infections.
2. True suffocation deaths are rare.
3. Aspiration is a common agonal event but seldom is the cause of death.
4. Mononuclear cells may be present in the

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alveolar bronchiols in several different conditions and are not diagnostic of pneumonia.

Streptococci, staphlococci, meningococci and E. Coli septicemia have all been shown to cause sudden death in infants.[1]

I am confident that most doctors who care for babies have seen instances where suffocation did seem to be the cause of death. The baby is found face down in a soft pillow, or bed clothing is wound about the head, or the head is caught between the rungs of the crib with the face against the mattress, or the baby has slid between the rungs or between the crib and the mattress and the baby has strangled. Some of these deaths can be prevented. The doctor must preach safety in the crib as part of his post-natal instructions to the mother. The mattress must be firm, it must fit the crib well with no spaces on the ends or sides; pillows must not be used. The crib sides should be solid or the rungs spaced closely.

The doctor must also be alert to minor symptoms in the small infant as warnings of possible serious disease. The immune mechanisms of the small infant are poor. Chemotherapy or antibiotic therapy may be prescribed without all the criteria that the conscientious doctor may demand in an older patient.

If death does occur in the crib, then the doctor must try to find the true cause of death. To learn that infection rather than suffocation caused their baby's death may spare the parents profound feelings of guilt. Autopsies should be done on all infant crib deaths.*

Doctors need to be accident-conscious. Accidents kill many more children than diphtheria, tetanus, pertussis and smallpox. The doctor does not consider his work done unless his child patients are immunized against these diseases. He should not consider his care adequate until he has inoculated the germ of accident prevention into both child and parents. This can be done at his office, as part of the first-year, well-baby instructions. Brochures and check-lists to supply to the parent can be obtained from the Minnesota State Department of Health, Section of Maternal and Child Health, University Campus, Minneapolis, Minnesota.

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*If the services of a local pathologist are not readily available, the doctor may remove tissue specimens and send them to the Pathology Department, University of Minnesota Medical School, where tissue preparations and interpretations of the pathological findings will be made without charge.

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MECONIUM PERITONITIS

(Continued from Page 92)

either gross, microscopic, or radiographic, of organization or calcification.

We came to the conclusion that Kneidel's recommendations, as to the value of a scout film of the abdomen, should be taken whenever a newborn infant shows persistent vomiting, abdominal distention, or fails to gain weight in a normal fashion.

Acknowledgment

Acknowledgment is gratefully given to the medical illustrator, J. E. Prince, United States Air Force, who was able to make black and white drawings from the Kodachromes taken at autopsy.

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Teratoma of the Recto-Sigmoid

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TERATOMAS are embryonic tumors which are characterized by the fact that they contain tissues from one or all of the three germ layers; therefore they contain skin, hair, bone, cartilage, muscle, teeth, nerve tissue, fat, intestinal mucosa and other organs. There is no order in the arrangement of tissues, as they are a veritable pot-pourri of fetal tissues. The tissues may be adult or embryonal type. The former are prone to give rise to benign tumors, and the embryonal elements are more apt to give rise to malignant tumors. The nature of these tumors can be understood best in the light of their mode of formation. They are apparently due to the partial separation of a part of the ovum in the blastomere stage. The younger the ovum is, the more nearly totipotent its cells are, and the more capable of producing more than one tissue. In the blastomere stage a small part of the dividing ovum is partly separated and continues to live as a mass of cells, attached to the host and causing no disturbance until for some unknown reason this embryonic tissue is stimulated to independent growth. They are therefore faulty efforts at twin formations.

Depending upon the age of the blastomere and multipotency of its cells, teratomas of any simplicity or complexity may be formed. In dermoid cysts, the tissue is more adult and more specialized than in other teratomas; organs are better formed and more adult in type. In other teratomas, the tissue is often completely unorganized, may be a solid mass, and organs are more rudimentary and embryonic. Teratomas may be found anywhere in the body but have a predilection for certain locations, especially the ovary. Teratomas of the posterior rectal wall space were described by Middeldorf as early as 1885 and still frequently are referred to as Middeldorf tumors. Many cases have been reported of numerous variations of this tumor in the medical literature, but nowhere in the American recordings have I been able to find a case of teratoma arising from the mucous membrane of the pelvic colon. One case of dermoid arising from rectal wall was found recorded in the *British Journal of Surgery* by R. Nigam in 1947, in which the author stated that the mass protruded from the anus and was 4 by 2 inches in size. It was attached to the anterior rectal wall by two pedicles one-half inch thick and about 5 inches from the anal margin. There was no obvious connection between the mass and any of the pelvic viscera other than the rectal wall.

Another case was reported in Bland-Sutton's monograph on tumors in 1922 of London, England. They found a pedunculated teratoma hanging from the mucous surface of the sigmoid flexure which led to intussusception in a girl of sixteen years of age.

Dermoids in the wall of the rectum are rarely met with and the majority of cases reported so far have been of the nature of dermoid cysts. The following case is of interest because the tumor was a solid mass containing bony rudiments and glands covered with hairy skin, and microscopically a true teratoma.

Case Report

Mrs. L. P., aged twenty-seven, a white woman, presented herself on January 9, 1953 with the following history:

Since the age of approximately fourteen years this patient had noted a mass presenting at the anus whenever she had a bowel movement. As time went on, this mass became gradually larger and larger producing symptoms of pressure in the rectum; and finally, in recent months, produced so much obstruction that it was necessary for her to push this mass back into the rectum each time before she could evacuate her bowel. She had consulted a physician several years ago after passing blood for the first and only time, and after examination he reassured her there were only a few external hemorrhoids present which did not necessitate any treatment.

The family history was essentially negative. The past

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history of illness included measles, mumps, and chicken pox. She had had a tonsillectomy at age fourteen. The menstrual onset was at age 11 years, and was regular and normal. Dysmenorrhea was present during the first day. She had been married for eight years and has not been able to conceive to date, even though examination of the husband's sperm was normal.

On examination this patient was a well-developed female 63 inches tall, weighing 146 pounds. The hemoglobin was 11.8 gms. white blood count 7,000, Wassermann negative, and urinalysis negative. General physical examination was essentially normal. The genital pelvic organs were normal except for a large firm mass which was palpable in the rectum through the posterior vaginal floor and on digital rectal examination. On the latter examination, the pedicle attached to the right lateral rectal wall was palpable; the mass seemed to be about the size of a large hen's egg, and there was a sharp calcified structure palpable in one area of this mass. On proctoscopic examination this mass was partially visualized and the pedicle attachment was readily recognized. The passage of a sigmoidoscope was attempted past the mass without success. A flat plate of the abdomen unfortunately was not taken, nor a barium enema study made.

On January 16, 1953 after the bowel had been sterilized for twenty-four hours previously with neomycin, this tumor mass was removed under Pentothal anesthesia. The pedicle arose from the right lateral rectal wall and was about one-half inch in diameter and very short, so that it was almost flat against the bowel wall. The mucosa of the bowel did not extend onto the stem but surrounded it in a manner much like fine sand would surround a mushroom stem. It was difficult to extrude the mass and only after considerable traction was the entire mass presented. The pedicle was clamped flush with the mucosal surface and ligated with chromic catgut, and allowed to retract back into the pelvis.

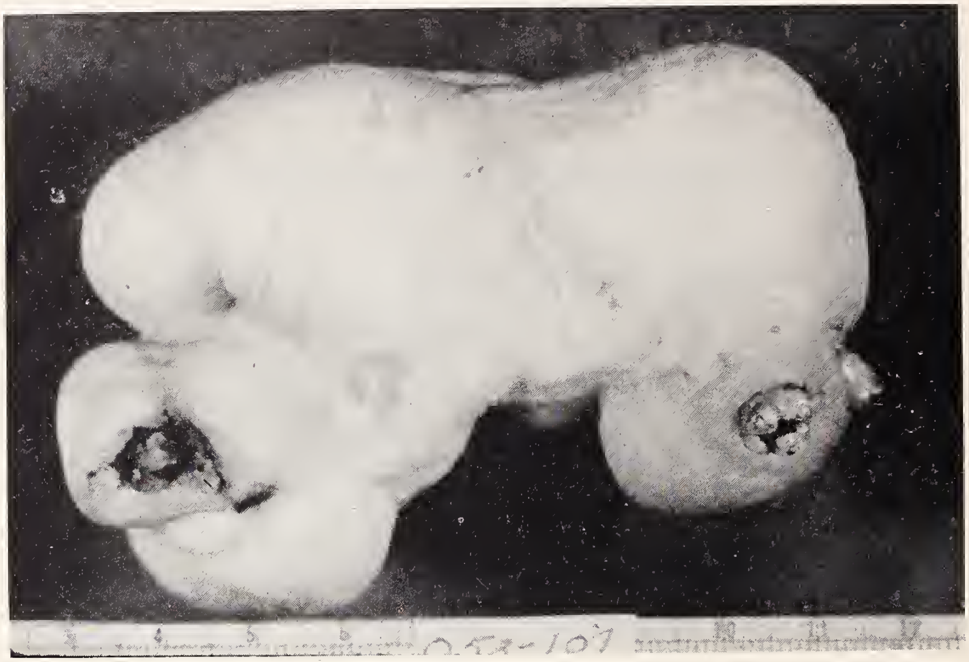

Fig. 1.

The specimen removed consisted of an irregular shaped mass of tissue measuring approximately 9 x 5 x 4.5 cm. in size. It was covered externally by skin from which a number of long fine hairs were growing. At one end there was a protuberant mass from which protruded two molar teeth. Opposite this was a small verrucous area measuring 1 cm. in diameter. On cut section the central portion appeared to be composed primarily of fat and connective tissue. Microscopic examination of sections established this as a true teratoma. The outer surface was covered by tissue having all of the characteristics of skin with hair follicles and sebaceous glands present. A cyst found in the deeper tissue was lined by a pseudostratified columnar ciliated epithelium and small islands of cartilage laid adjacent to this similar to what one would see in large bronchi. The intervening tissue showed fat, callogenous connective tissue and apocrine glands of various types. No evidence of malignancy was found in any of the sections.

Five days after the surgical procedure a sigmoidoscope was passed up to 20 cm. and no sign of the pedicle area was found. A month after surgery a sigmoidoscope was passed 22 cm. and no evidence of scar of previously removed tumor was found.

A barium enema was given six months after surgery, and no evidence of former tumor site or any other abnormality was found. Postoperative course was uneventful and the patient has been well and symptom free since.

(Bibliography on Page 131)

DIAGNOSTIC ENIGMAS OF DISEASES OF BONE

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In most fields of oncology, all eyes turn to the pathologist for the last word—*his histologic diagnosis.* For the recognition of diseases of bone the clinical and roentgenologic consultants are frequently of equal or greater importance. Though there are many clear-cut blacks and whites in the diagnosis of osseous lesions, there exist, on the other hand, unusually large grey areas of frustration. Examples of mutilation resulting from "overdiagnosing" and the fatalities from "underdiagnosing" bony diseases are legend in the annals of pathology. There is probably no group of diseases in the body where the pathologist's ignorance has done relatively more harm. Paucity of experience and rather recent organization of knowledge in the field are partly to blame. It behooves the responsible clinician to know some of the more important weaknesses in the histologic diagnosis of diseases of bone so that he may not be so easily led astray by his most trusted microscopist.

The day is long past when the cellularity of the benign giant cell tumor will lead to an unnecessary amputation. Furthermore, pathologists have learned that the presence of multinucleated giant cells does not establish the diagnosis of giant cell tumor any more than fever is a disease. The combination of fibrous connective tissue and giant cells occurs abundantly not only in giant cell tumor but also in fibrous cortical defects, unicameral bone cysts, osteitis fibrosa cystica, benign chondroblastoma, polyostotic fibrous dysplasia, monostotic fibrous dysplasia, renal osteitis fibrosa, xanthomas and other diseases. The most important errors of diagnosis in this group of diseases of bone include: (1) failing to locate small sites of malignancy in an otherwise benign giant cell tumor; (2) completely ruling out osteitis fibrosa cystica (hyperparathyroidism) without serum calcium, phosphorus and phosphatase analyses; (3) making a prognosis as to the possible future recurrence and malignant changes from a histologically benign giant cell tumor; (4) not recognizing the potentially dangerous benign giant cell tumor because of much hemorrhage and degenerative changes in the lesion and (5) misjudging in either direction a giant cell tumor of bone with histology in the borderline of malignancy.

Many limbs have been unnecessarily amputated because of the presence of benign, cellular, growing osteoid tissue. A list of the various benign conditions misdiagnosed as osteosarcoma certainly would include, among others: myositis ossificans, osteoid osteoma, exuberant callus of fractured or otherwise traumatized bone, ossifying displaced periosteum, Caffey's disease, Garré's osteitis, syphilitic osteitis, polyostotic fibrous dysplasia, monostotic fibrous dysplasia, ossifying bursitis and Charcot's joint. One fourth of the written diagnoses of pathologists at the 1953 annual tissue seminar of the American Society of Clinical Pathologists listed the slide of myositis ossificans as osteosarcoma. On the other hand, the pathologist may mistake osteosarcoma for any one of these benign lesions listed above. As in other neoplasms, borderlines between benignancy and malignancy in a small percentage of osteoblastic lesions are impossible to determine. This is especially true in the parosteal bone tumors.

An unusually difficult histologic diagnostic problem exists in the potentially dangerous enchondromas of long bones. It is entirely possible today that competent pathologists have a tendency to "overdiagnose" these enchondromas, while the inexperienced may fail completely to appreciate the importance of the subtle changes of edematous degeneration of the matrix, multiple cells per lacuna, and plumpness of nuclei found in the low-

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From the Department of Pathology, St. Luke's Hospital, Duluth, Minnesota.

This is the twenty-fourth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

grade malignancies of cartilage. This paradox is emphasized by the necessity for immediate radical surgery of chondrosarcoma, no matter what its histologic grade of malignancy. Most pathologists must depend on utilizing the experience of others for the proper evaluation of many of these rare diseases of the skeleton. The fact that only isolated focal areas of malignancy may be present does not help solve this diagnostic problem.

Neoplasms arising from cartilage cells in other bones for the same reasons may be difficult to classify. In a series of forty patients treated in a large Minnesota medical center for what was originally considered histologically benign chondromata, nine died of their malignancy and eight had recurrences. The destructive and cellular benign chondroblastoma may lead both the roentgenologist and the pathologist to advise radical surgery. At the seminar cited above, one third of the pathologists voting listed this nonmetastasizing lesion as malignant. There may be an abundance of immature cartilage in the callus at the site of a march fracture. The cartilage in an enchondroma of the phalanx may have startlingly large nuclei and be unusually cellular with evidence of bone destruction and yet be benign. These and other difficulties may becloud the histologic interpretation of neoplasms of cartilage.

There is a long series of miscellaneous booby traps set in the path of the histologic recognition of lesions of the skeleton. Some of the more important of these include: (1) mistaking a cellular fibrocytic reaction at the site of an osteomyelitis for fibrosarcoma; (2) being deceived by the large closely compact cells of a bone-destroying benign xanthoma; (3) advising an unnecessary radical cr disfiguring operation for the nonneoplastic disease, polyostotic fibrous dysplasia; (4) considering the frequently destructive and large aneurysmal bone cyst malignant and (5) failing to recognize the secondary nature of a bony lesion metastatic from occult primary malignancy. A patient with a cavernous hemangioma of the tibia refused to have his leg amputated for "cancer" in one hospital. In a second institution, the condition was diagnosed by biopsy and x-ray examination as Paget's disease. Here the man may well have lost his leg as the result of therapeutic procrastination. In a third hospital, a diagnosis of cavernous hemangioma was made and he was given radiation therapy for the benign but progressing vascular disease. After ten years, he appears to have recovered.

Conclusion

Four basic rules should be listed so that the responsible clinician will get the most out of his pathologist consultant for the patient with a bone disease:

1. Do not overestimate the value of the histologic diagnosis.
2. If there is any question of diagnosis, the pathologist will welcome "outside" consultations. The best and the most experienced minds of pathologists in the country are available without charge.
3. Present the pathologist with all of the clinical and roentgenologic evidence; he may be lost without it.
4. The clinician should know the principal areas of uncertainty in this difficult field of microscopic pathology.

The pathologist is responsible for his own diagnosis. He should not make many serious mistakes before he recognizes most of his personal limitations and the range of accuracy of the methods of his specialty on his battle line of practical experience.

MODERN MEDICINE AND MORBIDITY

The data which witness modern medicine's triumphs, the statistics that reflect the declining death rates and the increase in life expectancy (at birth), when critically examined add up to the summation that modern medicine has in a large measure converted mortality into morbidity. Those whose lives it has helped to save in many instances face not an extension of existence in well-being and in health, but merely a prolongation of endurance. Those who by the skills of modern medical science have been saved from death all too often have been saved only to longer endure the burdens of a variety of illnesses, and to die at a later age. In the statistical tabulations such individuals appear to have gained years of welcome life, but these may be, and too often prove to be, years of painful travail, years of dependency, unproductive years which in the last analysis are social and individual liabilities rather than assets.—Iago Galdston, M.D., "The Meaning of Social Medicine," Harvard University Press, 1954.

Clinical-Pathological Conference

CASE PRESENTATION

ARTHUR C. AUFDERHEIDE, M.D.

Clinical History

The patient, a sixty-seven-year-old white man, retired woodcutter, was first admitted to St. Mary's Hospital May 7, 1954. Complaints were nocturia, difficulty starting stream and gross hematuria for one and one half months. Hematuria had been severe. He had lost weight from 250 pounds to 180 pounds in the preceding two years, ascribed to voluntary dieting resulting from accompanying dyspnea during that time. Anorexia had been present for the past several months but no abdominal pain. Past history, family history and system review was negative. Physical examination showed a few small hemorrhages in the oral mucous membrane; otherwise the eyes, ears, nose and throat were not abnormal. Several bean-sized hard nodules were palpable in the supraclavicular areas in each side. Except for occasional premature contractions the heart was not abnormal. The liver was palpable 4 cm. below the rib margin in the right midclavicular line, but was not tender. The extremities and reflexes were not abnormal. Rectal examination showed an enlarged, hard, nodular prostate gland. An attempt was made to pass a catheter but failed.

Laboratory findings are shown in Table I.

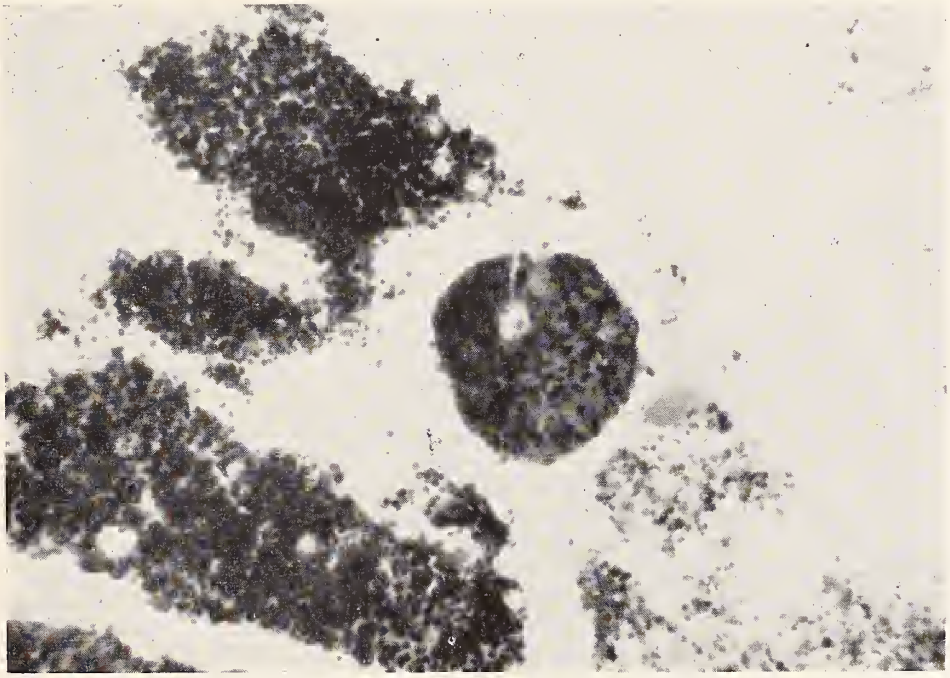

Fig. 1. Histologic section of bone marrow particles (from the bone marrow aspiration specimen performed antemortem) showing metastatic carcinoma. Hematoxylin and eosin, ×425.

On the day following admission 2500 cc. of blood were given the patient. The same day a suprapubic catheter was put in place; bloody urine was draining freely when the patient left the operating room. A radiograph including the kidneys, ureters and the bladder showed arthritis, but no evidence of metastatic malignancy. On May 10, another 2500 cc. of blood were given, but the pubic drain clogged and a urethral catheter was passed successfully which drained more bloody urine. On May 11, 6300 cc. of blood were given and a bone marrow aspiration performed. Serious bleeding occurred for at least twenty-four hours from the sternal puncture wound. The bone marrow was reported as showing erythroid and myeloid hyperplasia in the bone marrow and thrombocytopenia in the peripheral blood. Megakaryocytes were numerous and not abnormal morphologically. Histologic sections of marrow particles showed metastatic carcinoma (Fig. 1). In the afternoon, on May 11, the patient again passed into severe shock. Noradrenalin, digitalis, and intravenous aminophyllin were given. The skin appeared jaundiced. ACTH was started.

Additional history at this time revealed a past history of chronic alcoholism for many years. The patient continued to bleed severely until about June 2, when bleeding lessened markedly. By June 10, only a tinge of

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From the files of the Department of Pathology, St. Mary's Hospital, Duluth, Minnesota.

Edited by Donald Gleason, M.D., under the auspices of the Minnesota Society of Clinical Pathologists.

blood was seen in the suprapubic discharge. On that date, auricular fibrillation at a rate of about 100/min. was noted. The patient suffered dyspnea and had demonstrable leg edema. Râles were heard in the lower chest posteriorly. Clinically, he was much better and much more alert. On June 11, rectal examination revealed the prostate to be somewhat smaller, probably as a result of the stilbestrol therapy which had been administered since admission, but it was felt that the decrease in size was mild. The patient expired on June 14 at 4 a.m. Earlier that evening, even though he had been improving, he awoke at 11:20 p.m., very confused and soiled himself with a large loose stool. He complained of pain in the throat and chest pain. He was very restless and passed several more loose stools. He became irrational, and respirations were rapid and labored. He obtained slight relief from opiates and expired quietly.

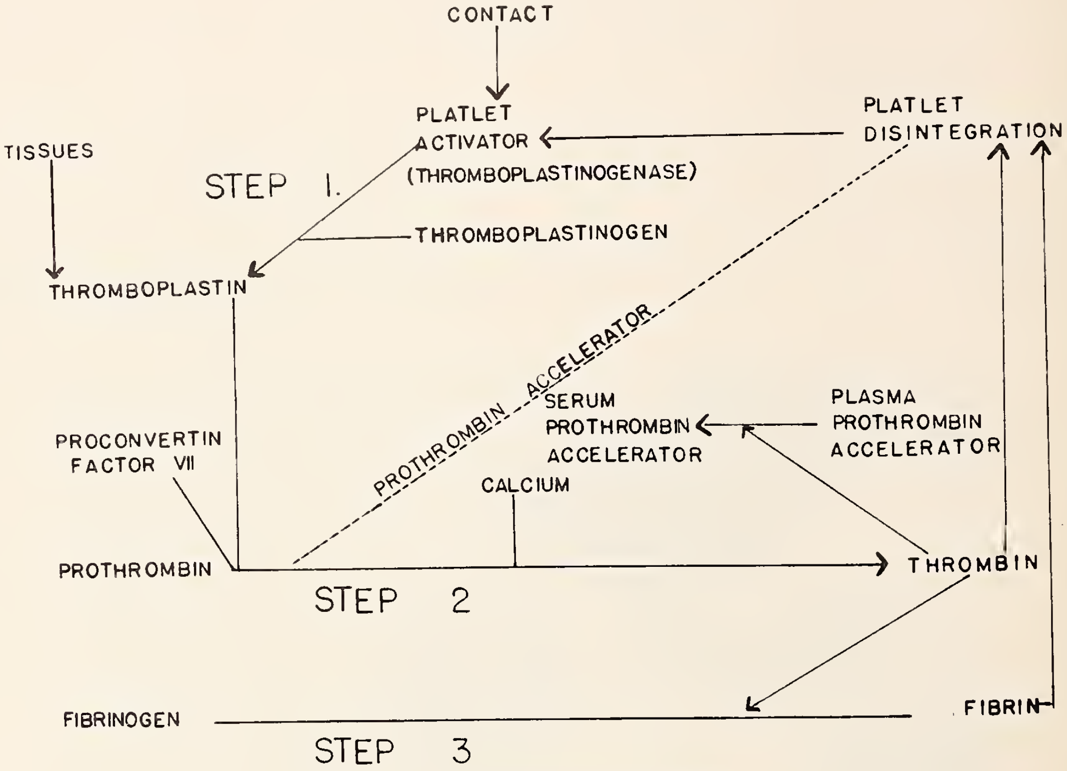

Chart 1. (Adapted after Stefanini[5])

Discussion

Dr. Robert Goldish (Duluth): It is worthwhile to consider briefly blood coagulation in general and how it relates to the present case. Pertinent methods of "screening" hemorrhagic disorders include a careful history and physical examination, detailed study of the family history, bleeding and clotting time, clot retraction, platelet count, prothrombin time, and capillary fragility test. Caution and perspective must be exercised in interpreting these laboratory tests. The laboratory technique must be standardized and the tests performed with care and controls, since seemingly minor variations in technique can cause grossly misleading variations in results. For instance, just to mention one or two variations which may be quite common, a falsely prolonged bleeding time may result from too deep a puncture, and—in some laboratories, at least—the coagulation time done by the Lee-White method may normally run as high as twenty minutes.

Quick's prothrombin consumption test is actually a measure of available thromboplastin, comparing the prothrombin time of plasma and of serum to determine how much prothrombin was consumed in clotting. Normally, about 80 per cent or more of the originally available prothrombin will be consumed in clotting, while in hemophilia or thrombocytopenic purpura (where thromboplastin production is decreased) as much as 90 per cent may *remain* in the serum after clotting. Many other more specialized tests may be used, and at times it becomes necessary to study blood samples from the patient's relatives.

In 1905, Morawitz established the classical theory of blood coagulation which recent concepts have not contradicted but merely expanded. Morawitz postulated that prothrombin was converted to thrombin through interaction with calcium and thromboplastin; thrombin then caused fibrinogen to form fibrin. Much of the current confusion is probably due to the fact that different investigators independently discovered similar or identical phenomena simultaneously, resulting in a turmoil of nomenclature. Nevertheless, most investigators[5] agree on the following four points: (1) thromboplastin activation is a separate, preliminary phase in thrombin formation *in vivo;* (2) factors in addition to thromboplastin and calcium are involved in the conversion of prothrombin to thrombin; (3) serum contains one or more agents accelerating prothrombin to thrombin

TABLE I.

DATE	PROTHR. TIME	BLEEDING and CLOTTING TIME	HGB	PLATELETS	BLOOD TRANSF'S.	OTHER LABORATORY DATA	TREATMENT
5-7			6.2			BUN 26, Creatinine 2 mgm % Urine 1.022, 4+ Alb, 4+ RBC	Stilbesterol, Vit K Cont'd to death
5-8			6.0-8.3		2500 cc.	Acid Phosphatase 1.8 KAU. Alk. Phosphatase 4.0 KAU.	Penicillin begun and continued thru 5-31
5-9			7.8				
5-10	0 %	B.T. - 7'36" C.T. - 5' 0"	8.6	18,000	2500 cc.		Digitalized Levophed
5-11			11.4-9.3	13,200	6000 cc.	Bilirubin 3.7 mgm% direct 3.4 mgm% indirect	ACTH Levophed
5-12	0 %	C.T. - 5' 5"	9.5-7.5			Fibrinogen - 0.266 Gm%	ACTH 40 mgm q.d.
5-13	46%*	C.T. -14'50"	7.5			*P.T.: No clot P.T.: +thrombin - no clot P.T.: +normal plasma - clot	
5-14	65%*		5.8		1000 cc.	Coombs - negative (direct) Cephalin flocculation - 3+	
5-15			9.5		1000 cc.	Thymol turbidity - 0.6 units BUN - 78 Protein - 5.5 gm% total 3.7 albumin 1.8 globulin	
5-16	41%*	C.T. -20'	7.6	52,800		RBC fragility - normal Protamine Titration - 0.4 mg. protamine in 1 hr. (normal)	
5-19	0 %		7.8			Bilirubin 1.2 mg.% Direct 1.2 mg.% Indirect Fibrinolysin - Positive**	
5-20						Feces Urobilinogen - 350 Ehrlich units/100 gm	
5-21			6.9		500 cc.		
5-22						CO_2 - 24 meq/L; BUN 38	
5-23			8.4			Serum Potassium - 3.7 meq/L	
5-25						Feces urobilinogen - 260 Ehrlich units/100 gm	
5-26		B.T. - 1' C.T. - 8'23"	8.4	64,500		Reticulocytes 1.8%	
5-27	39%					Bilirubin 0.6 mg.% Direct 1.1 mg.% Indirect	
5-28						Fibrinolysin - 90% of clot disappears in 1 hr.	
5-29						Cephalin flocculation - 1+	
5-30			8.4		500 cc.		
5-31					500 cc.		
6-4			8.9				ACTH - 30mg. qd Thiomerin 2 cc. qd
6-8	80%	B.T. - 1'	8.0	84,700		Bromsulfalein - 16% retention Fibrinolysin - 50% clot Dissolution in 1 hour	ACTH - 10 mg. qd
6-11							TACE - 12 mg. qid

* One stage prothrombin method of Quick. Endpoint recorded on May 13, 14, and 16 was appearance of a few fine greyish strands which disappeared in a few seconds. They might better be recorded as 0%. Addition of thrombin produced no better clot, but addition of one drop of normal serum produced a clot in 12 seconds.

** Test performed by placing freshly clotted patient's blood in 37° incubator for 1 hour; disappearance of clot reported as positive test.

conversion; (4) the formation of thrombin proceeds as an autocatalytic, progressively accelerating process.

The first phase of blood coagulation concerns the production of thromboplastin (Chart 1). This substance may be formed either by release of formed thromboplastin from injured tissues or by interaction of plasma constituents with products contributed by lysed platelets. Platelet lysis occurs upon contact with certain surfaces such as disrupted tissue, air or glass. The material released from lysed platelets (platelet activator; thromboplastinogenase) reacts with a substance normally present in serum (thromboplastinogen: antihemophilic globulin; AHG) to form thromboplastin. Thus, platelet deficiency would result in decreased thromboplastin formation. It has been noted recently[6] that AHG is not the only substance necessary to combine with material from lysed platelets to form thromboplastin; at least one other substance (plasma thromboplastin component; PTC)[4] and perhaps two (plasma thromboplastin antecedent; PTA) have been identified.

Phase 2 of blood coagulation is concerned with conversion of prothrombin to thrombin. Thromboplastin and calcium will combine, perhaps along with other factors, with prothrombin to produce a small amount of thrombin, insufficient to activate fibrinogen. An accelerating factor present in the plasma as an inert substance (plasma AC-globulin, proaccelerin, factor V) rapidly becomes active as a serum prothrombin accelerator in the presence of thrombin.[3] Thus, the initially formed small amounts of thrombin activate the accelerator substance which, in turn, "speeds up" the conversion of prothrombin to more thrombin eventually to produce enough of the latter for fibrinogen activation. A second, less well-defined prothrombin accelerator has been described and has been named stable factor.[2,3]

Phase 3 is universally conceded to be a simple enzymatic process in which fibrinogen is converted into fibrin by thrombin, aided perhaps by other accelerators, such as those of platelet origin.

Further details of interest include the possibility that platelet lysis may be enhanced by thrombin, fibrin, or both; the release of a locally acting vasoconstricting substance, (serotonin), from lysed platelets and the release of other prothrombin accelerating factors from lysed platelets. Anticoagulating substances are also normally present; antithromboplastin, antithrombin, and even fibrin itself all act as buffers to the process of blood coagulation. It should be pointed out that Vitamin K has no effect on prothrombin other than to enable the liver to form prothrombin.

In the application of these concepts to the case at hand, several features are important. Shortly after admission, thrombocytopenia and prolonged prothrombin time were noted. Although there are suggestions in the literature that the two may be associated, I do not understand such an association since possible thromboplastin deficiency *in vivo,* which might be expected from thrombocytopenia, ought not to affect the simple one-stage prothrombin time determination, in which thromboplastin is deliberately added in excess.

Stefanini[5] has reported a case, in which this association was noted (secondary thrombocytopenic purpura) and in which he showed not only thrombocytopenia, but also decreased prothrombin accelerator factors, explaining the delayed prothrombin time on the coincident occurrence of the accelerator deficiency.

In the present case, the addition of a drop of normal plasma to the patient's plasma resulted in a normal prothrombin time, suggesting that no circulating anticoagulant was present in sufficient quantities to prevent prothrombin activity. The protamine titration, a test to determine the presence of a circulating heparin-like compound, was also normal. Yet when the thrombin was added to the patient's plasma no clot formed, even with the addition of thromboplastin and calcium (not normally needed to convert fibrinogen to fibrin in the presence of excess calcium). I do not understand this finding in the face of a normal coagulation time.

The presence of fibrinolysin may be significant. This is a substance which can cause dissolution of a clot. The fibrinolysin system has been incompletely described. The following scheme seems to be established thus far.[5] Profibrinolysin normally present in the plasma may be activated to fibrinolysin by an enzyme (fibrinolysokinase). This system is normally held in check by an antifibrinolysokinase and an antifibrinolysin. It is probable that fibrinolysin was present in excess quantity in the case we are discussing and that it may have been responsible, not only for the clinical hemorrhage, but also for the failure of a clot to form when thrombin was added to the patient's plasma *in vitro*. Carcinoma of the prostate has been reported to produce excess fibrinolysin. Furthermore, Wintrobe[7] and others report the suggestion of activation of the fibrinolysin system by the stress phenomenon, and that antifibrinolysin may be stimulated by ACTH, a drug the patient was receiving. Fibrinolysins have also been described in cases involving placental separation, transfusion reactions, and in bleeding diatheses associated with leukemias and cancer of various organs.

In summary, I think this patient had a prostatic carcinoma with widespread metastases including the bone marrow, with resulting secondary thrombocytopenia and excess circulating fibrinolysin. The situation was probably controlled, at least partially, by steroid therapy which the patient received.

Doctor Goldish's Diagnoses

Hemorrhagic diathesis, due to:

Thrombocytopenia, secondary, and excess circulating antifibrinolysin, due to:

Carcinoma of the prostate with widespread metastases.

Anatomic Diagnoses

Adenocarcinoma of the prostate with multiple metastases.

Widespread metastases to bone marrow.

Dr. A. C. Aufderheide (St. Mary's Hospital): At autopsy the heart weighed 500 grams, but except for mild coronary arteriosclerosis showed no further ab-

(Continued on Page 124)

President's Letter

THE CORNERSTONE

The county medical society is the basic cornerstone of medical organization. Just as a great and impressive building must have a firm foundation, replete with strong cornerstones, so the state and national medical organizations must have a firm foundation in strong county medical societies.

It is very difficult for medicine's critics to realize that the state and national groups have no power over the county medical society, and can dictate to it in *no* way. Medical organization is a confederacy, not an autocracy. Those who picture the AMA as a gigantic, all-powerful trust, are laboring under delusions; the state and national groups do not enter a county society problem unless specifically requested to give help.

The extensive value of a county medical society cannot be overemphasized. It is the entrance port for membership in the state and national organizations. A member of the county medical society and the state medical association does *not* have to be a member of the American Medical Association.

To my mind, one of the most important projects of the local society is the proper preparation and reception of new members. It is in the county medical society that the new member becomes aware of his many obligations, as well as his privileges—these are necessary things if the new member is to fulfill the ideals of what the practicing physician should be. In practical terms, the county society is the character-building organization, to the end that the nation and the profession are well served.

The job of informing long-time, as well as new members, is that of the county officers. They must keep themselves aware of the larger problems of state and national medical organizations. They can be aided by the conscientious efforts of state officers, councilors and AMA delegates to report the decisions and discussions carried on at a state and national level. The membership in general can be kept well informed by discussions at annual meetings with officers on problems of membership, programs, mediation committees, and the many varied activities of medical organization. One of the most valuable of these meetings is our recent County Officers' meeting, held to acquaint and re-acquaint county officers with the sometimes intricate, but always interesting, procedures and policies of the state and county organizations.

Another important and recurrent problem is how best to stimulate the "old" member to assume more active interest in his local society. I fear that there are too many who are simply "going along for the ride." How immeasurably more pleasant and satisfactory would be the work of the officers of the state and county organization if every one of our members took the active interest he or she should.

It would be hard to evaluate the worth of such interest, both to the public and the profession. Truly, the county medical society is, and rightly should be, a strong and indomitable cornerstone of medical organization. It is upon this solid foundation that the state and national groups depend for a secure base, for if it crumbles, the entire structure could be seriously impaired.

Arnold O. Swenson

President, Minnesota State Medical Association

Editorial

John F. Briggs, M.D.
Henry G. Moehring, M.D.
Arthur H. Wells, M.D.

THE DOCTOR AS A WITNESS IN INDUSTRIAL COURTS

The doctor is a highly important witness when testifying as an expert in his chosen profession before a referee of the Industrial Commission in a workmen's compensation case. He is called because of his knowledge gained through study and experience in a particular field of medicine. His testimony is not above being challenged by other experts in the same field, nor is it beyond being questioned or disbelieved by the average layman.

This leads to one thing: the expert witness, regardless of whatever field of endeavor in which he is qualified, must be prepared for the rôle he is to take, and his testimony must be straightforward and unbiased.

If the doctor has treated the patient, keeping complete medical records and having them while testifying is of the utmost importance. Without records, how can a medical witness testify as to the first day the patient came to him as a result of injury or illness? Unless the doctor has a superhuman memory, how can he recall what the patient's complaints were, what examination was made and what treatment given? History is essential as a part of the doctor's records, not only from the standpoint of care and treatment, but also for future reference, should litigation or a claim arise because of a trauma or occupational disease the patient may have suffered, which resulted in his need for medical attention. These records should cover not only the first visit, but subsequent visits, right down to the day of discharge. The first entry in the record in most cases, of necessity, will be quite detailed. Entries covering subsequent visits may be shorter, but not so sketchy that they do not contain the patient's symptoms, complaints, prognosis or treatment. Mere dates of visits are not enlightening.

A medical witness, as well as any other expert, must testify to the facts as he understands them to be. His testimony to be of value must be unbiased and his opinions based on sound medical principles backed by his knowledge and experience. To color one's testimony in favor of one side or the other in a dispute is unforgivable. The trier of facts soon becomes aware of the witness who does so and consequently his testimony is discounted to that degree. His efforts to become an advocate for one of the parties does not reflect credit upon himself or his profession, nor does it redound to the benefit of the recipient of his efforts. The medical witness who expresses his honest opinion, regardless of whether it is favorable to the party who calls him or to the opposing side, is the most valued and most respected in the eyes of the trier of fact. Medicine not being an exact science, very often it is impossible to make an exact diagnosis, either because of lack of experience on the part of the doctor in a particular field, or because the problem is so controversial that several diagnoses may be made. In such a situation, the expert witness should make a straightforward statement that he does not know, or give a qualified opinion, based on sound medical principles, of his knowledge as to what the diagnosis may be. If he pursues this course, the trier of facts will have much greater respect for him and give greater credence to his testimony, than if he engages in specious theorizing.

Norbert Willwerscheid
Chief, Division of Workman's Compensation

THE PHYSICIAN AND HIS BUSINESS INSURANCE

The medical men, by the very nature of his profession, has made himself vulnerable to both loss and abuse. If he is successful financially, there are many ways by which his future can be impaired. His possessions may be damaged, destroyed or stolen. His financial future may be ruined through claims brought against him charging malpractice.

By the very nature of a medical man's training, he is keenly aware of the value of professional or expert advice. If the physician's problems, insurance-wise, were laid fairly before a competently qualified insurance man, the problem of securing and preserving his economic status could be completely and adequately assured.

There are many exposures facing the physician today. In his business alone, great strides have been made to satisfy these needs. New and complete coverages have been devised to cover the actions of the doctor, whether he be acting professionally or in a personal manner. The fine equipment which graces his office or clinic also must be insured properly to hedge against a disastrous loss. The following list of coverages may or may not apply to each individual physician. They should, however, be given very careful consideration.

On building and contents coverages are available for:

Fire and Extended Coverage
Vandalism
Rent or Rental Value
Improvement or Betterment
Radium
Physician's and Surgeon's Instruments
Signs
Glass
Boiler and Machinery
Extra Expense or
Business Interruption

Under the professional activities, we find:

Premises Liability
Malpractice
Comprehensive Personal Liability
Office Burglary
Fidelity Bonds
Automobile Insurance
Non-Ownership Liability
Workmen's Compensation

Since both financial and professional ruin contantly face the physician today, he needs the best of help in choosing the specialized forms which are available for his protection. Again, we should repeat that the best insurance agent costs no more and it is of great importance to consult an agent of good reputation, one who represents insurance companies today that will have financial strength to take care of claims that will come in the tomorrow.

Ernest Collingham

SIGMOIDOSCOPIC EXAMINATIONS

Lord Dawson of Penn visited the United States in 1923, and, in one of his addresses, made the statement that in doing physical examinations, "if one did not put his finger in the rectum he would be certain to get his foot in it." That statement marked the beginning of the popularity of the digital rectal examination as a part of every physical examination. No longer was the performance of a rectal examination the sole province of the consultant. Pathology of the rectum and lower colon was brought into the reach of the general practitioner as well as the specialist, and better diagnosis and treatment resulted. Today, the public, as well as the medical profession, is interested in earlier diagnosis of serious lesions and in the early detection of precancerous growths. No longer is the digital rectal examination considered an adequate investigation of the rectum and lower sigmoid colon, since many lesions, particularly the precancerous ones, cannot be felt. Only by means of a well-performed sigmoidoscopic examination can early lesions of this part of the bowel be visualized. Reports from all cancer detection centers are uniformly impressive in pointing out the importance of sigmoidoscopy. Since approximately 10 per cent of all adults harbor precancerous polyps in the rectum and lower colon, the need for early detection cannot be overemphasized.

The time has come when we can state with didactic assurance that no general physical examination can be considered complete without the visualization of the interior of the rectum and lower sigmoid colon. The time has arrived also when accredited hospitals should no longer condone the performance of anorectal surgical operations on patients who have not had sigmoidoscopy.

Medical schools and teaching hospitals must assume the responsibility for the teaching of sigmoidoscopy to students, interns and residents. Physicians in practice should acquaint themselves with the technical aspects of sigmoidoscopy and should make this important examination available to their patients. When sigmoidoscopy is performed as a routine procedure in all complete physical examinations, the cancer detection program will have received one of its greatest lifts.

Wm. C. Bernstein, M.D.

"NEW OCCASIONS TEACH NEW DUTIES"

With the advancement of medical practice from the horse-and-buggy to the jet-plane age, the activities and setting for practice have been affected for nurses probably more than for any other group. Shall we look at some of the trends in nursing in which this advancement in medicine has played a part?

1. *Extension of the Physician's Service.*—

Whereas, formerly the physician looked to the nurse for provision of a simple service devoted primarily to physical care of the sick, the modern physician tends to rely on nursing for a much more complex type of nursing care—physical, mental, emotional, and spiritual. It should be borne in mind that he expects care of all ages with increasing emphasis upon middle, later life and long-term illness and not only care of the sick, but also of the convalescent and the well. And, whereas the nurse of earlier years was found usually in hospitals or with private patients, the nurse of today appears wherever there are human beings: in institutions of various kinds; in homes; in public health agencies such as visiting nurse services, clinics for the aged and group practice, diagnostic, child, and other types of clinics; in schools; college and university health services; in industry; in doctors' offices (35,000 in 1951); in admission and related services; in transportation; in the military; in departments, schools and colleges of nursing. Limitless, indeed, in type of care, of patient, or of locale are the opportunities of the nurse to extend the service of the physician.

Co-ordination of Service.—Just as in medical practice the physician has tended to share his increasing responsibilities with many types of personnel, among them the roentgenologist, the nutritionist, the social worker, the clinical psychologist, the mental hygienist and the nurse—so also has the nurse found it necessary to share her earlier functions with others: the practical nurse, nurse's aide, or other appropriate personnel. In 1952, for example, there were 70,000 more nonprofessional than professional nursing personnel in hospitals registered with the American Medical Association. This sharing in nursing, as in medicine, has resulted in the development of a team of workers—in the one, the comparatively small nursing team; in the other, the more comprehensive health team. Requisite to effective functioning of either group, as in baseball or football, is co-ordination of the efforts of all members of the team to the end that maximum use is made of every member and optimum patient care results.

Improvement of the Quality of Service.—Among the factors contributing to improvement of nursing is recognition of the unique contribution of the individual and of good human relationships. In the field of nursing as in other fields, where studies have been made, a correlation has been found to exist between recognition of the worker and the quality of the worker's service. In the opinion of many, the physician, by doing increasing co-planning with other personnel, is giving them status, and the end result is better patient care. Studies of human relationships in hospitals being made currently by Dr. Leo Simmons of Yale University throw into clear focus the increasing attention being given to this whole area and give earnest of improvement of service through improved interrelationships.

To safeguard the patient's care in today's complex medical practice, to best extend the physician's service and co-ordinate all service for the patient as well as to improve the quality of nursing—all of these have portentous implications in the preparation of the professional nurse. Entrants to professional schools of nursing must be of professional calibre. They must have general as well as professional education. They must know not only skills and techniques, but equally important they must have knowledge and understanding of what they are doing and why. In short, the new occasions and duties in nursing demand that the nurse have education for a profession with increasing status and recognition, one whose members are equipped for responsible partnership on the health and nursing teams, who are skilled in developing and maintaining effective interpersonal and group relationships and who are prepared for useful personal, family, and community living.

Katharine J. Densford

ACCIDENTS, THE KILLER OF CHILDREN

Suffocation

In this issue, your attention is again directed to another in the series of articles on Accidents, the Killer of Children in Minnesota. The author highlights icebox deaths—let each doctor help to maintain Minnesota's perfect record; hangings—we have several every year; and suffocation or crib deaths—let us be a little more careful in applying this diagnosis.

Dr. Berglund's comments are well worth your reading time.

Tague C. Chisholm, M.D.

The Dean's Page

DISTINGUISHED SCIENTISTS JOIN FACULTY

Since last July 1, three distinguished scientists have joined the Medical School faculty as professors in their respective department. Since each of them fills an important post, members of the medical profession in the state will be interested in learning something of their backgrounds.

On July 1, Dr. Arnold Lazarow succeeded Dr. E. A. Boyden, as Professor and Head of the Department of Anatomy. Dr. Lazarow had been Associate Professor of Anatomy at Western Reserve University School of Medicine since 1948. A graduate of the University of Chicago, where he obtained both the M.D. and the Ph.D. in Anatomy in 1941, he had been associated with Western Reserve's Department of Anatomy in various capacities since 1943. There he was recognized as an outstanding teacher and investigator. Much of his experimental work has concerned diabetes mellitus which he has studied extensively from histological, histochemical, and biochemical standpoints.

A member of Phi Beta Kappa, Alpha Omega Alpha, and Sigma Xi, he also holds membership in the American Association for the Advancement of Science, the American Association of Anatomists, the Society for Experimental Biology and Medicine, the American Diabetes Association, and the Society of General Physiologists. He is, indeed, a worthy successor to a most distinguished scholar.

Also on July 1, Dr. Ivan D. Frantz, Jr., became the George S. Clark Research Professor of Medicine. Dr. Frantz, a 1941 graduate of Harvard Medical School, prior to coming here was Associate in Medicine at Harvard Medical School, Tutor in the Biochemical Sciences at Harvard University, Assistant Physician at Massachusetts General Hospital, and Consultant in Biophysics at Massachusetts Institute of Technology. He was associated with Harvard Medical School from 1946 to 1954. His research work has dealt primarily with various phases of protein and cholesterol metabolism.

Dr. Frantz is a member of the American Society for Clinical Investigation, the American Association for Cancer Research, and the American Society of Biological Chemists.

On December 16 a distinguished alumnus of the Medical School, Dr. John A. Anderson, returned to his alma mater as Professor and Head of the Department of Pediatrics. Dr. Anderson, a native of Sioux Falls, South Dakota, graduated in 1933 and served his internship at the University of Minnesota Hospitals. After further training and research work in pediatrics in Denver, Colorado, he returned to Minnesota in 1937 where he was a member of the faculty of the Department of Pediatrics until 1943. From 1943 to 1949 he organized and served as head of the Pediatrics Department at the University of Utah Medical School, and since 1949 he has held a similar position at Stanford University Medical School.

Dr. Anderson succeeds as Head of the Department of Pediatrics Dr. Irvine McQuarrie, who requested that he be relieved of administrative duties in order to devote his remaining five years on the faculty to teaching research and writing. Dr. McQuarrie was recently honored for his twenty-five years of outstanding service to the University.

The Medical School is indeed fortunate to be able to add men of the caliber of Doctors Lazarow, Frantz, and Anderson to its faculty. I hope that members of the Minnesota State Medical Association will have an early opportunity to meet each of them personally.

Harold S. Diehl, M.D.
Dean of Medical Science
University of Minnesota

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

COMPULSORY HEALTH INSURANCE ENACTED FOR SWEDEN

A compulsory health insurance plan went into effect January 1 in Sweden. It will be jointly financed by the insured persons, employers, and the government. Information on the plan is explained in a recent issue of a news letter from the Research Council for Economic Security.

The letter states these figures on shouldering the costs of the plan: "Insured persons will pay 44 per cent of the cost; employers, 29 per cent; and the state, 27 per cent. The state's portion will be passed on to tax bills and thus absorbed by the higher-income earning groups. The total cost of the first year's operation of the insurance plan is estimated at $144 million."

Who Pays?

According to the article in the news letter, state health insurance premiums must be paid by every person above the age of 16 who has a taxable income of at least $233 a year. In Sweden, this would automatically blanket in virtually all of the population with the exception of those receiving national old-age and disability pensions. "Persons earning over $3000 a year can carry an additional amount of insurance if they so desire," the letter goes on, "and will be entitled to larger sick benefits. On an annual income of $1750, the health insurance premium is $25.22 yearly."

Benefits Outlined

Among other broad benefits, the plan provides maternity protection for working women. "Expectant mothers will receive 65 to 70 per cent of their normal earnings during the compulsory maternity leave," the letter states. "All mothers will receive a bonus of $52.38 for child-birth, in addition to free public-ward confinement in hospitals, or free obstetrical care at home."

Fees Noted

Fees for physicians will be reimbursed 75 per cent up to a set ceiling, as will fees for dental work in clinics, high schools, and hospitals. Public-wards will be free. Transportation to and from the hospital, or by the doctor to and from the patient's home, will be reimbursed wholly or in part. Certain medicines will be furnished free of charge and others at reduced prices.

Apparently this news letter information was based on a report in the *New York Times* by George Axelson who stated that "the project has split the Swedes," especially the medical profession. He said, "Some physicians fear that work will be doubled or tripled, and that half their time will be taken up with filling out forms and complying with other 'red tape' requirements. The majority of practitioners, however, are observing a wait-and-see attitude."

NEW GROUP TO STUDY HEALTH PLANS

Newly organized is a commission to study various types of plans through which the American people receive medical services. The group, composed of thirteen American Medical Association members, was announced at the clinical meeting of the AMA in Miami in December. A comprehensive survey is to be made by the commission, which will require at least a year to complete.

Member from Minnesota

Representing the AMA's House of Delegates is Dr. Frank J. Elias of Duluth; Dr. Leonard W. Larson of Bismarck, North Dakota will head the commission. In announcing the group's formation the chairman of the AMA Board of Trustees, Dr Dwight H. Murray, Napa, California said, it will have a direct bearing on the improvement of health benefit plans now offered to the American people.

Dr. Larson noted, "Because of the great interest in medical care plans at present, on the part of physicians, the federal government and the people, the American Medical Association has felt for some time that a complete factual study of all types of plans was needed. The survey will include both medical and lay-sponsored plans."

He also stated that the new commission will inquire into the nature and operation of medical service plans, the quality of medical care provided, as well as the legal and ethical status of the various arrangements now being used.

LEGION COMMANDER ADDRESSES M.D.'s

The AMA and the American Legion have agreed to try to reach a common ground on their differences.

Speaking at the AMA clinical session in Miami last December, Seaborn P. Collins, national Legion commander, offered to appoint a committee to meet jointly with an AMA-appointed committee to study these differences and try to work out some sort of solution to the debate on the VA program for non-service-connected disabilities.

In his talk before the House of Delegates, Mr. Collins pointed out that, in spite of recent differences, both AMA and the Legion are committed to the "common objectives of strengthening the security of our nation and the welfare of all of our people."

Mr. Collins urged the AMA to consider the Legion's viewpoint on the matter of service-connection. He stated:

> "The American Legion neither expects nor wants the Government to give carte blanche entitlement to medical care to all veterans. We have not asked for it. The VA's goal now is 128,000 beds—for more than 20 million veterans. We are not seeking any major increase in this goal.
>
> "At the same time, the American Legion does not want to see any war veteran who is sick and in need go without proper treatment. We know the medical profession doesn't, either. Further, we believe that the war veteran with honorable service has earned such necessary treatment from the Government he served."

In summary, Mr. Collins declared:

> "The American Legion and the American Medical Association are united in our separate determination to protect this nation from the insidious, destructive forces of Communism. We are united in our determination to secure for America an adequate, economical program of national defense. We are united in our determination to imbue our citizens with a fierce love of their Country and an abiding faith and confidence in the imperishable principles that are our heritage. . . .
>
> "Let us make certain that nothing ever comes between us to imperil this essential unity of purpose and action."

PUBLIC SERVICE EXPLAINED BY DOCTOR HESS

The president-elect of the American Medical Association, Dr. Elmer Hess, gave the keynote address before the PR conference at the Interim session of the AMA in Miami. His address, distinguished and comprehensive, carried many thoughts on what public service means, his key point being that personal sacrifice is the center of such service.

In bringing up this point, Dr. Hess quoted a seventy-two-year-old statement by an Illinois physician:

> "The amenities of professional intercourse, and the obligations of medical men toward each other and the public, were perhaps better observed in 1850 than now. Then the doctor, next to the minister, was the trusted friend and counselor of every family to whom he ministered. He shared their joys, soothed their sorrows, and every passing year added to and cemented the attachment of affection between them. Now the doctor is regarded more in the light of a tradesman or mechanic, and is employed from the same consideration that a grocer, tailor or shoemaker is. The strong ties of gratitude and affection have almost ceased to exist. Relationship is now placed upon a mere commercial basis, and for this the profession is more to blame than the public."

In spite of the age of this quotation, its application to medicine today is just as correct, and bitingly so. Although the field of public relations seems to many of us of fairly recent development, Dr. Hess noted, the attempt to bring the profession into good light in the public eye, has been long in operation. Dr. Hess said, "A public relations program, as I see it, is a concentrated effort to do good and to gain public recognition of such deeds. In too many instances the inverse attitude is maintained. A drum-beating campaign is established to create the impression of altruistic purpose with no sincere desire to back up fine words with suitable deeds."

Dr. Hess warned that in trying to bring the profession into a more favorable light, the profession may have neglected to do some soul-searching on its own. He asked, "Is it possible that we spend too much time telling each other what great humanitarians we are while the pot of lay discontent is boiling over in the next room?"

He urged physicians to reconsider a rededication to the firm basis of the profession itself: service. "So far as I'm concerned the most important rule that physicians should follow is that we *must* care for the sick and infirm, regardless of fees, salaries or honorariums," he declared. "The backbone of medical ethics is summed up in a short sentence: 'Service to suffering humanity, period.'"

His concluding remarks were: "Words alone are not sufficient to convince a cynical public of the humanitarian purposes of medicine. Let us not just try to **convince** the public; let us DO for the public."

REPORT SHOWS UNITED STATES COST OF SICKNESS

Some favorable information on doctors' fees in relation to the cost of sickness in the United States, is shown in a recent special article in *U. S. News & World Report* magazine. Quoted in the AMA Secretary's Letter, the article states: "Government experts attribute most of the rise in hospital expense to higher charges resulting from better pay scales for hospital workers and the higher cost of food."

Doctors' Fees Less

"Dentists and physicians, on the other hand," the article goes on, "are getting a somewhat smaller share of the nation's private medical outlays. This finding runs counter to criticism of physicians' fees." Further explanation is made:

> "Physicians' fees used to account for 30 per cent of the out-of-pocket cost; now they are 29 per cent. Dental bills are less than 10 per cent today, compared with more than 12 per cent in 1948. These fees tend to stay fairly constant for periods of years. The last big jump came during World War II. Today many physicians are charging the same for office calls and sickroom visits as they did shortly after the war."

The report stated that medical care is costing people a total of 12.4 billion dollars a year, of which 2.1 billion is met through health insurance.

Almost 400,000 nurses now are on active duty in the United States, an increase of 16,000 in five years, according to a Public Health Service survey. The estimate shows the following: 231,000 hospital nurses, a 15 per cent increase in four years; 74,000 private duty nurses; 35,200 nurses working in physicians' offices; 25,300 public health nurses; 14,000 industrial nurses; 8,200 nurse educators and 1,900 in a variety of other fields. Although the ratio of nursing personnel to patients is at an all-time peak, 74 per 100, Surgeon General Scheele said the need for nurses justified the present annual student nurse recruitment goal of 55,000.

CLINICAL-PATHOLOGICAL CONFERENCE

(Continued from Page 116)

normalities. The right lung weighed 730 grams, the left 510 grams. Moderate pulmonary edema and patchy bronchopneumonia were evident. The liver weighed 2480 grams, showed only engorgement of the central sinusoids microscopically; no evidence of hepatitis, cirrhosis or fatty metamorphosis could be found. The spleen weighed 210 grams, was not abnormal grossly or microscopically. The gastrointestinal system and pancreas were not abnormal. The right kidney weighed 150 grams, the left 225 grams; they showed no gross or microscopic pathology and the ureters were not dilated. A suprapubic cystotomy opening was still patent and the urinary bladder mucosa very hemorrhagic. The prostate was enlarged considerably, measuring 7 by 6 by 6 cm., remarkably firm, homogeneous and gray-white. The microscopic appearance was that of a characteristic well-differentiated adenocarcinoma. The adrenal glands were not abnormal grossly or microscopically. Many periaortic and supraclavicular lymph nodes showed metastatic carcinoma. The sternum and lumbar vertebrae showed metastatic carcinoma to such a degree that only an occasional focus of uninvolved marrow could be found on microscopic examination.

In view of the clinical course and laboratory findings, it was our opinion that the hemorrhagic diathesis could not be explained on the basis of bone marrow replacement by metastatic tumor alone, and that the fibrinolysin system, as pointed out by Dr. Goldish, was probably the major factor in the bleeding process.

Dr. John Wolff (Duluth Clinic): I agree that this patient had a thrombocytopenia, probably myelophthisic, and circulating fibrinolysin in significant amounts; I doubt whether they are related to each other. The apparent paradox of failure to clot with addition of thrombin but an almost normal plasma fibrinogen may be resolved by the concept of antifibrinolysin in normal plasma. The possibility of acute adrenal insufficiency as immediate cause of death must be kept in mind because of the relatively rapid withdrawal of ACTH.

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Public Health

GUIDE FOR MEDICAL POLICIES FOR LOCAL PUBLIC HEALTH NURSING SERVICES

These medical policies have been jointly agreed upon by the Public Health Nursing Committee of the Minnesota State Medical Association and the Section of Public Health Nursing, Minnesota Department of Health.

Preamble

A public health nursing program for any community includes:

1. Part-time home nursing care and rehabilitation nursing for the sick and disabled.
2. Health guidance for families.
3. Participation in community measures to prevent disease.

In addition to part-time home nursing care service, the public health nurse offers health guidance to the entire family. She serves families through the home, school, at work and in clinics. She has contacts with many persons who are not ill but who might benefit by health instruction and health guidance.

Public health nursing services should be available to everyone in the community—rich or poor and without regard to race, color or creed. Bedside nursing care services should be provided on a fee basis in accordance with the individual's ability to pay.

Another principle of a sound public health nursing organization is that every local public health nursing service appoint a medical advisory committee. A public health nursing service can function successfully only in close co-operation with the medical profession.

Representation on the medical advisory committee of the local public health nursing service should include the local medical society, medical specialty groups, and the health officer. In areas where there are few physicians, all of them may be members of the committee.

The chairman of the medical advisory committee should be a member of the administering body (the board) of the local public health nursing agency. Written medical policies for the agency should be prepared and signed by the members of the medical advisory committee.

Functions of the Medical Advisory Committee

1. To discuss and recommend medical policies to be followed by the nursing staff.
2. To make recommendations to the Board regarding the initiation and continuance of special phases of the agency's program that involve medical consideration.
3. To help prepare or to approve any publicity that involves medical information or that is to be sent to physicians; and to help with the selection of medical speakers for annual and public meetings.
4. To act as liaison between the public health nursing service and the medical society.
5. To interpret the organization to the medical society.
6. To approve the content and frequency of health examinations of the staff before and during employment and to advise regarding a health program for the staff.

Suggested Medical Policies

Except under highly unusual circumstances, public health nurses should not be expected to answer to the directions of persons other than licensed physicians and surgeons. Provided, however, that public health nurses may carry out nursing functions under the direction of other licensed health practitioners insofar as the practices of the latter, in medical and surgical fields, are within the limits of their licensure, and insofar as such orders may be compatible with the training and experience of public health nurses to perform.

The public health nurse rendering bedside nursing care and rehabilitation nursing services to acute or chronically ill persons at home, shall obtain verbal or written orders directly from the physician. Chronically ill patients shall be seen by a physician not less often than every six months if nursing service is to be continued.

Written orders for any treatment or for hypodermic therapy must be obtained from the physician.

Records of nursing services to individuals or families are kept in a confidential file in the office of the local public health nursing agency. The nurse should make a written report to the referring physician and/or agency of the services she has rendered to the family.

Approved by the Minnesota State Medical Association, December 12, 1954.

Hypodermic Policy.—Nurses are permitted to give intradermal, subcutaneous and intramuscular injections on written orders from the physician. Orders should include the name of the drug, dosage, frequency and method of administration and any precautions necessary The nurse may accept verbal orders if arrangements are made to have them in writing by the next day. The administration of one dose of insulin upon direct verbal order of the physician is included in this policy. Families may be instructed to give intramuscular injection on written request of the physician.

The nurse may *not* give intravenous injections, cold shots, or drugs which may commonly cause severe reactions. The Medical Advisory Committee will be consulted about the requests for any questionable or unfamiliar therapy.

At such times as the services of licensed practical nurses, nurses aides or homemaker services are indicated, the public health nurse will plan accordingly with the family and the family physician. However, the public health nurse will continue to assist the worker and the family on the basis of need. The public health nurse, after consultation and in co-operation with the family physician, may refer patients to the local public and private health and welfare facilities.

The establishment of clinics or other health activities which involve medical service shall be undertaken only upon approval of the local physicians in the community or county or District Medical Society.

Suggested Standing Orders

(Until nurse can contact physician)

The following orders are used only until the nurse can get in touch with the physician. No more than two visits shall be made to an ill patient who is not under the care of a physician. If there is no physician, the family is urged to select and call one and an explanation is made that the nurse cannot continue care except under the physician's directions.

1. New Patient: Bathe and make patient comfortable. Instruct family as to care.
2. Burns: Apply sterile dressings to superficial burns. If burns are severe, advise and help arrange immediate medical care. If transportation of the patient is indicated, extensive burns may be covered with a sheet. Avoid use of ointments.
3. Surgical Dressings: Dressings may be reinforced.
4. Minor abrasions and cuts: Wash with soap and water and cover with clean dressing.
5. Varicose Ulcers: Cleanse; apply sterile dressing and bandage firmly.
6. Sore Throats and Colds: Isolate, urge fluids.
7. Suspected communicable diseases: Isolate and give nursing care and instruction as indicated. If family refuses to call physician, notify local health officer.
8. Discharging Ears: Clean outer ear with swabs moistened with boiled water. Dry thoroughly. Do not irrigate. Urge prompt medical attention.
9. Accidents: The nurse will limit herself to those accepted procedures described in the American Red Cross First Aid Handbook. Medical care should be arranged for immediately.

Suggestions About Program

Health of Mothers.—The public health nurse's work with mothers and infants is an important part of public health service.

In working with expectant mothers the aim is to bring the women under early and continuous medical care and to teach the elements of prenatal hygiene and good nutrition practices. The public health nurse should secure orders from the individual physicians for treatment and nursing procedures to be carried out for their patients. Prenatal classes for patients are highly encouraged. They should be organized under the auspices or with the approval and active support of the local physicians.

It is most important that the mothers be instructed to return to their physicians for their postpartum medical check-up.

Encouragement is given to mothers to breast feed their infants except where contraindicated. Instruction is given in the care of the breasts and the proper techniques for breast feeding.

Health of Infants and Preschool Children.—Every effort should be made to contact the mother of a new infant within 48 hours after the infant's discharge from the hospital. In the case of the prematurely born infant the mother should preferably be contacted before she leaves the hospital and plans should be made for preparing the home for the prematurely born infant and for subsequent visits to the home with the approval of the attending physician.

The value of continuous health supervision and appropriate medical services for the infant and preschool child by the private physician or clinic should be stressed. This health supervision includes determination of the health status of the child and deviations from normal; immunization against such diseases as diphtheria, pertussis smallpox and tetanus; and counseling in all phases of infant and child care and growth and development. The public health nurse supplements the instruction given by the physician and elaborates on the details, especially as concerns techniques of food preparation and feeding, daily care of the infant, good hygiene practices, habit training accident prevention, et cetera.

Health of School Children.—School health work may operate under the immediate direction of the school administrator. The "School Health Manual for use in Minnesota Schools" should constitute the base line upon which programs for the health of school children are constructed. In addition, the nursing board and the school health council (if there is one), each with medical, dental and lay representation, should help guide the service. The nurse consults with parents on problems of child health and environment and guides parents to other sources of aid which may be needed. The nurse co-operates with the school staff, parents, physicians and dentists in arranging for and conducting health programs in such a manner that they become an educational experience for all participants.

The school nurse aids in the control of communicable disease by helping parents and teachers recognize conditions for which the child should be isolated. Primary responsibility for exclusion of sick children is fixed by Minnesota State Board of Health Regulation number 318 which reads:

> "The person in charge of a school shall exclude from school all children who (a) return to school after an illness of unknown cause, (b) appear to be in ill health, (c) have lice or other vermin. The parents of such children shall be notified of the reason for exclusion and such cases shall be referred to the family physician, the school physician or the health officer. Children shall not be readmitted until they obtain a certificate from the health officer or his authorized agent."

Crippled Children Services.—The State Service for Crippled Children merits full co-operation of all interested local agencies to fulfill its functions. The prevention and correction of physical handicaps due to bodily defects needs supervision over long periods of time. The nurse works with the individual physician in the care and supervision of these cases.

Due to her close contact with the patient, the public health nurse may discover deviations from the normal—not only in the infant, but also in the preschool and school child. She helps parents make precise and concrete plans for the regular and continuing medical care of the crippled child.

The nurse's duties in the individual case lie in the education of the family in the patient's care, the demonstration of and instruction in home nursing, the general health supervision of the afflicted child, and the supervision of therapeutic exercises recommended by the physician in charge. The nurse will aid the rehabilitation of the patient in every way.

Communicable Disease Control.—The control of communicable diseases is the responsibility, not only of the public health nurse and the official agency, but is also the concern of the medical profession and general populace. If communicable disease is suspected, the nurse should explain to the family the elementary principles of isolation, concurrent and terminal disinfection and such other measures which will aid in preventing the spread of disease.

In aiding physicians in the control of preventable diseases, public health nurses are expected to co-operate with the local health officer and to follow the Regulations of the State Board of Health.

The nurse should teach families that immunizations for infants should be started at 3 or 4 months of age and thereafter repeated at regular intervals as determined by the physician. Group immunization programs should be conducted under the auspices or with the approval of a local medical society. (A number of biological preparations for mass immunization programs are available without cost upon application to the Minnesota Department of Health.) The nurse's contribution to such programs consists of participation in preliminary education of the community, assisting with the necessary arrangements for carrying through the programs, assisting the physicians when the immunizations are being performed, summarizing reports, and keeping local records of immunization.

Tuberculosis Control.—The public health nurse contributes to the control program through her work with the family. She is often able to bring a suspected case of tuberculosis to the attention of the physician, thereby assuring an early diagnosis. She teaches the need for participating in tuberculin testing programs and in mass x-ray surveys.

Her service to the family includes sharing information about the disease; nursing care services to patient in the home; taking steps to prevent the spread of tuberculosis and helping the patient and family make necessary adjustments.

Rehabilitation and the return of the patient to his community and his family needs skillful guidance and arrangement of an environment in which the patient can carry on the recommended treatment.

If drug therapy is to be given a patient by the nurse, she should first secure from the supervising physician specific written instructions for her guidance; from time to time these instructions should be changed to fit changes in the prescribed treatment.

Minnesota Academy of Medicine

Meeting of March 10, 1954

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, March 10, 1954. Dinner was served at 7 p.m. and the meeting was called to order at 8:20 p.m. by the vice-president, Dr. C. N. Hensel.

There were forty-eight members and one guest present.

Minutes of the February meeting were not read due to the length of the scientific program.

The following memorial was presented at this meeting.

Arthur Edwin Benjamin

1868 - 1953

Dr. Arthur Edwin Benjamin, a member of this Academy since 1903, died October 1, 1953, after an active practice in Minneapolis covering a span of over sixty years. He was born in Hutchinson, Minnesota, in 1868. His father and grandfather were physicians.

Dr. Benjamin received his degree in medicine from the University of Minnesota in 1892. Even before graduation, his teaching ability was recognized and in 1890 he was made an assistant instructor in gynecology. After graduation, he continued in the department of gynecology and, after postgraduate study in London, Vienna and New York, was made chief of one of the gynecological services at the Minneapolis General Hospital. He served in this capacity until his retirement as professor emeritus in 1938. He continued on the service as consultant. By those of us who served under him during those years, his conscientious care of the patients, the excellent results of his diagnostic acumen, surgical judgment and operative skill, and his constant kindly and helpful consideration of his staff will long be remembered.

Dr. Benjamin was very active in the Hennepin County Medical Society and became one of its presidents. He was always most conscientious in the performance of any assignment given to him during his long association with that Society. Other medical society memberships included the Minnesota State Medical Association, the American Medical Association, the American College of Surgeons, the Western Surgical Society and the Southern Minnesota Medical Association. He belonged to the staffs of St. Barnabas and Northwestern Hospitals, and was once chief of staff of the latter.

Dr. Benjamin, though singularly quiet, retiring and unassuming, was a man of tremendous energy and wide interests, social, political, religious and artistic. His affiliations with organizations devoted to these fields have been amply memorialized in the public press and scientific journals.

In the course of his membership in this Academy, he was always very modest, never presenting or discussing any paper unless he felt strongly that his opinion, founded upon sound study and a wide personal experience, would be of value. As those acquainted with his writings well know, he devised many clever techniques and operations.

In our social hours, we shall miss his friendly presence and his studied comments on any one of a wide range of important subjects in which he was interested. Especially shall we miss his observations upon the changes that took place during the long span of his practice, observations spiced with humor and always kindly.

Dr. Benjamin is survived by two sons, Edwin G. and Harold G. Benjamin, who are carrying on the family tradition of medicine and are practicing in Minneapolis; two daughters, Mrs. Charles J. Hoover of St. Louis Park, and Alice, who is the principal of Summit School, Saint Paul; a sister, Mrs. Olive Bacon of San Gabriel, California; a brother, Dr. Winifred G. Benjamin, a dentist in Minneapolis; and seven grandchildren.

R. T. La Vake, M.D.

The secretary announced the Executive Committee had approved the transfer of Dr. A. G. Schulze to the Senior list. Upon motion, this was seconded and carried.

The scientific program followed.

Dr. C. A. McKinlay, of Minneapolis, read his inaugural thesis on "Clinical Observations Related to Pulmonary Eosinophilia." Lantern slides were shown.

CLINICAL OBSERVATIONS RELATED TO PULMONARY EOSINOPHILIA

C. A. McKINLAY, M.D.
Minneapolis, Minnesota

This paper presents two cases of eosinophilic pulmonary infiltration, both of them transient and recurrent, in which observations were made during illness and subsequent health over a period of twenty patient-years.

Personal interest in eosinophilic pulmonary infiltration had followed observation in 1939 and report in 1941 by Ellis and myself[9] of a case of pneumonia (S. L., a twenty-two-year-old white man) with massive migratory pulmonary infiltration associated with high (48 per cent) blood eosinophilia. Prompt clinical recovery followed discontinuance of prontosil, which had been given on another service for the treatment of colitis. At no time had there been marked prostration. Our attention was called to the report by Löffler,[14] in 1932, of fleeting pulmonary infiltration with eosinophilia. Other reports from Europe followed soon. The importance of the lung as a shock organ had long been recognized, and true bronchial asthma was universally accepted as a manifestation of hypersensitiveness. However, extensive pneumonitis as an allergic response to agents other than infection was a relatively new concept; in fact, no cases occurring in the United States were then found reported. The next case I wish to cite is one that had been observed by the late Dr. P. Hallock and by Dr. W. W. Spink in 1941, and by the writer since 1949.

Case Reports

Case 1.—H. F., a white man aged forty-six, a contractor, was admitted to the hospital March 24, 1949, because of a respiratory infection of two weeks' duration associated with moderate malaise.

Past History.—The hospital record of 1941 was reviewed. At that time, the patient had been observed in the hospital with atypical pneumonia and had been discharged as recovered. Following readmission to another hospital one week later, tachycardia and bouts of fever were noted. There were areas of infiltration in both lungs. The leukocyte count varied from 8,100 to 15,200 with maximal eosinophilia of 27 per cent. There were occasional eosinophils in the sputum. The electrocardiogram was abnormal, with a negative T_2. Toxic myocardium was suggested. Dr. Spink suggested Löffler's syndrome. Omission of sulfadiazine was suggested. Potassium iodide gr. XV daily appeared beneficial on repeated trials. The patient was afebrile when discharged.

Physical Examination.—On March 24, 1949, there were râles, chiefly at the left base. The chest x-rays showed multiple extensive areas of infiltration in both lungs. The leukocyte count varied from 8,100 to 18,900, with eosinophils of 6 to 21 per cent. The sedimentation rate was 96 mm. per hour; heterophile agglutinins were present in a titer of 1:20. The sputum showed no predominating organisms. The urinalysis gave negative findings. Skin tests using multiple allergens from various sources were negative. Muscle biopsy findings excluded periarteritis nodosa in one specimen. The maximal temperature was 102° F.; usually it was below 101° F. There was depression of the temperature for three days following the use of potassium iodide gr. V, but fever recurred. Potassium iodide gr. X was resumed April 18 and continued until discharge on April 30, with associated relief from fever on most of the days.

The patient was next admitted to the hospital on June 16, 1949, one week after having had an attack of biliary colic with associated jaundice and clay-colored stools for two days. During hospitalization, the patient improved, was afebrile, and the eosinophils dropped to 7 per cent. Eleven days later, during readmission to the hospital and after the jaundice had subsided, fever. leukocytosis and eosinophilia reappeared. While jaundice was present, there was amelioration of the pulmonary eosinophilia. At the present time the patient is well, thirteen years after his first attack, the x-ray shows no evidence of infiltration, and the blood count remains normal.

Case 2.—Mrs. C. B., a farm housewife aged thirty-five and mother of five children, was first examined on April [illegible], 1948, with the complaint of fatigue. About six months previously, the patient had an acute respiratory infection with systemic reaction for which she was hospitalized for two weeks. X-ray examination elsewhere suggested pulmonary tuberculosis to the radiologist. For ten days, she received penicillin and, for four days, a small amount of a sulfonamide. During this period, asthma developed. This subsided after a brief interval, but ever, malaise and persistent cough continued and she equired almost constant rest in bed. Scratch tests with penicillin were negative.

The laboratory findings at variance from normal were: emoglobin 11.3 gm., leukocytes 11,100 and 39 per cent osinophilia. At another time 45 per cent eosinophilia was noted; the sedimentation rate was 114 mm. per hour. Negative findings of importance were: absence of ova nd parasites, normal serum protein, normal heterophile eaction, negative blood culture, absence of acid-fast acteria and negative bone-marrow except for marked eosinophilia. The treatment was symptomatic; the temperature was only slightly elevated on three of eleven hospital days.

After three years of good health, the patient was next observed on March 18, 1953, with fever, fatigue and dyspnea of six weeks' duration. There were minimal physical findings in the chest. X-ray examination showed extensive infiltration in both lung fields. The routine laboratory findings were: hemoglobin 11.7 gm., leukocyte count 11,800 and 20,600, with 38 to 48 per cent eosinophils; sedimentation rate 112 mm. per hour; sputum showed mixed growth. Clinical improvement occurred, and the patient was discharged. She was readmitted about two months later because of recurrent bouts of respiratory symptoms, fever and fatigue. Liver function tests were normal. Moderate fever subsided during ten days' hospitalization. The patient was readmitted July 13, 1953, because of recurrence of fever, fatigue and cough. The hemoglobin was now 10.1 gm.; the leukocytes were 14,000, with 36 per cent eosinophils. Cortisone was started orally and given daily in the maximal amount of 200 mg. There was immediate clinical improvement. On the third day of treatment, with a leukocyte count of 12,400, the eosinophils numbered 1 per cent; on the following days the eosinophils were 0, 1, 3, 1 and 10 per cent. This response is in keeping with the known influence of cortisone in lowering blood eosinophilia in persons with intact adrenals. She is now on a small maintenance dose of cortisone and is in excellent health, according to Dr. R. F. Gammell, through whose courtesy the patient was examined. Rose[6] first reported benefit in a case of Löffler's syndrome following the use of cortisone.

Other Cases

C. T., a man aged twenty-four, currently being observed, is cited with an upper respiratory tract infection associated with eosinophilia (32 per cent). This case illustrates that various infections and infestations provoke eosinophilic blood response without causing pulmonary infiltrations.

The high degree of eosinophilia (53 and 44 per cent) noted in asthmatic bronchitis without pulmonary infiltration and in intestinal parasitism is illustrated by the case of V. S., a man aged twenty-three, a native of India, recently observed. It has been suggested that eosinophils develop as a result of destruction of body tissue. The dissimilarity of pulmonary eosinophilia to eosinophilic granuloma of the lung should be noted. Histamine may be contained in the eosinophils of the blood.

Blood eosinophilia is an incidental finding in certain pulmonary conditions, such as sarcoidosis, hydatid disease of the lung, Hodgkin's disease and the resolving stage of pneumonia. Since 1941, there has been reported an increasing number of cases in which pulmonary infiltrations have been observed radiologically and which have been accompanied by blood eosinophilia. The term "Löffler's syndrome"[9] has included only a part of this group of diseases and should be limited to those cases with transient pulmonary infiltration and eosinophilia. There is a growing tendency to link Löffler's syndrome, erythema nodosum and tropical eosinophilia, in which there may or may not be infiltrations, under an inclusive term. Crofton and associates,[6] in their review in 1952 of some 450 cases reported in the literature, suggested the term "pulmonary eosinophilia." Under this term, they suggested the classification of (1) simple pulmonary infiltration or Löffler's syndrome, transient infiltrations; (2) prolonged eosinophilia, prolonged or recurrent infiltrations without asthma; (3) pulmonary eosinophilia with asthma, infiltrations with asthma; (4) tropical pulmonary eosinophilia usually with asthmatic symptoms; (5) polyarteritis nodosa.

Löffler's syndrome in the continental cases reported has commonly been associated with infestation by Ascaris lumbricoides. The complete syndrome was reproduced by Vogel and Minning[21] in five of six volunteers by feeding Ascaris eggs. Other worms have been incriminated, such as Ancylostoma braziliense causing creeping eruption or itch reported by Wright and Gold,[22] Taenia saginata by Benda and Weinberg[2] and hydatid of the liver by Dörig.[8] Among miscellaneous allergens are pollens, such as privet,[10] beeswax,[11] and sulfonamides.[5] Cases with significant agglutinin titers for Brucella abortus were found. There were two with Strongyloides stercoralis infestation,[4] and one with microfilariae in the sputum. This syndrome has also followed penicillin therapy.[17]

Tropical eosinophilia, or tropical pulmonary eosinophilia, has been reviewed by Danaraj[7] and may represent another variant. In a report of 150 cases in the Orient, 81 per cent had pulmonary infiltration that showed by x-ray. This disease often has an initial febrile stage with respiratory symptoms followed by bronchitis, which becomes the most prominent feature and which may last for months or years. There is an asthmatic stage which may likewise last for months. The most dramatic aspect is the response to organic arsenic administered orally or intravenously. The cause is not yet known. Danaraj[7] rejected the theory of hypersensitivity.

Pulmonary eosinophilia may occur with asthma. Harkavy,[13] in 1943, described fifteen cases of asthma associated with recurrent pulmonary infiltration and eosinophilia. In fatal cases, in addition to edema and cellular exudate, there were lesions in the blood vessels, including acute necrotizing arteritis. This observer[13] noted widespread tissue involvement. A transient eosinophilic gastric lesion has been described.[20] Bayley and co-workers[1] reported a fatal case of Löffler's syndrome in which the outstanding pathologic findings were the large number of eosinophils in the pneumonic exudate, granulomatous lesions and necrotizing arteritis. Bergstrand[3] described similar and widespread findings of polyarteritis nodosa in four asthmatic persons who had transient pulmonary infiltrations and eosinophilia. The pathological variation from simple pulmonary eosinophilia, or Löffler's syndrome, to the widespread necrotic arteriolar changes of polyarteritis nodosa somewhat parallels the range of clinical manifestations from simple pulmonary eosinophilia. Crofton's group[6] argued that the variants described are different grades of the same fundamental process. The diversity of causative agents, the eosinophilia, and frequently the family history of allergic illness, suggest that the process is one of hypersensitivity. Rich[18] has shown that polyarteritis nodosa is a reaction of hypersensitivity. Some allergens such as those that might be implicated in tropical eosinophilia appear to be powerful enough to produce the reaction in most people. Reeder and Goodrich[16] reported, among others, a fatal case in which progressive periarteritis nodosa accompanied the "PIE-syndrome"; this latter term was suggested by these authors for pulmonary infiltration with eosinophilia. Mark[15] recently reported twenty-three cases of Löffler's syndrome and considered that the role of allergies is minor in the development of pulmonary change.

Summary

Cases herein reported include an interesting experiment by nature in which the fortuitous occurrence of jaundice benefited a patient who had recurrent pulmonary eosinophilia in a manner similar to that of the influence of jaundice upon rheumatoid disease, first stressed by Hench.[13] Another patient received benefit from the use of cortisone, which linked this case physiologically to the case in which benefit was derived from jaundice. Because of their relatively benign nature, these cases, with recovery after repeated transient pulmonary infiltrations, contrast with the irreversible stage of periarteritis nodosa which sometimes supervenes in the syndrome of pulmonary eosinophilia.

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Discussion

Dr. Leo G. Rigler, University of Minnesota: It is interesting to me to hear Dr. McKinlay mention tropical eosinophilia. When I was in India, I heard Dr. Sanjivi report on a large number of such cases. They differed somewhat from the typical Löffler syndrome which Dr. McKinlay has discussed in detail. In India, tropical eosinophilia lasts for a long time and the condition has been concentrated in children and young adults. It simulates miliary tuberculosis very closely in the roentgenogram. Since miliary tuberculosis is extremely common in India, my first impression on seeing such cases was tuberculosis, but the clinical picture is not at all like miliary tuberculosis. The eosinophilia is commonly as high as 60 to 70 per cent. Dr. Sanjivi studied the cases intensively but did not succeed in determining the etiological factor; so far as I could determine, he was completely mystified as to the cause.

Dr. F. W. Lynch, Saint Paul: As Dr. McKinlay has pointed out, the problem of eosinophilic infiltration interests practitioners in many fields of medicine. Observations in the dermatologic field may be of interest to others because we can more easily perform biopsies and thoroughly study the lesions which we see. Eosinophilic leukemia is rare and even when present it is very rarely accompanied by specific cutaneous manifestations and therefore this problem does not deserve our consideration tonight.

Eosinophilic infiltration often accompanies allergic dermatoses but this too is beyond the range of tonight's discussion. We also see fixed granulomatous circumscribed lesions, chiefly on the face, persisting for months or several years and not easily influenced by treatment. They have been of particular interest because one would suspect they would be accompanied by some systemic manifestations, though this has proved not to be the case. Perhaps they are comparable with fixed eosinophilic infiltrations in other tissues, as in the bone or the lung. However, many or most of the osseous lesions seem more closely allied with Hand-Schüller-Christian disease and therefore have much more serious implications than do the fixed circumscribed lesions. To the dermatologist it seems difficult to link all these varied eosinophilic infiltrations and we regard them as separate entities. They have in common only the presence of large numbers of eosinophils. Changes in white blood cell counts do not parallel the cutaneous nor the osseous findings.

Dr. E. P. K. Fenger, Oak Terrace: I enjoyed Dr. McKinlay's paper very much. Dr. Rigler has made the statement that pulmonary eosinophilia may be mistakenly diagnosed as tuberculosis. I find that at Glen Lake Sanatorium, during the past fifteen years, we have had seven such cases admitted. These seven cases fit into Dr. J. W. Crofton's classification very well. There were two in the simple eosinophilia group, three in the prolonged pulmonary eosinophilia group, and two in the group of pulmonary eosinophilia with asthma. I think there are two other clinical entities that should be mentioned; one is eosinophilic pleural effusion and the other is eosinophilic granuloma of the lung.

Dr McKinlay, in closing: I wish to thank the members very much for their discussions. It may be of interest to mention that patient H. F., while on a trip in the Caribbean area in 1951, had recurrence of eosinophilic pulmonary infiltration. The patient was advised by local physicians of the relative frequency of occurrence of the disease in the neighboring tropical communities.

* * *

Dr. Lyle J. Hay, Minneapolis Veterans Hospital, Department of Surgery, gave his inaugural thesis on "Transplantation of the Duct of Wirsung in the Treatment of Chronic Pancreatitis." Case reports were given and lantern slides were shown. (Thesis to be published later)

Discussion

Dr. A. A. Zierold, Minneapolis: It has been very interesting to listen to Dr. Hay's recapitulation of the theory of the common pathway of the biliary and pancreatic ducts. Those of us who are accustomed to employ operating-room cholangiograms have found this to be demonstrable in a surprisingly large percentage of cases. Considering acute pancreatitis, there can be little question as to its identity, but I believe that what we are accustomed to call recurrent subacute or relapsing pancreatitis may quite as well be a symptom complex arising from the reflux of the pancreatic secretion into the biliary tract. Whatever the case may be, Dr. Hay has demonstrated that symptoms arising out of the common pathway may be obviated by transplantation of the pancreatic duct. It is probable that the only other choice of treatment is section of the sphincter of Oddi.

It is gratifying to hear Dr. Hay's experience and I shall be interested in a comparison of the two procedures after more cases have been accumulated and further time has elapsed.

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

TERATOMA OF THE RECTO-SIGMOID

(Continued from Page 110)

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In Memoriam

LYLE V. BERGHS

Dr. Lyle V. Berghs, native son of Owatonna, died in Albert Lea on December 4, 1954, of coronary thrombosis. Dr. Berghs was on his way to Kiester where he was to have re-entered private practice with another physician. He was forty-eight.

Dr. Berghs' death followed several years marked by extensive periods of hospitalization and surgery in an attempt to correct his circulatory condition.

Dr. Berghs was the first volunteer to answer the Steele county medical draft during World War II. He served thirty-seven months of intensive service, with twenty-two months overseas, in England and in North Ireland. As ship surgeon, he was wounded in a strafing attack before D-Day and was hospitalized in England, but he recovered enough to join the combat and attack forces on D-Day and served with his medical unit at the front lines through the heaviest drives.

He was born in Owatonna on April 20, 1906, and attended grade and high school there. He entered the University of Minnesota and graduated from medical school in 1929, interning at Minneapolis General Hospital. In 1930, he began his practice in Owatonna with Dr. D. H. Dewey, and renewed the practice after his service during World War II. In 1952, he was appointed to the staff of Moose Lake Hospital and served for two years.

He had been a member of the Steele County Medical Society, the Minnesota State Medical Association and the American Medical Association, the American Legion, the Veterans of Foreign Wars, Alpha Omega Alpha, the BPOE and the Holy Name Society.

He is survived by his wife, the former Adele Whetstone; one son, Thomas, Winona; one daughter, Peggy, Minneapolis; two brothers, Charles, Park Ridge, Illinois; Larry, Owatonna; two sisters, Mrs. Max Argent, Hampton, Iowa, and Mrs. Warren Gray, Waseca.

KARL EMIL BERGQUIST

Dr. Karl E. Bergquist, Battle Lake, died at Lake Region Hospital on December 10, 1954. Dr. Bergquist, well-known and loved resident of Battle Lake, would have been eighty years old on Christmas eve. He was born in St. Peter.

Dr. Bergquist came to Battle Lake from Duluth in 1935, and specialized in eye, ear, nose and throat work. He was appointed resident physician for the Otter Tail County Sanatorium in December, 1943 and served in that capacity until October, 1954.

Dr. Bergquist received his medical education at the University of Minnesota and at Hamline University, graduating in 1897. He interned at Bethesda Hospital in Saint Paul, and did postgraduate work in Chicago in his specialty.

He was married to Borghild Torkelson of Minneapolis in 1902. Mrs. Bergquist preceded him in death in 1945.

Dr. Bergquist was a life-time member of the Trinity Lutheran Church in Duluth; the Vasa Ama Society, the Park Region District and County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are two daughters: Mrs. Ethel Campbell, South Bend, Indiana; Mrs. C. A. Boline, Battle Lake; four grandsons: John Lloyd Campbell, U. S. Navy; Karl Allan, Jon Edward and Robert A. Boline, Battle Lake; three brothers: Theodore, Lafayette; Oscar, Galesville, Wisconsin; and Ernest, Rapid City, South Dakota.

ROBERT ALLEN CAMPBELL

Dr. Robert A. Campbell, retired Minneapolis physician and surgeon, died on January 7, 1955. He was eighty-six.

Dr. Campbell was an eye, ear, nose and throat specialist who practiced in Minneapolis more than fifty years. He was born in Detroit on December 27, 1868, and graduated from the University of Minnesota Medical School in 1896 and was licensed in the same year.

Dr. Campbell was a member of Phi Gamma Delta and Nu Sigma Nu, medical fraternities; a former member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was a fellow of the American College of Surgeons and was for many years on the staff of Abbott Hospital.

He is survived by his wife, Mary S.; one daughter, Dorothy; one son, Gilbert M., Sacramento, California; one brother, George G., Portland, Oregon; two sisters, Mrs. Mert Patterson, Portland, Oregon and Mrs. William Cowing, Hamilton, Canada.

ROBERT WILSON CAMPBELL

Dr. Robert W. Campbell, prominent Cass Lake physician for many years, died at Rochester, December 13, 1954. He was seventy-five.

Dr. Campbell was born at Parkers Lake in Hennepin County, on February 14, 1879. He obtained his preliminary education at Tracy and received his medical education at the University of Minnesota, from which he graduated in 1905. He set up his practice in Swanville in 1905, moving to Deering, North Dakota, in 1914 and remaining there until 1919. From 1919 to 1926 he practiced in Blackduck, Minnesota, and then moved to Bisbee, North Dakota, until 1930 when he located at Cass Lake, where he remained until his death.

After his graduation, he took one year's internship at City and County Hospital in Stillwater and did postgraduate work in Chicago for six months.

Dr. Campbell founded the Cass Lake General Hospital. He was chairman of the Civic Improvement Committee of Cass Lake, and as such he drew the first plans for the community's lakeshore park. He also belonged to many civic and fraternal organizations.

He retired from active practice about ten years ago. He has been a member of the Upper Mississippi Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Campbell is survived by his wife; a son, Robert, Rochester; and three daughters, Mrs. A. B. North and Mrs. Glenn Harris, both of Los Angeles, and Audrey, believed to be in Minneapolis.

ENOCH HAUGSETH

Dr. Enoch Haugseth, retired Twin Valley physician, died at his home at the age of eighty-three on December 2, 1954.

Dr. Haugseth had been in retirement for about fourteen years at the time of his death, but had been in Twin Valley since 1906, practicing actively for about twenty-five years.

Dr. Haughseth was born on September 21, 1871, in Rendalen, Norway. He became a citizen of the United States in 1896, two years after he graduated as a pharmacist from the University of Minnesota. In 1902, he received his M.D. degree.

After practicing at Lake Park from 1902 to 1906, Dr. Haugseth came to Twin Valley. He was much interested in local church activities and also served for a number of years as president of the local school board, and as local health officer. He was also local surgeon for the Northern Pacific Railway.

Dr. Haugseth had been a member of the Red River Valley Medical Society and the Minnesota State Medical Association.

He is survived by his wife, the former Elizabeth Wold; three daughters, Mrs. Walter Johnson, Twin Valley; Mrs. Arthur Tillinghast, Great Neck, New York; and Mrs. Ralph Fjelstad, Northfield; two sons, Dr. Ellsworth, South Bend, Indiana, and Enoch of Seattle; and fourteen grandchildren. One son, Clarence, Portland, North Dakota, preceded him in death.

GUSTAV M. HELLAND

Dr. Gustav Helland of Spring Grove died at his home after a long illness, on December 8, 1954. He would have been seventy-nine on December 23. He was one of Spring Grove's early physicians, arriving in 1910 and serving until 1945 when ill health forced him to retire.

Dr. Helland was born in Lisbon, Illinois, in 1875. He lived on a farm near Black Earth, Wisconsin, as a child and attended grade and high school there. From 1892 to 1898, he worked as a pharmacist in Chicago; later he owned and operated a drug store in Mount Horeb, Wisconsin. Entering medical school, he received his medical degree from Marquette University at Milwaukee in 1908, practicing for one year in Mount Horeb.

In 1910, he moved with his family to Spring Grove. He served in the medical corps during World War I and rose to the rank of major, remaining a member of the reserve medical corps.

Dr. Helland was a member of the American Legion; the Commercial Club; the National Association of Military Surgeons; the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association, the American Medical Association; the Shrine and the Spring Grove A.F. and A.M. Lodge. He served as president of the Spring Grove Hospital for fifteen years and also served on the school board for a number of years. In 1950, the community of Spring Grove paid tribute to his many years of service.

He is survived by his wife, Julia; four daughters, Mrs. V. V. Lindgren, Portland, Oregon; Mrs. Ronald Shaw, Oakland, California; Mrs. Bevis Ash, Redding, California; Mrs. Dan Morris, Alamo, California; one son, Kenneth W. C. of Janesville, Wisconsin; four brothers, Dr. J. W. Helland and C. J. Helland of Spring Grove; Dr. C. A. Helland, Argyle, Wisconsin; and Randolph O. Helland, Portland, Oregon.

BERT R. KARN

Dr. B. R. Karn, who practiced at Ortonville for forty-seven years, died of a heart attack at the Dyckman Hotel in Minneapolis, January 4, 1955. He was sixty-five.

Dr. Karn, who operated a clinic in Ortonville with his son, J. Francis, began practice with his father in Ortonville in 1907. He received his medical degree the same year from the University of Minnesota.

Dr. Karn was born in Ontario, Canada, in September, 1882, and had his preliminary education in Ortonville. After graduation in 1907 from the University of Minnesota, he took his internship at Washington Hospital in Chicago, where he also took postgraduate work, specializing in eye, ear, nose and throat work in 1916.

Dr. Karn is survived by his wife, and three sons: J. Francis, Ortonville; Richard and William.

DENNIS MICHAEL O'DONNELL

Dr. D. M. O'Donnell died at his home in Ortonville, on December 5, 1954, after a long illness. Dr. O'Donnell had practiced at Ortonville for thirty-seven years. He was sixty-nine years old.

Dr. O'Donnell was born on September 28, 1885, at Aberdeen, South Dakota. In 1910, he graduated from the University of Illinois and was licensed to practice medicine in Minnesota, South Dakota, Illinois and California. He attended Aberdeen High School and Aberdeen State Teachers College at Aberdeen, graduating in 1906. He interned at Chicago Hospital for eighteen months. He practiced in Odessa, Minnesota, for one year; in Huron, South Dakota, for one year before coming to Minnesota to practice in Ortonville. He was a staff member of the Ortonville Evangelical Hospital.

Dr. O'Donnell served as physician for the Milwaukee Railway, the Fire Department and also was chairman of the Board of Health. In 1953, the community paid its tribute to him with a gathering in his home sponsored by the Civic and Commerce Association.

(Continued on Page 136)

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

National

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American College of Chest Physicians (Council on Postgraduate Medical Education) and Laennec Society of Philadelphia, eighth annual postgraduate course on Diseases of the Chest, Bellevue-Stratford Hotel, Philadelphia, Pennsylvania, March 7-11, 1955.

American College of Surgeons, sectional meeting, The Fort Harry, Winnipeg, Manitoba, Canada, April 25-26, 1955.

American Goiter Association, annual meeting Skirvin Hotel, Oklahoma City, Oklahoma, April 28-30, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

International

International Academy of Proctology, seventh annual meeting, Hotel Plaza, New York, New York, March 23-26, 1955.

International College of Surgeons, United States Section, Southwestern Division regional meeting, Shamrock Hotel, Houston, February 28-March 1, 1955.

Inter-American Congress of Radiology, Shoreham Hotel, Washington, D. C., April 24-29, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

International Symposium on Cardiovascular Surgery, Henry Ford Hospital, Detroit, March 17-19, 1955.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled Examinations (Part II) oral and clinical for all candidates for certification by the American Board of Obstetrics and Gynecology will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from May 12 through May 20, 1955. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

FAMILY DOCTORS' DAYS
at the
University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

February 16—Division of Anesthesiology
March 16—Division of Urology
April 20—Division of Neurosurgery

Activities begin with luncheon at 12:15 noon with the staff of the Department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, University of Minnesota Hospitals.

NATIONAL HOSPITAL WEEK

Each year the nation sets aside one week in which to become better acquainted with its hospitals. National Hospital Week in 1955 will be May 8-14. During this week the attention of the American public is focused on our hospitals, in which one out of every eight of us receives care each year. The theme of National Hospital Week in 1955 will be "Your Hospital . . . A Tradition of Service."

National Hospital Week is sponsored annually by the American Hospital Association. Celebration of a National Hospital Day began in 1921 and was expanded to a week in 1953. National Hospital Week is traditionally built around the May 12 birthday of Florence Nightingale, the famous nurse crusader of the Crimean War, whose pioneer service led to the improvement of hospital care in both England and America.

AMERICAN HEARING SOCIETY

The American Hearing Society announces March 1 as opening date for competition for the 1955 Kenfield Memorial Scholarship, an award made annually to a prospective teacher of lipreading. Application blanks may be obtained by writing to the society's national headquarters, 817 14th Street, Northwest, Washington 5, D. C.

Deadline for returning completed applications is May 1. They are to be mailed to Mrs. Dorothy J. Cornett, 4310 S.W. 14th St., Coral Gables 34, Fla. Mrs. Cornett is chairman of the American Hearing Society's Teachers Committee.

Winner of the Kenfield Memorial award is entitled to take a teacher training course in lipreading from any school or university in the United States acceptable to the Teachers Committee. The scholarship is to be used within one year from the date of award.

A satisfactory applicant for the award must be a well-adjusted person with a pleasing personality, legible lips, good speech and voice and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement.

Specifications for a hard-of-hearing contestant include 30 clock hours of private instruction in lipreading from an approved teacher or 60 hours of lipreading in public school classes under an approved teacher. Rules for competition state that an applicant shall plan to teach lipreading with or without other types of speech or hearing therapy.

PHYSICIAN PILOTS ORGANIZE

Some time ago several physicians simultaneously conceived the idea of forming a national society of flying physicians. Initial action was started by Mark E. DeGroff of Tulsa, Olkahoma, medical equipment manufacturer, who offered to act as a central office until preliminary arrangements could be made. A notice in the AOPA *Newsletter* brought forth over one hundred interested inquiries. About twenty-eight physician pilots attended the American College of Surgeons meeting in Atlantic City and twenty-four attended the AMA session in Miami. It was felt that enough interest was shown to warrant an attempt to organize.

It was decided that the purposes of this society should be scientific, educational, and social. Physicians have a considerable influence which should be passed on to everyone to promote greater aviation safety. Further, physicians who have this interest could learn much about the technical aspects of flying from association with each other.

The immediate objectives are: compilation of a complete list of physician pilots; appointment of temporary local area chairmen; the collection of ideas and suggestions; and encouraging physicians to fly to the AMA Meeting at Atlantic City, June 6-10, 1955.

A scientific and social program can be arranged at Atlantic City, if enough interest is shown.

Physician pilots who are interested are requested to send their names, plane flown and landing field to the local chairman of their area, or, if not known, to H. D. Vickers, M.D., 25 Jackson Street, Little Falls, New York, temporary chairman.

MENTAL HEALTH GRANT

The Hamm Foundation of St. Paul has announced a new grant to St. John's Institute for Mental Health at Collegeville, Minnesota.

Except for a nominal tuition fee to be paid by participants, the Foundation gift will cover the complete cost of three week-long pastoral psychology workshops to be held next August at St. John's University for clergymen of all faiths who are interested in mental health problems.

The first series of workshops were held last August. Originally planned for clergymen of the Upper Midwest, the workshops also attracted participants from California, Nebraska, Kentucky, Missouri, Illinois, Rhode Island, Washington, D. C., and Canada.

Minnesota physicians who served on the faculty were Doctors C. Knight Aldrich, Louis L. Flynn, Adelaide Johnson, Hyman S. Lippman, Howard P. Rome, and Clarence J. Rowe.

The second series of pastoral psychology workshops will be held next August. The following Minnesotans have been named as full-time faculty for next summer: Doctors David A. Boyd, Howard P. Rome, Don Hastings, and Louis L. Flynn. Other physicians, such as Dr. Adelaide Johnson and Dr. Hyman S. Lippman, will serve as special lecturers.

NORTHFIELD CITY HOSPITAL

On January 5, 1955, the Northfield City Hospital Staff elected the following officers for the year 1955: Chief of Staff, George N. Rysgaard, M.D.; Vice President, Robert F. Mears, M.D.; Secretary-Treasurer, Donald Peterson, M.D.

The Staff meets once a month for business and a scientific program, and once a month for a pathological conference conducted by Dr. John I. Coe, pathologist.

CONTINUATION COURSES

A continuation course in *Emergency Surgery for General Physicians* will be presented by the University of Minnesota, March 31 to April 2, at the Center for Continuation Study. The management of injuries and other acute surgical problems will be taken up in detail. The program will be presented under the direction of Dr. Owen H. Wangensteen, professor and chairman, Department of Surgery.

* * *

The University of Minnesota and the Minnesota Heart Association will join in sponsoring a continuation course in *Cardiovascular Diseases for General Physicians* which will be held at the Center for Continuation Study from March 21 to 23. The common cardiovascular disorders will be discussed and treatment will be emphasized. The course will be presented under the direction of Dr. C. J. Watson, professor and head, Department of Medicine.

* * *

A continuation course in *Gynecology for General Physicians* will be presented by the University of Minnesota at the Center for Continuation Study from April 14 to 16, 1955. This year's course, which will be presented under the direction of Dr. John L. McKelvey, Professor and Head, Department of Obstetrics and Gynecology, will deal with the general problem of vaginal bleeding. The various causes of vaginal bleeding will be discussed from the standpoint of diagnosis and management.

* * *

The University of Minnesota will present a continuation course in *Radiology for General Physicians* from April 11 to 13, 1955, under the direction of Dr. Leo G. Rigler, Professor and Head, Department of Radiology.

Registrants for the course will spend most of their time in the University Hospital Department of Radiology reading films and carrying out procedures in association with members of the faculty of that department. A minimum of didactic material will be presented. Because of the nature of this course, registration will be strictly limited. Early application is therefore suggested. Accommodations will be available in the Center for Continuation Study as usual.

* * *

The University of Minnesota announces a continuation course in *Allergy and Chest Diseases for General Physicians* to be held at the Center for Continuation Study from April 18 to 20, 1955. Special emphasis will be placed on the management of commonly-met problems in these fields. Skin testing methods will be demonstrated and discussed. The program will be presented under the direction of Dr. C. J. Watson, Professor and Head, Department of Medicine.

LICENSURE EXAMINATION

An examination for medical licensure will be held by the Minnesota State Board of Medical Examiners beginning on Tuesday, June 14, 1955, continuing on Wednesday and concluding with a practical examination on Thursday, June 16, 1955. Applications for such examination, accompanied by required credentials and fees, should be on file thirty days prior to the examination at the headquarters office, 230 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota.

RAMSEY AUXILIARY REPORTS ACTIVITIES

Mrs. L. T. Simons

The Woman's Auxiliary to the Ramsey County Medical Society resumed its activities after a short holiday rescess. On January 17, a Board meeting was held in the Medical Library, with Mrs. W. P. Gardner, Saint Paul, president, presiding. Members made cancer dressings during and following the meeting.

On January 24, a luncheon meeting was held at the Athletic Club. The program consisted of a showing of a rehabilitation film. Winners of the TB Essay Contest read their prize-winning essays at the luncheon.

A delegation of Ramsey County auxiliary members attended the annual Mid-Winter Board meeting of the Woman's Auxiliary to the Minnesota State Medical Association, held on February 10 at the Town and Country Club. Officers and state committee chairmen presented reports, and members enjoyed a pleasant luncheon.

In Memoriam

DENNIS MICHAEL O'DONNELL

(Continued from Page 133)

He was a member of the West Central Minnesota Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Dr. O'Donnell is survived by his wife, Florence, and one daughter, Kathleen.

LEON GRANT SMITH

Dr. Leon G. Smith, prominent Montevideo physician and long-time councilor active in the Minnesota State Medical Association, died of a heart ailment at his home in Montevideo, January 13, 1955. He was sixty-seven years old.

Dr. Smith was elected Third District councilor to the Minnesota State Medical Association in 1947, serving through 1954, when he retired because of ill health. He has been active and well known in the western part of the state for many years for his activities in medical organization work. He served as president of the Camp Release District Medical Society in 1925 and 1926, having served on various committees also. For many years he was a member of Chippewa County's Advisory Committee to the Division of Social Welfare, and was also a member of various other state and county committees.

Dr. Smith was born on September 27, 1887, at Benson, where he obtained his preliminary and high school education. He received a B. S. degree from the University of Minnesota in 1909 and graduated in medicine in 1910. He then served a one-year internship at Minneapolis City (now General) Hospital.

He served in the Army Medical Corps during World War I and took several postgraduate short courses at Dyon, France, during this time. Dr. Smith has practiced at Montevideo since 1912, except for his war service and two years of practice at Buffalo, Minnesota.

He was a member of the Camp Release District Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Dr. Smith was a staff member of the Montevideo Hospital and served for many years on the local Board of Education.

He is survived by his wife, Edith; four children, William G., Greenwich, Conn.; Mrs. Paul Larson (Margaret), Sacramento, Calif.; Eugene A. and Leon Gordon, both of Montevideo; one sister, Mrs. Grace Osborne, Minneapolis, and fifteen grandchildren. His parents and one sister preceded him in death.

Of General Interest

Dr. and Mrs. Titus C. Kreuzer of Marshall, Minnesota, are on an extended European vacation. They will fly to Lisbon, Portugal, then tour Spain, before settling down for a month at Palma de Mallorca.

Before returning home in April, the Kreuzers also plan to visit France.

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Dr. H. L. Williams, of the Eye, Nose and Throat Department, Mayo Clinic, Rochester, is scheduled to present a paper on "Ménière's Disease" at the annual convention of the American Medical Association in Atlantic City, New Jersey, which will convene June 6-10, 1955.

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Dr. Merrill D. Chesler, Minneapolis, spoke on "Traumatic Deformities and Their Reconstruction" at a luncheon meeting of the Junior Section of the Hennepin County Bar Association in Minneapolis, January 28, 1955. The talk was illustrated with slides in color.

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Dr. Della Drips, Oronoco, is the Medical Woman of the Year for Branch Twenty-six, of the American Medical Women's Association, for Minnesota. "Her pioneer investigations in the field of hormones; her study of gynecologic endocrinology in Berlin; the development of the first laboratory in this field at the Mayo Clinic; her authoritative knowledge of the subject; and her readiness and ability to teach, has resulted in many warm friends among those who have been privileged to know and work with her."

Dr. Drips retired from the active practice of medicine and her specialty of gynecology at the Mayo Clinic in 1949.

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The January issue of *The Journal of the American Medical Women's Association* is the special number of Branch Twenty-six, Minnesota, with **Dr. Nellie N. Barsness,** Saint Paul, as guest editor.

Articles included were "Anoxia and the Surgical Patient," **Dr. Margaret C. Anderson,** Mankato; "Primary Amenorrhea," **Dr. Della G. Drips,** Oronoco; "Cerebral Vascular Accidents," **Dr. Marguerite Schwyzer,** Saint Paul; and a special article, "Alaskan Interlude," by **Dr. Catherine Burns,** Albert Lea.

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Dr. David M. Potek has opened offices for general practice at 3657 Emerson Avenue North in Minneapolis following completion of a fellowship at Glen Lake Sanatorium, which he began in January, 1952. Dr. Potek was engaged in practice in International Falls from 1936 to 1940. He served in the Army from 1940 to October, 1945, when he returned to International Falls for a six-year period.

Four physicians and surgeons who recently completed fellowships in the Mayo Foundation have been appointed to the staff of the Mayo Clinic. They are **Drs. Richard Reitemeier, Norman G. Hepper** and **Paul A. Green,** all in general medicine, and **Dr. C. Roger Sullivan,** who will be in orthopedic surgery.

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Dr. Gordon R. Kamman of Saint Paul spoke at the January 11 meeting of the Cerro-Gordo County Medical Society in Mason City, Iowa. Dr. Kamman's topic was "Psychometrics in General Practice." About fifty society members were present.

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Dr. and Mrs. Gilbert Wheeler were honored by the Roseau Lions Club recently. The Wheelers, who have resided in Roseau for several years, are moving to Winnipeg, where Dr. Wheeler will take a four-year course in radiology.

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Recently appointed county jail and work farm physician is **Dr. John V. Thomas** of Duluth. He succeeds **Dr. Cyril Smith,** coroner-elect and former jail physician, and **Dr. J. W. Ekblad,** Duluth, retiring coroner and workhouse physician.

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Dr. Leo A. Nash of St. Paul attended the fortieth annual meeting of the Radiological Society of North America in Los Angeles, California.

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A Clinton man, **Lt. Paul A. Williams,** was one of eighty-three Air Force physicians to receive diplomas recently from the School of Aviation Medicine at Randolph Air Force Base, Texas. He is presently assigned to the USAF infirmary at the Harlingen Air Force Base, Texas.

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President of the staff at Murray County Memorial Hospital for 1955 is **Dr. Hugh D. Patterson,** Slayton. Vice President is **Dr. Thomas Harada** of Lake Wilson, and secretary is **Dr. D. D. Nywall** of Slayton.

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Dr. S. F. Haines has been re-elected chairman of the Mayo Clinic board of governors. **Dr. J. T. Priestley** will be vice chairman; **J. W. Harwick,** secretary; and **Dr. H. R. Butt,** second vice chairman.

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New president of the Blue Earth County Medical Society is **Dr. J. J. Eustermann** of Mankato. He succeeds **Dr. R. W. Kearney.** Other new officers include **Dr. William S. Chalgren,** vice president, and **Dr. George R. McNear, Jr.,** secretary-treasurer. The election was held at the December annual meeting. Delegates to the Minnesota State Medical Association will be **Dr. Orville Jones** and Dr. Chalgren.

Dr. Philip R. Heise of Winona was elected president of the Winona County Medical Society at the group's quarterly meeting last month. Other officers named for one-year terms are: **Dr. W. O. Finkelnburg,** Winona, vice president; **Dr. L. J. Wilson,** Winona, secretary, and **Dr. Pat Rollins,** St. Charles, treasurer.

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Dr. Chester E. Thiem, Mankato, has been elected to serve another term as president of the Immanuel Hospital medical staff. Others elected to positions on the Mankato hospital staff are **Dr. H. O. Morgan,** Amboy, and **Dr. B. Niles Batdorf** of Good Thunder. Re-elected to the executive committee were **Dr. James Anderson** and **Dr. Robert Engstrom,** both of Mankato, with **Dr. Mervin W. Dobson,** Mankato, as new committee member.

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At a regular monthly meeting of the staff of St. Michael's Hospital, Sauk Centre, **Dr. A. B. Nietfeld** was elected president of the staff. **Dr. J. F. DuBois, Jr.,** will be vice president, and **Dr. F. H. Baumgartner,** Albany, will be secretary.

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New president of the Red River Valley Medical Society, succeeding **Dr. Alan McKaig** of Red Lake Falls, is **Dr. Harold Thysell,** Crookston. **Dr. Byron R. Kinkade** of Ada was elected vice president and **Dr. R. O. Sather** was named secretary-treasurer. Guest speaker at the meeting was **Dr. James Garvey** of Minneapolis, who talked on emotional disturbances of children.

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Dr. Roger W. Ridley, a member of the Mayo Clinic staff since 1948, has resigned to enter private practice in Riverside, Calif. Dr. Ridley has been a consultant in the section of anesthesiology and intravenous therapy at the Clinic and an instructor in anesthesiology in the Mayo Foundation since 1948.

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Dr. Stanley S. Chunn of Willmar, formerly associated with the Lakeland Medical Center in Willmar, is now engaged in private practice. Dr. Chunn specializes in eye, ear, nose and throat.

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Dr. K. A. March of Cambridge, former chief of staff at the Community Hospital, has moved to San Diego, where he will take up his practice.

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Dr. Samuel Miller, former radiologist at the Naeve Hospital in Albert Lea and in hospitals at Wells and Blue Earth, has joined the DePuy-Sorkness medical clinic in Jamestown, North Dakota. Dr. Miller, who once practiced general medicine in Jamestown, will specialize in radiology.

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Dr. Maxwell Barr was elected chief of Maternity Hospital, Minneapolis, at the hospital's annual medical staff dinner meeting on January 18. He succeeds Dr. Milton Abramson in the office. Other officers include **Dr. Marie Moorhead,** vice president; **Dr. Francis Swain,** secretary, and **Dr. Sidney Scherling,** treasurer. Chief speaker at the meeting was Dr. John Randall, professor and chief of obstetrics and gynecology at the University of Iowa.

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After completing a three-year residency in obstetrics and gynecology at the Mayo Clinic, Rochester, **Dr. Walter Eisenman** has moved to Hibbing, where he is associated with the Mesabi Clinic. He was formerly with the clinic for four years, practicing in Coleraine.

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Copies of a book written by the late **Dr. J. A. Sanford** are now available at the Sanford Hospital, Farmington. The book, entitled, "A General Practitioner Speaks," was written by Dr. Sanford over a number of years and deals with the subject of general practice. Dr. Sanford died on November 15, 1954, while doing the final proofreading for the volume.

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At a meeting of the Mahnomen County board of commissioners in January, **Dr. Kenneth W. Covey,** Mahnomen, was named as a member of the county board of health.

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Dr. W. H. Valentine, Tracy, president of the county tuberculosis and health association, announced that a tuberculin testing survey was carried out at schools in Marshall from January 24 through February 4.

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The board of commissioners of Mille Lacs County has elected **Dr. W. R. Blomberg,** Princeton, a member of the county board of health.

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Dr. Bernard J. Spencer, Minneapolis, has been elected a diplomate of the American Board of Surgery. A graduate of the University of Minnesota Medical School, Dr. Spencer spent two years as assistant surgical resident at Boston Children's Hospital, Boston, and three years as a surgical fellow at the Mayo Clinic, Rochester. He is now practicing pediatric surgery in Minneapolis.

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The commissioners of Stearns County have appointed **Dr. John B. Beuning,** St. Cloud, a member of the county board of health. **Dr. Charles F. Brigham, Jr.,** St. Cloud, has been appointed Stearns County physician.

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Dr. S. W. Giere, Benson, showed motion pictures he had taken in the Scandinavian countries at a public gathering in the Benson high school on January 22.

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Dr. M. Duane Sommerness, clinical director at Fergus Falls State Hospital, was named temporary acting superintendent of Hastings State Hospital in January. He was to serve while Dr. Jorge Lazarte, clinical director at Rochester State Hospital and acting superintendent at Hastings, was on a vacation trip to South America. Dr. Dale Cameron, medical director of the Department of Public Welfare, said that Dr. Lazarte would return to his part-time post at Hastings after his vacation unless a full-time superintendent had been found in the meantime.

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The new West St. Paul Medical Center was opened for the practice of medicine on January 3. The center is staffed by **Dr. J. A. Malerich, Sr., Dr. J. A. Malerich, Jr., Dr. Joseph P. Connolly,** and a dentist, Dr. H. E. Schad.

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Dr. Robert P. Koenig, Paynesville, has reported for active duty in the Air Force. Dr. Koenig, who has been associated with the Myre Clinic at Paynesville for the past two years, expects to be stationed at the Air Force Hospital at Geneva, New York.

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Dr. Paul A. Robertson, Austin, gave a talk on health at a meeting of the Woodson 4-H Club early in January.

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Dr. George Haggard, Minneapolis, celebrated his ninety-eighth birthday on January 18.

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Dr. Charles Mayo, Rochester, is one of forty-one distinguished Americans named to a lifetime appointment on the college of electors of the Hall of Fame for Great Americans. The members elect candidates to the Hall of Fame, which is located on the Bronx campus of New York University.

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Dr. H. P. Van Cleve, Austin, attended a one-week postgraduate course in dermatology at New York University in mid-January, following which he vacationed in Florida for several weeks.

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Dr. Milton M. Howell, Glencoe, has purchased a half-interest in the Glencoe Clinic. Dr. Howell has been associated in the clinic with **Dr. C. W. Truesdale** for the past year.

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Dr. D. H. Bessesen, Olivia, has been elected chief of staff of the Renville County Hospital. **Dr. J. A. Cosgriff, Sr.,** Olivia, was elected vice chief of staff, and **Dr. Donald Metz,** Buffalo Lake, was named secretary-treasurer.

NEW LOCATIONS

Dr. Robert J. Cesnik, who has been associated with Dr. L. M. Evans for the past five years, has entered the Army. He will be stationed at Fort Sam Houston, Texas.

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Dr. James Nixon, who has been in the Navy about two years, has returned to his practice at the Cuyuna Range Clinic at Crosby. **Dr. R. A. Kallsen,** who has been associated with **Dr. C. M. Marshall** and **Dr. R. E. Kelley,** at the Clinic, has left for California to join his family.

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Dr. Donald R. McFarlane, who completed his internship at General Hospital in Minneapolis in July, has become an associate in the Oliver Clinic at Graceville. **Dr. C. J. Swendseen,** who formerly practiced at Alexandria, also is a new member of the Clinic.

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Dr. H. C. Habein, Jr., of Rochester, who was called into active service with the Air Force in November, is now with the Air Force Hospital in Wiesbaden, Germany. Capt. Habein is the son of **Dr. Harold C. Habein** of Rochester.

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Dr. William Furlow, formerly of Belle Plaine, has taken over the practice of the late Dr. R. W. Maertz in Faribault. Dr. Furlow is a native of Rochester.

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Dr. Stanley E. Roberts, a former Fellow in surgery at Mayo Clinic, is now associated in practice with **Dr. Paul F. Meyer** and **Dr. Robert P. Meyer** at Faribault.

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Dr. Bela Gallo is now located at Taconite Harbor after fifteen months at Lake Bronson. Dr. Gallo has accepted a position with the Erie Mining Company.

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Dr. William Hunt, obstetrician and gynecologist of the Mayo Clinic staff, has moved to Fergus Falls, where he is now associated with the Park Region Medical Center.

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Dr. John C. Mickelson has been granted a two-year leave of absence from the Mankato Clinic at Mankato. He began a residency in urology in Durham, North Carolina, January 1.

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Dr. James W. Halvorson, formerly of Goodhue, has purchased the practice of Dr. Martin G. Flom at Zumbrota. Dr. Flom has accepted a position on the staff of the Veterans Hospital, Fargo. Dr. Halvorson recently completed a tour of duty in the Navy.

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Dr. Kermit L. Stensgaard, formerly of Lafayette, California, is now associated with a clinic at Thief River Falls.

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It was announced in mid-January that **Dr. James S. Barr,** formerly of Wells, planned to open a practice at Elmore.

MINNESOTA BLUE SHIELD-BLUE CROSS

Blue Shield News

At its meeting in Miami, Florida, on November 30, 1954, the Board of Trustees of the American Medical Association approved the appointment of a commission to conduct a comprehensive survey of the different types of plans which provide people financial assistance in obtaining medical services. Interest in the matter was stimulated through the Second Conference on Management-and Union-Sponsored Health Centers, arranged by the American Medical Association, and held February 24, 1954, in Louisville, Kentucky.

On August 27, 1954, the Board of Trustees of the American Medical Association appointed a Task Force to consider the desirability of appointing a commission to survey the various types of medical care plans in effect. This Task Force in November, recommended to the Board of Trustees that it appoint such a commission for this purpose.

Among the types of plans which are likely to be studied by the commission are prepaid closed-panel plans, non-contributory closed-panel plans, non-contributory free-choice plans and industrial medical programs. Such plans include the Health Insurance Plan of Greater New York and the Kaiser-Permanente plans of California, as well as many others. In addition to these, indemnity insurance plans, prepaid full payment plans and combination indemnity service plans will undoubtedly be studied by the commission. Such plans include the various Blue Shield plans throughout the country.

It has been reported that the commission will have as its chairman, Dr. Leonard W. Larson of Bismarck, North Dakota, a trustee of the American Medical Association. Minnesota physicians will be interested in knowing that Dr. F. J. Elias, of Duluth, has been appointed a member of the commission. The only member of the commission who is not a physician is Jay Ketchum, executive vice president of Michigan Medical Service. Dr. Leo Price, director of the Union Health Center of New York, is the only physician member of the commission who is not an American Medical Association trustee, delegate or council member. Other members of the commission are Doctors David B. Allman, Homer L. Pearson, Jr., Joseph D. McCarthy, Percy Hopkins, H. Russell Brown, James R. Reuling, H. Gordon McLean, John Conway and William P. Sheppard.

Blue Cross News

Blue Cross, through twenty-five years, has given more people more benefits than all other types of hospitalization plans combined. The start of the Blue Cross plan was made possible at Baylor University Hospital for Dallas school teachers to protect themselves against the sudden cost of unexpected hospital bills. Each teacher paid a small amount monthly into a common fund and this fund paid the expenses of those who had to be hospitalized.

Out of this idea of people helping each other grew Blue Cross. It spread to communities across the nation. Minnesota Blue Cross, one of the pioneer prepaid hospitalization plans, was created in 1933 when there were few, if any, people in the state with any protection against hospital expenses. Since that time, just twenty-two years ago, Minnesota Blue Cross has enrolled over one million subscribers in Minnesota.

Today, throughout the nation, 47 million people are protected by Blue Cross. Blue Cross has grown because it has held fast to its ideal of public service. Its aim still is to help people secure the hospital care they need.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

VIRAL AND RICKETTSIAL DISEASES OF THE SKIN, EYE AND MUCOUS MEMBRANES OF MAN. Harvey Blank, M.D., Squibb Institute for Medical Research, Columbia University, University of Pennsylvania; and Geoffrey Rake, M.B., B.S., University of Pennsylvania, Wistar Institute for Anatomy and Biology, and Squibb Institute for Medical Research. 285 pages. Illus. Price $8.50, cloth. Boston: Little, Brown & Co., 1955.

PLANNING FLORIDA'S HEALTH LEADERSHIP—FLORIDA'S HOSPITALS AND NURSES. John M. MacLachlan, Ph.D. 122 pages. Illus. Price $1.50, paper cover. Gainesville, Florida: University of Florida Press, 1954.

DRUGS IN CURRENT USE 1955. Edited by Walter Modell, M.D., Associate Professor of Clinical Pharmacology, Cornell University Medical College. 148 pages. Price $2.00, flexible binding. New York: Springer Publishing Co., 1955.

HEALTH SUPERVISION OF YOUNG CHILDREN. 180 pages. Price $2.00, paper cover. New York: American Public Health Association, 1955.

The volume, "Health Supervision of Young Children," has been five years in preparation and is the product of collaboration by more than sixty prominent professional persons. It bears the endorsement of the American Academy of Pediatrics, U. S. Children's Bureau, National League for Nursing, and Association of State Maternal and Child Health and Crippled Children's Directors.

Latest available information about well infants and pre-school children is organized so as to be helpful to persons professionally concerned with child health, whether in private practice or in the fields of public health, welfare or education.

Part I, planned for physicians, nurses and other professional persons, is a guide to health supervision, incorporating newest techniques in parent counseling and health appraisal and care. It deals primarily with everyday problems in normal development but also gives consideration to the handling of less usual problems and abnormal conditions.

Part II, written especially for professional persons concerned with community health and welfare, is a guide to carrying out child health supervision through the child health conference, the "well-baby clinic" or "child health station."

This is the first of a series of manuals being prepared by the A.P.H.A. Committee on Child Health, under the chairmanship of Dr. Samuel M. Wishik of the University of Pittsburgh Graduate School of Public Health. The text was written by Nina Ridenour, Ph.D., staff consultant to the committee.

Original Contributions

Medical Aspects of Peptic Ulcer

JAMES MYHRE, M.D.
Minneapolis, Minnesota

PEPTIC ULCER is a sharply circumscribed loss of tissue resulting from the digestive action of acid gastric juice. It is clearly differentiated from an erosion in that peptic ulcer is a penetrating process involving the muscularis mucosa as well as mucosa.

It has been estimated from autopsy statistics that ten per cent of all individuals at some time or another in their lives have suffered from a chronic gastric or duodenal ulcer. The duodenal ulcer occurs about four to one in the male whereas gastric ulcer occurs three or four to one in the female. A further breakdown as to sex includes approximately only a two percent incidence of perforation of peptic ulcers in the female.

Peptic ulcer may occur in the lower esophagus, stomach, upper portion of the duodenum, in the small bowel when it is anastomosed to an acid-bearing section of the gastrointestinal tract, and very rarely in a Meckel's diverticulum in the jejunum or ileum. In the latter instance it occurs with ectopic gastric mucosa. The vast majority are in the first one to one and one-half inches of the duodenum or the duodenal bulb.

Etiology is a big subject but certain elementary things seem very definite and also pertinent as far as understanding the problem of treatment is concerned. It appears to be simply a digestion of tissue by the acid gastric juices. The old dictum of "No acid, no ulcer" seems incontestable. Peptic ulcer occurs only in those portions of the gastrointestinal tract exposed to the action of the acid gastric juice, and peptic ulcer does not occur in the complete absence of acid gastric juice. The psyche has been increasingly incriminated. There is at present a strong association of peptic ulcer with tension, nervous instability, fatigue and worry. Frequently the emotional aspects in etiology are lacking in gastric ulcers. The psychoanalyst finds strong dependent wishes which may be overt or not at all obvious in these people. In the group with hidden dependency these wishes are rejected by the patient's conscious personality and frequently overcompensated for in a capable energetic attainment of social or economic success. It has been said that a person could have a rousing business if he himself had a good ulcer and had twelve ulcer patients working for him. Harry Truman may have been nearer the core of things than he realized when he spoke of a three-ulcer man in a four-ulcer job. It has been said that hyperthyroidism originated from stress in the home or family and an ulcer originates from stress with external environment, e.g., a business or job. Wolf and Wolf in their patient, Tom, with a gastric fistula, observed hypersecretion during tense anxiety and noted then the transition from a mucosal erosion into an ulcer by the digestive action of acid gastric juice. During the Blitz in London there was a very definite increase in perforation of ulcers. Patients with chronic recurring ulcer distress frequently correlate an exacerbation with fatigue, stress, or tension.

The patient generally gives a history which is rather definite. He might describe the pain as burning, hurting, gnawing, annoying, or aching or just a dull distress or an awareness of discomfort. Sometimes the word "hunger pain" is used but actually hunger pain is an intermittent type of distress whereas ulcer distress is steady and continuous from fifteen minutes up to one or more hours. The presence of a pain in the back suggests chronic perforation. Chronicity of pain is generally characteristic for most patients with an

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Presented before the Minneapolis Academy of Medicine, April 19, 1954.

Dr. Myhre is clinical assistant professor of medicine in the University of Minnesota Medical School, Minneapolis, Minnesota.

ulcer for they experience their pain over a period of years. Histories of twenty to thirty years of distress are not uncommon but the common incidence is stated as being between five and eight years. Of course, one can find asymptomatic peptic ulcer or peptic ulcers of very brief duration as only a few days or few weeks. Generally one can bring out or the patient will spontaneously volunteer a daily rhythm to his distress. If the patient is given time and encouraged to give a "typical day," he will frequently describe the distress as coming on one to four hours after eating and he will get relief by eating, drinking milk, or taking soda. Vomiting and rest frequently help. Frequently he will describe waking with distress between midnight and two o'clock in the morning. This suggests pyloric obstruction and/or an acutely inflamed ulcer with a very high gastric secretion. The periodicity of ulcer distress is quite interesting and is classically stated as being in the spring and fall of the year. Many people do notice this periodicity but statistical studies do not bear this out except perhaps in farmers, for the greater distribution is more through the winter months. Periodicity may be correlated with infections, emotional stress, fatigue, or strain.

The final court of appeal is the x-ray or gastroscopic examination. Frequently there is a negative x-ray examination in a patient with a classical ulcer history. In this instance repeating the x-ray will frequently bring out the true state of affairs although there are errors depending on the efficiency of the x-ray examination, body type of the individual, and the difficulty in demonstrating posterior wall lesions. These people should be considered for gastroscopic examination.

When confronted with patients with gastrointestinal distress, part of the job is to decide which patients should have the benefit of x-ray examination. Obviously, the severity of the complaint is considered and whether or not it fits in with what is called classical ulcer distress. In the uncomplicated ulcer, it is quite unusual to have distress before breakfast, and noting this you may be much less inclined to feel an x-ray examination is necessary. The pain of an irritable bowel is much more likely to be intermittent and spread out over the abdomen, and classically the patient will obtain good relief with a good belch or particularly by flatus or a bowel movement. If the patient has hypertension, it is less likely that he has a peptic ulcer. Food relief is strongly suggestive, but soda relief is not entirely conclusive for it produces a belch which can cause relief of functional gaseous distress. Just nausea is uncommon with peptic ulceration, although it is very occasionally the only symptom in a gastric ulcer. A minor pearl is that epigastric distress which disappears with a dark stool is likely due to peptic ulcer. Peptic ulcer is rare in hypoparathyroidism and not uncommon with hyperparathyroidism. It is apparently rare with adrenocortical hyperactivity of the adrenogenital type. Ulcers frequently heal in the first part of pregnancy, and the general incidence in females goes up after menopause. On physical examination, the main finding is rather localized epigastric tenderness over the lesion. On laboratory examination, the gastric analysis is most helpful. "No acid, no ulcer" is most helpful in cataloguing the patient's distress. It is not infrequent, unfortunately, that a gastric analysis shows no free hydrochloric acid and on being repeated two or sometimes three times occasionally will show a plentiful supply of acid. Apparently the tip of the aspirating tube is in the esophagus or is passed into the duodenum. Blood in the stool by Guaiac test is, of course, suggestive of an ulcer with bleeding.

Relief of distress is generally easy. The patient hardly needs a doctor for this but just some advice from an ulcer friend or Aunt Nellie who has taken soda bicarbonate most of her life. Curing an actual ulcer crater is a bigger job, but the real challenge and the real job of the physician is healing the patient's tendency to recurrent ulcer formation or in other words healing a chronic disease. This generally involves changing his reactions to his environment.

Rest is perhaps the most important thing. This is best obtained in a hospital for from two to three weeks, and this is particularly true when the peptic ulcer is first discovered for it is here that the patient will get the best orientation which is so desirable at the onset in his probable chronic disease. It is in this new ulcer group that the best chance for a permanent healing of the disease is found. I have a friend who has a large number of patients apparently of a two to three packages of cigarettes, eight to ten highballs, and sixteen to eighteen hours a day type. They characteristically always have another $25,000 to $50,000 deal coming up the next day, and therefore continually put off concentrated treatment until the physician, who has considerable stature, has been known to go into their offices and take them by the arm to the hospital. There he pulls the shade, closes the door, orders that there be no telephone calls, no telegrams, no mail, and no visitors (probably particularly the patient's wife). Then with a minimal amount of medication and diet in a matter of a few days, the patient quickly becomes comfortable. Alleviating the chronic tension and fatigue of these people is of the utmost importance and is very nicely abetted by the cheap and practical phenobarbital in sufficient doses so that they can easily succumb to your good advice to never resist an urge to doze. Time should be taken to explain an ulcer to the patient, and this is most easily done with the patient in the hospital. You then can also further observe his reaction to himself, his business, his family, and his environment. I find it most helpful to sit down with a piece of paper and draw out the rough diagram of the stomach, duodenum, small bowel, and colon. Many patients have no appreciation of what the word ulcer means, and some understanding comes for the first time when you compare it to the collar sore on a horse, varicose ulcer on a lady's leg, or a running sore on a dog. Then you can very profitably carry it further by telling them that the acid in their stomach is just like acid in a cream station or acid from their car battery. A previously rather uninterested person becomes rather interested with this explanation and responds rather dramatically to the question, "What do you suppose would happen if battery acid or cream station acid were dropped in the collar sore of a horse for twenty-four hours a day?" He will then understand the value of rest when he realizes that tension and anxiety greatly increase the formation of acid. He will then understand that the frequent feedings are intended to neutralize the acid. He should, of course, entirely and forever quit smoking and drinking. This is not easy to do, of course, but it is best done in the hospital with the help of some sedation. I oversimplify it, but get the point across by telling them about the gastric analysis. I explain that if their gastric analysis showed thirty degrees and the tube were left in place and he were to smoke a cigarette, then the acid if it were aspirated again might show sixty degrees of acid. This makes sense to the patient and makes it much easier for him to quit smoking. Of course, the only way is to quit completely and not to "taper off." Spending this amount of time with the patient convinces him that you are interested, and your interest and enthusiasm are an exceedingly potent weapon in the therapeutic program.

The classical Sippy type of diet has its dating back to antiquity but was organized by Sippy and has very well stood the test of time. Ulcer treatments and ulcer drugs come and go many times a year, and the three to four ounces or half glass of milk and cream stands the test of time. This should be given with regularity on the hour from seven in the morning until seven at night. The half glass is important as it allows physiologic stomach rest, and this, too, should be explained to the patient inasmuch as large feedings cause distention and increased secretions. It is unnecessary in most instances to add antacid. It is expensive and just the milk alone is sufficient with the Sippy diet program of gradually increasing foods like poached or boiled egg, cooked cereal, toast, pureed fruits and vegetables, rice, jello, custard, chicken, and ground beef. In order to impress the patient of the necessity for this diet, you must know about it yourself and be convinced about it. The "three bland feedings a day" has no place in the treatment of peptic ulcer. The drug companies have flooded the market with multiple antacids and minor insignificant variations of them all. When you explain to the patient, as you should, that his ulcer treatment may take anywhere from six months to two years and then figure out what his cost would be for antacids, you will realize that it is not economically desirable. If you elect to add one of the aluminum hydroxide preparations as an antacid, it is probably best done on the half hour from 7:30 in the morning until 7.30 at night. A multiple vitamin preparation should be included on a Sippy diet.

The antisecretory drugs are the great hope in present peptic ulcer investigation and the emphasis in research has swung away from antacids to this type of medication. Banthine is the original and best known of all the drugs, and at the present is preferred because it is so well developed. It is hoped that some of the many brother and sister preparations will have less of the side effects of dryness of the mouth, blurring of vision, and slowing of micturition and above all not to be so variable and unpredictable in their effect. Our standard procedure is to give one hundred milligrams of Banthine at eight p.m., two a.m., eight a.m., and two p.m. or every six hours around the clock. We have the patient set his alarm clock at two a.m. to do this. At the present time, the antisecretory drugs must just be considered an adjunct to the rest and dietary program. It is rather disconcerting to see some of the sales propaganda encouraging the use of antisecretory drugs alone with three bland meals.

During the daily hospital visits, the various features of ulcer management are discussed with the patient, and he is encouraged to ask questions. The complications of hemorrhage in the form of tarry stools or coffee-ground vomitus should be mentioned. The symptoms of perforation should be impressed on the patient, and the necessity of getting to a physician early should be stressed. Pyloric obstruction should be mentioned. After the orientation in the hospital, the patient should have well in his mind that relief of his distress is easy and, of course, no indication whatever of degree of healing. He should also understand that complete healing is a slow process, and in our patients we do not even bother in the case of a duodenal ulcer to repeat the x-ray for evidence of healing any sooner than three months. The duodenal ulcers heal slowly, and the patients also are a little inclined to relax if there is no x-ray evidence of a crater. It is impossible to know when the ulcer crater has been completely filled in, and the patient should understand that his efforts and your efforts should be toward the healing of the disease, and this is a six-months-to-a-year program. You should discuss with him things that aggravate him and discuss with him getting full nights of rest, straightening things out on his job, getting a nap over the weekend, getting outdoors every day, and getting a regular vacation. For many years or perhaps his whole life, he should plan on a glass of milk in midmorning and midafternoon. We keep these patients in the hospital routinely for two weeks and then keep them out of work if possible one or two weeks more after which time they have then progressed from the hourly milk feedings of the first twenty-eight days of the Sippy diet and are then ready for the convalescent ulcer diet which requires a half glass of milk or milk and cream twice in the morning and twice in the afternoon at approximately an hour's interval. After hospital discharge we see the patient at two-to-four-week intervals in the office and carefully review his diet, his rest program, and his medications. Frequently the Banthine is continued up to two to six months, and sometimes we substitute tincture of belladonna in the latter part of this period. Tincture of belladonna should be begun at approximately fifteen drops three times a day before meals and at bedtime and increased two drops per day until the patient notices some visual symptoms. This might be anywhere from twenty-five to thirty to forty drops four times daily. When the patient notices symptoms referable to his eyes, then he should decrease the dose two drops and maintain it at this level. You should reassure the patient that if he has a duodenal ulcer he has no fears of cancer, and you should also explain to him that many a greater or lesser man, including laborers or business executives, take their thermos bottles with them to work and take milk as you outline for them. A good plan is to have them take with them on trips or carry in a pocket some of the antacid tablets such as Amphojel or Gelusil which they can handily chew as a substitute for their between-meal milk feedings.

I have borrowed liberally from many sources, especially from Walter Lincoln Palmer, M.D.

Discussion

Dr. Herbert Plass: This is a good paper. It is comforting to reflect that this conservative method of managing peptic ulcer continues to be in vogue in the best circles. However, it makes it difficult to discuss the paper because I cannot disagree with it very much. On one point I should like to add an opinion and it is that many times superficial mucosal erosions are all the evidence which can be found radiologically of ulcer in a patient with clear-cut ulcer type story extending over a period of years. This may be the matter of timing of the x-ray examination in that a real crater might be found at the site of the mucosal erosion if the picture had been taken a week or two later without treatment. Of course I am not referring to gastritis of the common variety.

A second point is that it sometimes is fun to try and predict the type of occupation of an individual who comes complaining of ulcer-like distress. I have a feeling that by and large the ulcer-bearing individual prefers to work for someone else and to report to someone else, and that he is more efficient in doing so than if he has to be the top man. This certainly is not universally true, but I think that the hypertensive type of personality is more likely to be top man, as he is also more likely to be a good salesman.

I would like to emphasize that it is not fair to the radiologist to place on his examination the burden of "ruling out" or excluding ulcer. I do not know what number or percentage of ulcers are not demonstrated by x-ray but should guess that it may be in the neighborhood of five per cent, and in the hands of less skilled radiologists perhaps it is higher than this. One certainly cannot eliminate ulcer, for instance, as a cause of gastrointestinal hemorrhage by x-ray examination. Another point which interests me is that a considerable number of patients who have ulcer, easily demonstrated by x-ray or by the presence of one of the complications of ulcer, have been quite asymptomatic over a long period of time. I believe these people come into a sort of uneasy adjustment with their ulcers, and the indigestion they suffer is so much a part of themselves that they don't regard it as a complaint. Sometimes I can find out about these by asking specifically if anti-acid medications are used or carried daily in the pocket.

I heartily subscribe to Dr. Myhre's emphasis on rest as the primary agent in treating ulcers. I am sure nothing else is more important. Finally, I should like to ask a question, "Why do so many ulcer patients interrupt treatment, discontinue their anti-acids and antispasmodics when they have been told to continue them, and blandly turn up a month later having the same kind of distress and not knowing what to do about it?"

Dr. John Rosenow: I have been asked, as a matter of general interest for those of the audience not having direct contact with the problem of peptic ulcer, to say a few words to summarize some of the related surgical considerations.

At the outset I should like to make a distinction between gastric and duodenal ulcer. The accepted surgical procedures when done for gastric ulcer tend to be easier technically and to give better symptomatic results; the mortality is less, and there are fewer postoperative complications. The complication of anastomotic ulcer is rare following resection for gastric ulcer; this is not the case with duodenal ulcer.

The one great distinction between the two types of ulcer that must influence one's thinking in regard to gastric, as opposed to duodenal, ulcer is the question of malignant potentiality. Malignancy in duodenal ulcer is extremely rare. On the other hand, a fair summary of the experience in the literature, I think, would be to say that ten per cent of apparently benign gastric ulcers are actually malignant. I came across an interesting statistical comparison of the surgical versus the medical treatment of gastric ulcer in the new book by Moore and Harkins (Moore, H. G., Jr. and Harkins, N. H.: The Billroth I Gastric Resection. Boston: Little, Brown and Co., 1954). This contains statements that seem surprising but actually represent a reasonable summation of the figures appearing in the literature. Let us take two groups of one hundred cases each. One group will be treated medically, and the other will be treated surgically. First the surgical group—six patients will have carcinoma and six of them will die; two of the total series will die from operation, and nine patients will have disabling postgastrectomy symptoms. Thus a total of seventeen of the surgical patients will not be benefited (including eight deaths) and eighty-three will be benefited. In the medical group, all ten of the carcinoma patients will die. Twenty of the rest will have difficulty of a serious nature in the form of intractability, hemorrhage, perforation or obstruction, and about two of these twenty will die, so a total of thirty of the medical patients will not be benefited (including twelve deaths) and seventy will be benefited.

In considering the general indications for surgery in peptic ulcer, bear in mind that the following indications apply equally well to both gastric and duodenal ulcers, over and above the special consideration of malignant potentiality in those of the gastric variety. On the question of *perforation* there is pretty widespread agreement that the treatment of choice is simple closure of the perforation. However, in recent years there is a good deal of writing being done in this country advocating resection in these people; most men are hesitant to accept this. I think that perhaps in the patient who has been seen early, who is in excellent general condition, and who has had serious trouble with an ulcer for a long time in the past (and therefore might be expected to have difficulty in the future), resection should be considered. *Obstruction* is another generally accepted indication for surgery, almost always resection. But I would like to interpose here a word of caution. All apparent obstruction is not true obstruction, and the patient should have the benefit of hospital care of the medical variety—thorough and with the attention to detail so ably brought out here tonight by Dr. Myhre. In many cases the obstruction will be on the basis of spasm or edema, and these will relent under proper medical care.

As regards *hemorrhage*, there is some difference of opinion, there being all gradations of opinion from those who say all massive bleeders should be operated upon at once, to those who say that practically no one should ever be operated upon for hemorrhage. A few points deserve emphasis. First, bear in mind that the indication is massive hemorrhage, not just hemorrhage. The vast majority of hemorrhages will cease under adequate medical management. The surgical literature is full of work on massive hemorrhage, and I think that the things that are being written pertain to all types and degrees of hemorrhage. The decision as to whether to operate or not must be individualized. Age enters into it, certainly. Perhaps in the older patient who cannot be kept stabilized with 500 cc. of blood every six to eight hours, surgery should be the treatment of choice. In one's thinking on this matter, it is very easy (and I have heard this line of reasoning more than once) to say—"Well, the mortality for resection is only two to three per cent." In the face of massive hemorrhage, the mortality is not two to three per cent but more likely in the neighborhood of ten to fifteen per cent.

Our fourth usually accepted indication for surgery is *intractability*. And here there come about some of the greatest discrepancies in opinions over the country. I think we should remember that surgery for peptic ulcer, although occupying a very valuable place in our therapeutic armamentarium, is no glittering panacea. Gastric resection is, after all, when one really thinks about it, a crude, barbaric assault on the human body. And it is irrevocable. It is in this group of cases with so-called intractability that the proper, really thorough, medical treatment as outlined by Dr. Myhre can make a big difference. In the course of my training, I had a rather interesting chance to compare different ways of handling this problem.

At the hospital where I interned, a chief of one of the medical services had been associated with Dr. Sippy for years, and his thinking and influence affected the treatment of ulcer at this hospital quite strongly. The medical treatment of ulcer was extremely thorough and meticulous, and few patients came to surgery because of intractability alone. Then, when I entered upon my surgical graduate work at another institution, I had some work during that time in the medical sections. I was quite impressed in a not entirely agreeable way with the difference as to medical treatment of ulcer that was sometimes evident. The treatment of ulcer does not consist of giving the patient a diet form, chatting hastily with him as to the great importance of taking it easy, and giving him about ten prescriptions, and writing his doctor. I am sure that many patients over the country as a whole who are subjected to surgery on the basis of intractability would get by on medical treatment of the thorough type described to us tonight.

After all, there is about a five to ten per cent incidence of serious "dumping" of a disabling type after gastric resection, as well as other troubles. You'd rather not have the postgastrectomy patient coming back to you, and saying, "Ye gods, give me back my ulcer." In this connection, I think a rather forthright statement of a preceptor of mine at one stage in my training might well be quoted, namely, that for a patient to be advised to have surgery (in the absence of perforation, hemorrhage, or obstruction), he felt that the patient should have had "one h—l of a lot of trouble." My own personal feeling, had I a troublesome duodenal ulcer, without other complication, about having surgery would be about along those lines—that I'd have to have "one h—l of a lot of trouble."

Dr. Walter Ude: It has been a privilege to hear this splendid paper. The essayist is to be complimented. He has not only given us a complete analysis of the problem in hand, but has also outlined a program of management and therapy which indicates a wide personal knowledge and experience.

A careful and complete radiological examination of the upper gastrointestinal tract plays an important part in the study of patients suspected of ulcer. The fact that the radiological examination should not be the primary procedure to be placed ahead of a careful clinical study is self-evident, but is too often ignored. Certain limitations, which we cannot deny, make it impossible to always "rule in" or "rule out" an ulcer. For instance, we are limited in the extent of the examination in the presence of gross hemorrhage, or in the presence of marked debility from other causes. The case may be referred after a prolonged therapeutic management which may have considerably modified the findings. The ulcer may also be so located that it can be demonstrated only with great difficulty, often in a region where it is not accessible to palpation. A close co-ordination, therefore, of the clinician's findings and the radiologist's examination will yield far better results than the performance of the radiological examination as a routine procedure without direct guidance by the clinician. This brings us back to the age-old admonition that the request for the examination should bring with it this guiding information. Without it the radiologist attempts to get what information he can from the patient while he does the examination, a procedure which certainly cannot compete with the analysis of the clinician.

Permit me to discuss briefly difficulties encountered in four distinct regions of the upper portion of the gastrointestinal tract:

1. Hiatus hernia may be readily demonstrated in the routine examination, but at times is not so readily detected. In my experience, the shortened esophagus with hiatus hernia is best shown by studying the lower end of the esophagus in the prone oblique position during the ingestion of barium suspension through a drinking tube. This method is more successful than an attempt to demonstrate hernia in the Trendelenburg position. Ulcer in the herniated portion of the stomach may be obscured by the barium suspension, but often is clearly in evidence. It should always be kept in mind in cases of hiatus hernia.

2. The high, posterior wall of the stomach is a region where ulcer may be easily overlooked, especially in the sthenic individual.

3. Prepyloric ulcer, either on the posterior wall or on the lesser curvature, may be lost in prominent mucosal folds or obscured by peristaltic activity which may seem to obliterate a suspicious fleck of barium retention. Here, compression spot films with demonstration of mucosal pattern are of great help. If ulcers in this area fail to improve or even seem enlarged after therapeutic management they must be considered as questionably malignant, even though all other findings point toward benign ulcer.

4. Postbulbar ulcer of the duodenum is frequently the cause of silent bleeding from the upper gastrointestinal tract. Our pathologists have impressed us with the importance of finding these ulcers. Penetration directly into the pancreaticoduodenal artery may lead to fatal bleeding, and thus the pathologist will find the ulcer. The radiologist should be just as keenly interested in this region of the duodenum as he is in the duodenal cap. If there is persistent spasm and marked change in the mucosal pattern, a presumptive diagnosis of hidden postbulbar ulcer should be entertained as a working diagnosis, even though a definite crater cannot be found. Where such ulcers are of relatively short duration, the spastic contracture may completely hide the crater. Later

re-examination, often after only a few days, may visualize the crater.

We should not hesitate to re-examine a patient if there is the least doubt as to the findings, or if the clinician is not satisfied that the problem has been solved. We frequently recall a patient because of some suspicious finding on routine films, or the presence of hypersecretion or food retention in the stomach. We do not hesitate to examine in the presence of active bleeding, but avoid deep palpation or compression.

Let me again emphasize the necessity of a close co-ordination of the work of the clinician and the radiologist. Such co-operative effort yields dividends.

Dr. R. S. Ylvisaker: I enjoyed Doctor Myhre's paper very much. In my opinion his excellent summary indicates to us especially the main details which go into good medical care of the peptic ulcer patient. The importance of the subject of peptic ulcer is well known to all of us, and it is also indicated by the frequency with which many phases of this subject are covered in the annual meetings of the American Gastroenterological Association. Dr. Anton Carlson once remarked at one of these meetings that the meeting of the American Gastroenterological Association might be referred to as the "Annual Symposium on Peptic Ulcer."

I believe the main point that Doctor Myhre stressed in his paper was the need for a meticulous attention to detail in both the accurate diagnosis and the treatment of peptic ulcer. One is often discouraged with the laxness with which medical treatment is prescribed in this condition. In fact, many papers have appeared in the literature, even by men well respected in their fields who recommend more frequent surgical treatment because of failure of medical management when such management has been anything but careful. There is no question in my mind but that a good ninety-five per cent of peptic ulcers can be cured or well controlled by careful medical management carried out over a protracted period of time.

I would like to comment briefly on a few other points mentioned by Doctor Myhre:

1. The well-known dictum of no acid, no ulcer. This is undoubtedly the general rule and the safest guide to follow. In regard to gastric ulcer, any ulcer that appears in the presence of achlorhydria should be considered malignant until proved otherwise. However, I am sure that all of us have had the experience of occasionally finding a benign gastric ulcer even in the presence of a well established achlorhydria.

2. In regard to the incidence of peptic ulcer in hypertension, it is true that the usual case of peptic ulcer is apt to have a low to normal blood pressure and a slow pulse. However, I have recently had several cases of proven peptic ulcer come under my care with hypertension.

3. I cannot accept the position held by some of routinely advising surgery in gastric ulcer, chiefly for two reasons. The first is that by meticulous attention to detail and calling in every possible diagnostic aid, including gastroscopy in suspicious cases, I believe that the error of overlooking malignant ulcers can be dropped down well below the ten per cent mark. In the second place, I frankly do not believe that the results of surgery in cancer of the stomach are good enough to warrant submitting ninety per cent of the people to unnecessary surgery when perhaps only one out of ten of the malignant cases can be cured. Here again, each case must be studied individually and all factors evaluated before a choice of method of treatment is made.

4. In regard to gastric hemorrhage, I believe it is a mistake to routinely transfuse these cases on admission to the hospital. There is evidence to show that immediate transfusion increases the risk of continued bleeding, recurrent bleeding, and even increases the mortality rate. By far the majority of cases of gastric hemorrhage will subside promptly on complete bed rest. The crying need is for closer observation of each case and the use of transfusion only in those in which evidences of shock persist. Later after there is good evidence that bleeding has stopped, if the hemoglobin is low, then it is justifiable to transfuse in order to shorten the period of convalescence and overcome the severe anemia.

Dr. Jay C. Davis: I do not agree with Dr. Myhre that all patients with an ulcer should be hospitalized. There are some patients who can be healed while ambulatory. There are some who respond to treatment faster if they can get away on a vacation out of their own city and completely away from all disturbing factors that may exist in their family life or business.

Psychosomatic factors are very important and until the factors can be discovered and talked over with the patient one may not succeed in curing the patient.

Dr. Ylvisaker stated in his discussion that all peptic ulcer hemorrhages do not need surgery. It is important, however, to remember that slow bleeding over a period of many days may result in an anemia and drop in blood pressure severe enough to cause angina and changes in the electrocardiogram suggesting coronary insufficiency especially in an older patient with coronary sclerosis. A massive hemorrhage with a marked drop in blood pressure may result in such a marked coronary insufficiency that myocardial infarction may ensue without closure of a coronary artery. If the fall in blood pressure and the hemorrhage is massive, or if more than one severe hemorrhage occurs before surgery is resorted to the patient may suffer cerebral damage from anoxia. Degeneration of the kidney tubules may also occur with uremia resulting, or the patient may die in hepatic coma if a damaged liver existed at the time of the hemorrhage.

Why Milliequivalents?

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THE FOLLOWING presentation says nothing new or original. It is an attempt to answer the question posed by the title by giving in as simple a form as possible, some of the information gleaned from the literature about milliequivalents as a system of units. This information is too often found in journals not readily available, or in highly technical and complicated form.

What is a milliequivalent? The chemical potency of a substance in solution depends on the amount of it present by weight, the substance's atomic weight and the substance's valence. The comparative potency of the substances in solution can be expressed according to how much hydrogen ion they will neutralize or be equivalent to. The unit of measure in chemistry is the equivalent.

BASE (mEq/L)	
Na^+	142
K^+	5
Ca^{++}	5
Mg^{++}	3
	155

ACID	
HCO_3^-	27
Cl^-	103
HPO_4^{--}	2
SO_4^{--}	1
ORG.AC.	6
PROTEIN	16
	155

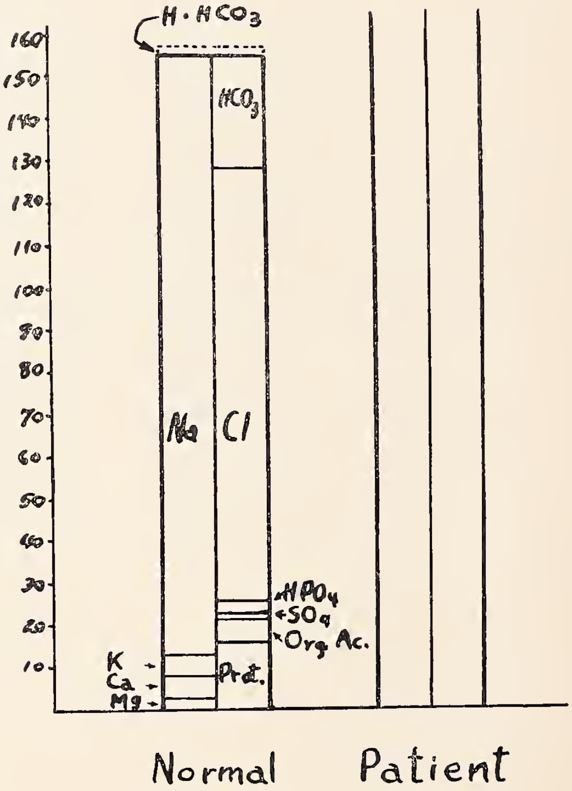

Fig. 1. Acid-base composition of blood plasma.[4]

Definitions

An electrolyte is a chemical substance, which when dissolved in a solution, separates or dissociates into electrically charged positive and negative particles, or atoms, called ions. The positive ions are called cations and the negative ions are called anions.

An equivalent is the weight of an ion, which in neutralization reactions, is equivalent to, or will neutralize one gram-atom of hydrogen. For example, one atom of chlorine will neutralize one atom of hydrogen since they are of the same valence, a valence of one. Therefore, since the weight of one atom of chlorine expressed in grams is 35.5, and that of hydrogen is one, then 35.5 grams of chlorine will neutralize or be equivalent

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Abridgement of inaugural thesis presented before the Minneapolis Academy of Medicine, March 16, 1953.

to one gram of hydrogen. If the amount of chlorine is double, then we have two equivalents of chlorine present, and so on. Again, for example, a single atom of calcium, having a valence of two, will combine with or neutralize two atoms of chlorine. Since the atomic weight of calcium is 40 and that of chlorine is 35.5, then 40 grams of calcium are equivalent to twice 35.5 or 71 grams of chlorine in a chemical solution. Equivalent=(grams x valence)÷atomic weight. Equivalents as units of measure are satisfactory for use in chemistry but not in medicine. In medicine, the amounts of ion dealt with are too small, so the milliequivalent is used, or one thousandth of an equivalent. Milliequivalents are expressed as units per liter, and are abbreviated "mEq" or "meq." Under normal conditions, there are 155 mEq of basic ions (cations), and 155 mEq of acid ions (anions) per liter of plasma. For practical purposes, the sum of the basic ions always equals the sum of the acid ions.

General Remarks

It would seem high time that we stop considering the various electrolytes as so much dead weight measured in ounces on the grocer's scales. We are dealing, in plasma, not with amounts by weight of chemical salts such as NaCl, KCl, and the like, but with separately controlled, independently reacting ions such as Na, K, Cl, and so on. The ions are active elements and we should evaluate them on their chemical activity. To do this and to understand the actual chemistry of fluid balance is to talk in terms of mEqs. It would be folly, for instance, to compare engines solely on the basis of their weight. A more valid and useful comparison is on the basis of their capacity for producing power, i.e., horsepower. One gram of chloride ions in plasma is roughly 100 times as powerful as one gram of protein when measured from the standpoint of chemical combining capacity, and 800 times as powerful osmotically. One gram of sodium in plasma is about 150 times as potent chemically and 1,200 times as potent osmotically as one gram of protein.

The rating of ions in the fluids of the body on the basis of such differing units as milligrams per 100 cc., grams per 100 cc., and volumes per cent, is perhaps like trying to work with fractions having different denominators. It would be impossible to add ⅓, ¼, and ⅛, let us say, if we did not have that very useful echo from grade-school days, the least common denominator. It then becomes simple to add 8/24, 6/24, and 3/24 to get the answer of 17/24. MEqs might be thought of as the least common denominators of fluid balance.

An example of this and of the differing worth of dead weight units and activity units (or mEqs) is readily found in a patient who loses 7.0 grams of chloride and 4.6 grams of sodium in tube drainage. It would be immediately apparent only to a chemist that loss of basic and acid ions is going on equally. If it were stated, as is actually the case, in this instance, that the patient lost 200 mEq each of chloride and sodium, the acid-base significance would be clear. It would also be clear that the relation of the two is high in chloride relative to extracellular fluid composition where the ratio is 142 of sodium to 103 of chloride. He will deplete his chlorides faster than his sodium. The loss of such a solution wholly from extracellular sources would thus lower chloride concentration differentially and produce metabolic acidosis despite the chemically neutral nature of the loss.

Examples of Use of Milliequivalents

Before embarking on the examples, let me call attention to the normal values in Figure 1. The first example is based on the fact that the sum of the basic ions equals the sum of the acid ions. Before the development of the flame photometric method of analysis of plasma, the only way to arrive at a rapid, rough estimate of the electrolyte concentrations, especially that of sodium, was by application of the equality of the acid and base ions, using other more easily obtainable values, such as for HCO_3 and Cl. And where a flame photometer is not available it still can serve, in spite of obvious shortcomings. For it to be valid, we must be able to assume that potassium is normal, and that the acid ions other than HCO_3 and Cl are present in normal amounts, often, of course, not the case.

(R^+ represents the sum of the basic ions other than sodium, and R^- represents the sum of the acid ions other than HCO_3 and Cl.)

$$\begin{array}{lcl}\text{sum of base ions} & = & \text{sum of acid ions}\\ \text{Na} + R^+ & = & HCO_3 + \text{Cl} + R^-\\ \text{Na} + 13 & = & HCO_3 + \text{Cl} + 25\end{array}$$

Now let us say that we have values for HCO_3 and Cl of 23 and 88 mEq/L respectively.

$$
\begin{aligned}
\text{Na} + 13 &= 23 + 88 + 25 \\
\text{Na} &= (23 + 88 + 25) - 13 \\
\text{Na} &= 136 - 13 = 123 \text{ mEq/L}
\end{aligned}
$$

The value for serum protein in mEq could of course be inserted or interpolated in the equation and would increase its accuracy somewhat. This method, while a thin reed to lean on, can be used if the exceptions and inaccuracies are kept in mind, with an occasional check by the older, time-consuming direct chemical determination of sodium.

Let's take up another oversimplified example. One could estimate roughly the amount of solution necessary to return the electrolyte concentration to normal. If we assume that the extracellular fluid volume is 20 per cent of the body weight (or body weight÷5), the following formula could be used as an approximate guide:

$$
\text{Total amount of ion needed} = \frac{\text{Pt's wt. in kg.}}{5} \times \left\{ \begin{matrix}\text{normal value}\\ \text{of ion in}\\ \text{mEq/L}\end{matrix} - \begin{matrix}\text{pt's level}\\ \text{of ion in}\\ \text{mEq/L}\end{matrix} \right\}
$$

Let's run through that with an example of a man weighing 175 pounds, who has a sodium of 132 mEq/L, remembering, of course, that to convert pounds to kilograms, divide by 2.2. His weight is thus 175÷2.2=80 kg.

$$
\text{Total needed} = \frac{80}{5} \times (142 - 132) = 16 \times 10 = 160 \text{ mEq}
$$

Physiological saline contains 154 mEq of sodium per liter, so one liter would give the approximate amount, theoretically, which would restore the sodium to normal.

Now for a more complicated example. Given a patient who has lost 3 liters of body fluids by suction drainage. Her normal weight is 114½ pounds (52 kilograms). She has the following values for electrolytes, which are converted using the factors found in Table I,

CO_2.......... 50.6 vol.% x 0.45 = 23 mEq/L
Cl (as NaCl) 550.0 mgm. per 100 cc. x 0.171 = 94 "
Protein....... 7.4 Gm. per 100 cc. x 2.43 = 18 "
Sodium .....298.0 mgm. per 100 cc. x 0.435 = 130 "

The patient's three liter loss of fluid, for the purposes of this example, is presumed to come from the extracellular fluids. So we first figure the normal amount of extracellular fluid for this patient and then subtract the loss,

$$
\begin{array}{rl}
52 \text{ kg} \div 5 = 10.4 & \text{liters, her normal extracellular fluid} \\
-\ 3.0 & \text{liters, loss} \\
\hline
7.4 & \text{liters, extracellular fluid after loss (a rather broad assumption)}
\end{array}
$$

$$
\begin{array}{lrr}
\text{Na}\begin{cases}\text{Normal} \\ \text{After loss}\end{cases} & \begin{matrix}10.4 \times 142 = \\ 7.4 \times 130 = -\end{matrix} & \begin{matrix}1477 \text{ mEq of Na}^1 \\ 962 \quad \text{"} \quad \text{"} \quad \text{"}^2\end{matrix} \\
 & & \overline{515 \quad \text{"} \quad \text{"} \quad \text{"}^3}
\end{array}
$$

[1] Total amount normally present
[2] Amount present after loss of 3 liters
[3] Of Na which have been lost

$$
\begin{array}{lrr}
\text{Cl}\begin{cases}\text{Normal} \\ \text{After loss}\end{cases} & \begin{matrix}10.4 \times 103 = \\ 7.4 \times 94 = -\end{matrix} & \begin{matrix}1071 \text{ mEq of Cl}^1 \\ 695 \quad \text{"} \quad \text{"} \quad \text{"}^2\end{matrix} \\
 & & \overline{376 \quad \text{"} \quad \text{"} \quad \text{"}^3}
\end{array}
$$

[1] Total amount normally present
[2] Amount present after loss of 3 liters
[3] Of Cl which have been lost

It remains to pick the repair solutions, which can be done without too much difficulty when we know the mEq content of the various available solutions (Table II).

As to chlorides—we see from the table that normal saline has 154 mEq/L of chloride, so to supply the deficit of 376 mEq, we need 376÷154 =2.4+liters. This amount of saline will supply us with the same amount of sodium, or 376 mEq. So we are left with 515—376=139 mEq of sodium to supply in some other way. Preferably we would like a solution having the sodium in combination with an acid ion that is easily metabolized, in order not to increase unduly the acid ions. Sixth molar sodium lactate is an excellent solution for this purpose. How much is needed? A liter of this solution, according to the table, contains 167 mEq of sodium, so we need 139÷167=832 cc. of sixth Molar sodium lactate.

For practical purposes, then, we would need about 2½ liters of saline and 800 cc. of sixth Molar sodium lactate. This would give us a little more total volume of fluid than we need. These calculations are for replacement of an existing immediate deficiency and do not provide for replacement of basic requirements such as losses through lungs, skin, obligatory volume needed by the kidneys and the like. The extra fluid we gave in our example could be reckoned in when those basic requirements are being calculated.

I cannot emphasize too strongly that the foregoing examples are purely for the purposes of illustration. They are not in any way advanced as specific guides in solving fluid-electrolyte problems. There are no pat, fool-proof rigid formulae that can be applied to all cases. But with the use of milliequivalents, at least such problems can be approached with some degree of intelligent understanding, and they may well lose some of their weird, cabalistic, mysterious aspects, and may become, while not always exactly simple, at least possible of rational solution.

TABLE I. NORMAL ELECTROLYTE VALUES—CONVERSION FACTORS

	Normal (mgm/100 ml)	×	Factor	=	Normal (mEq/L)
Na^+	317—340		0.435		137—148
K^+	16—21		0.257		4.1—5.4
Ca^{++}	9—11		0.5		4.5—5.5
Mg^{++}	1.2—2.4		0.82		1—2
HCO_3^- (CO_2 vol. %)	55—75	(or ÷2.22)	0.45		25—34
Cl^- (as NaCl)	585—645		0.171		100—110
(as Cl)	352—389		0.282		100—110
PO_4^{--}	3—5		0.58		1.7—2.9
SO_4^{--}	1.6—2.4		0.625		1.0—1.5
Protein (Gm. %)	6.5—8.0		2.43		16—19

$$\text{Concentration (mEq/L)} = \frac{\text{Concentration (mgm/100 ml)} \times 10}{\text{atomic weight}} \times \text{Valence}$$

$$\text{Conversion factor*} = \frac{10}{\text{atomic weight}} \times \text{Valence}$$

*does not apply to HCO_3, PO_4, and protein.

TABLE II. ELECTROLYTE CONTENT OF INFUSION FLUIDS (mEq/L)

Fluid	Na	K	Ca	Cl	Effective CO_2	NH_3	Mg	HPO_4
NaCl 0.9%	154.0			154.0				
Sodium bicarbonate 1.2%	143.0				143.0			
Ammonium chloride 0.75%				140.0		140.0		
KCl ampules (1.59 Gm.)		20.0		20.0				
Ringer's solution	147.0	4.0	4.5	155.5				
Butler's solution	30.7	20.8		23.2	20.5			7.8
Hartmann's solution	136.0	5.3	3.6	112.0	33.0			
Aminosol	6.1	0.053		17.0				
Amigen	30.0	15.0		42.0				
Travamine	110.0	5.6		65.0				
Plasma (250 cc.)	35.5	1.25	1.25	25.75	6.75		0.75	0.5
Whole blood (500 cc.)	40.0	2.0		16.0				
Polysal (Cutter)	140.0	10.0	5.0	103.0	55.0		3.0	
Mod. duod. solution (Baxter)	80.0	36.0	4.6	63.0	60.0		2.8	
Travert 10%—Electrolyte Solution No. 1 (Baxter)	80.0	36.0	4.6	63.0	60.0		2.8	
Travert 10%—Electrolyte Solution No. 2 (Baxter)	57.0	25.0		50.0	25.0		6.0	12.5
Travert 10%—Electrolyte Solution No. 3 (Baxter)	63.0	17.5		150.5		70.0		
Ammonium chloride 2.14% (Baxter)				400.0		400.0		
Darrow's solution (Baxter)	121.0	35.0		103.0	53.0			
M/6 sod. r-lactate (Baxter)	167.0				167.0			
Travert 10%—KCl 0.3% in water (Baxter)		40.0		40.0				
Travert 10%—KCl 0.3% in 0.45% NaCl (Baxter)	77.0	40.0		117.0				

Discussion

In the field of fluid-electrolyte balance, a vast amount of research is being done, and major advances and changes in concepts are taking place. The work is, and of necessity must be, done in terms of milliequivalents. The article in the literature on this subject which reports in other terms than milliequivalents is rare indeed. If we wish to have an intelligent understanding of what is being done, and want to put it to enlightened practical use, we should be familiar with the language in which it is written, so to speak.

For those of us who do not work exclusively in the so-called teaching institutions, the habit of thinking and working in terms of milliequivalents is made difficult to acquire by the fact that most hospital laboratories report electrolytes in the older system. It is suggested that the advent of the newer system might be hastened if hospital laboratories were asked routinely to report all electrolyte values in both systems for a time, with the eventual end in view of reporting only in milliequivalents. The conversions can be done far more easily in the laboratory, from prepared tables and graphs, without the necessity for actual calculations. Thought might be given to the provision in charts of a special electrolyte form, either separately or as part of the regular laboratory sheet; part could be a form for the reporting of the patient's values in both systems, and part could be a table of normal milliequivalent values.

It is unquestioned that in the ordinary case, all of this isn't too necessary. But the occasional complicated problem almost demands use of the milliequivalent system for intelligent handling.

Summary

It is hoped that the foregoing presentation may in some small measure help to answer the question posed by the title by bringing together from the literature some items of interest, edification, illustration and justification regarding the use of milliequivalents.

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REGULAR CORPS EXAMINATION FOR MEDICAL OFFICERS

A competitive examination for appointment of Medical Officers to the Regular Corps of the United States Public Health Service will be held in various places throughout the country on June 7, 8, and 9, 1955.

Appointments provide opportunities for career service in clinical medicine, research, and public health. They will be made in the ranks of Assistant and Senior Assistant, equivalent to Navy ranks of Lieutenant (j.g.) and Lieutenant, respectively.

Entrance pay for an Assistant Surgeon with dependents is $6,017 per annum; for Senior Assistant Surgeon with dependents, $6,918. Provisions are made for promotions at regular intervals.

Benefits include periodic pay increases, thirty days' annual leave, sick leave, medical care, disability retirement pay, retirement pay which is three-fourths of annual basic pay at time of retirement, and other privileges.

Active duty as a Public Health Service officer fulfills the obligation of Selective Service.

Requirements for both ranks are U. S. citizenship, age of at least twenty-one years, and graduation from a recognized school of medicine. For the rank of Assistant Surgeon, at least seven years of collegiate and professional training and appropriate experience are needed, and, for Senior Assistant Surgeon, at least ten years of collegiate and professional training and appropriate experience are needed.

Entrance examinations will include an oral interview, physical examination, and comprehensive objective examinations in the professional field.

Application forms may be obtained by writing to the Chief, Division of Personnel, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D. C. Completed application forms must be received in the Division of Personnel no later than May 6, 1955.

Clinical Aspects of Fluid and Electrolyte Management

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IN THE PRACTICAL day-to-day management of a patient on total, or even partial, artificial alimentation it would be ideal if one could order a uniform or standard intravenous feeding solution. However, in illness and stress every patient presents a variation that must be accurately dealt with.

Fluid, electrolyte, nitrogen and caloric needs require an assessment of basic needs, an estimate of deficits to be repeated and accurate information on losses that are occurring.

Adequate caloric and nitrogen intake is difficult to achieve by intravenous solutions unless all of the nutritional elements are utilized, and therefore the basic solution should contain 5 or 6 per cent amino acids to provide nitrogen in 10 to 15 per cent invert sugar or fructose to provide the carbohydrates, and 5 to 7.5 per cent alcohol to provide the additional calories which are ordinarily supplied by fat. Such a solution contains 1000 calories per liter and is capable of supplying at least 100 per cent of energy requirement without exceeding the fluid needs. All the fluid requirement of any but an anuric patient should be supplied in this form.

In the sick individual the fluid and electrolyte needs may vary greatly from the average normal. There is, therefore, no substitute for frequent, detailed analyses of the chemical levels and of careful calculation of balances determined from body fluid and excretion levels. With this information one can then more accurately order the volume of fluid and quantity of electrolytes as they are needed from day to day. The basic solution of amino acids and invert sugar or fructose must be one of low sodium, potassium and chloride content so that calculated quantities may be added to accommodate the requirement of the patient.

Such electrolyte additions may be easily made by injecting them into the basic solution from stock concentrates made up in sterile vials by a pharmacist. In this manner the physician can avoid ordering a liter of this and a liter of that—a method certain to be deficient in one or more aspects of nutrition.

At the outset a base line must be determined. Weight, hemoglobin, hematocrit, carbon dioxide, chloride, BUN, protein, urine specific gravity, and usually serum sodium and potassium are sufficient. The state of hydration and deviations from electrolyte equilibrium are then evident. If a deficit is present it is obvious that the balances attained on succeeding days must be on the positive side until repletion is achieved.

Fluid balance can be maintained only by careful attention to the following.

1. An accurate intake and output record must be kept. In this regard meticulous endeavor by the nursing staff is indispensable. An inlying catheter may be required in seriously ill or incontinent patients. Nasal suction should always be collected into a trap bottle.

2. Calculation or estimation of insensible water loss. The Darrow formula (adult) of 44 cc. insensible water loss for each 100 calories metabolized is quite easily calculated from the DuBois chart of the Lilly Co. slide rule for diabetic diets. Regardless of the caloric intake, an individual must satisfy his energy demands. From height, age, weight and sex factors on the DuBois scale one can find the caloric requirement. For normal bed activity and specific dynamic action the factor of basal calories plus 40 per cent is employed. To this factor 8 per cent is added for each degree of

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fever. The wide variance in insensible fluid loss is demonstrated by the following examples.

(A) A 6′, 200-pound man, thirty-five years old, with temperature of 102.6°
Basal = 2000 calories
Bed activity) + 40% = 800 calories
and SDA)
3° fever × 8 =)
+ 24% ¼ of 2000) = 500 calories
3300 calories requirement
33 × 44 = 1452 cc. insensible water loss per 24 hours.

(B) 5′ 2″ 100 pound woman, thirty-five years of age, with normal temperature
Basal = 1240 calories
Bed activity) + 40% = 500 calories
+ SDA)
17.4 × 44 = 766 cc. insensible water loss.

TABLE I. BLOOD ELECTROLYTES IN PATIENT DEVELOPING HYPOCHLOREMIC SHOCK AFTER HYDRATION

	Adm.	4th Day	4th Day +3 Hr.	5th Day	11th Day
Hematocrit	56	46		47	36
CO_2 comb. power mEq/L	31	46	30	31	27
Chloride mEq/L	97	71	103	86	98
BUN	15				72
Sodium mEq/L	149				156
Potassium mEq/L	3.8				2.3
Cumulative Na bal. mEq.		+23			+739
Cumulative K bal. mEq.		+2.1			+211
Cumulative Cl bal. mEq.		+49			+736
Cumulative Fluid bal. cc.		+804			+2449
		Day of shock			

TABLE II.

	Number of Determinations	Volume cc	Average mEq/L		
			Sodium	Potassium	Chloride
Gastric suction* No oral intake	68	795	64	9.7	87
Gastric suction* Oral intake av. 792 cc.	108	1267	45	6.1	89
Gastric suction after gastrectomy. Oral intake average 269 cc.	86	603	70	7.5	63
Urine	518	1404	64	36.0	39
Bile	51	195	136	4.7	100

*Gastrectomy patients not included.

From these examples one can see at a glance that reliance upon estimate of, say, 800 to 1000 cc. insensible water loss would lead to serious aberration in fluid balance before many days had passed.

3. Daily body weight, when determined carefully with proper apparatus for bed patients, is an ideal method of following fluid balance. Before dehydration becomes clinically evident a 6 per cent loss of body weight has occurred (9 pounds in a 150-pound man). When the measured fluid output and calculated insensible water loss are subtracted from fluid intake and this figure checked against body weight, one has a fairly accurate evaluation of a patient's fluid status.

Electrolyte Balance.—Reliance upon a normal or average behavior of electrolyte physiology may lead to disaster. Laboratory determinations of serum levels and excretion quantities provide a far greater safety.

1. The state of hydration has a profound effect upon the electrolyte status for overhydration leads to dilution of electrolytes and dehydration causes serum electrolytes to rise above normal levels.

Since the symptoms of electrolyte disturbances are direct results of the serum concentration levels rather than total body quantities, it is important to realize that an untreated patient who has suffered loss of both water and electrolytes may, under certain circumstances, have an entirely normal serum electrolyte pattern and therefore no clinical symptoms of electrolyte deficit. It is such a patient who, on repletion of too much water in proportion to electrolytes, will surprise the physician when he develops severe symptoms of mineral deficit. Conversely, a patient who has been overtreated (usually by saline solution) may exhibit symptoms of hyperconcentration if, in recovery of edema, he loses water faster than electrolytes.

Thus, one must gather as many facts as possible on the state of hydration in order to properly evaluate the significance of serum electrolyte determinations.

To illustrate, let us assume a hypothetical case of dehydration and consider the chloride ion:

Admission: Hematocrit, 56, chloride 97 mEq, weight 50 kg.
Normal: Hematocrit, 40, chloride 104 mEq

Since the chloride level is determined from extracellular fluid space, and since this space is 20 per cent of total body, take 20 per cent of 50. = 10 liters (this patient's normal extracellular fluid volume).

Hematocrit of 56 — 40 = 16. 16/40 = 40 per cent less fluid than normal.
40 per cent × 10 liters = 4 liters deficit.

To calculate predicted serum levels in re-hydration, a proportion may be set up.

Pt. hemat.:pt. cl.::normal hemat.:expected cl.
56:97::40:cl′
56 cl′ = 3880
cl′ = 69 mEq/L

Thus 69 mEq/L would be the chloride level in the serum if the 4-liter water deficit were replaced without giving any chloride ion.

To calculate the total existing chloride deficit in a 10-liter space (pt. normal):

69 × 10 = 690 mEq cl actually present on admission.
104 × 10 1040 mEq cl normal chloride content of 10 L. space.
1040 — 690 = 350 mEq cl calculated total deficit of chloride in extracellular space.

In order to adjust to shifts that may occur and to permit re-evaluation in the course of repletion, a wise plan is to replete the calculated deficit over a three-day period. Therefore, in this example, the total deficit of 350 mEq cl. and 4 liters of water are divided by 3, thus, 117 mEq cl. and 1333 cc. of water are added to each day's chloride and fluid requirement for three days.

TABLE III. TABLE OF AVERAGE URINE EXCRETION RATIO OF SODIUM TO CHLORIDE

	300 Determinations mEq/L		
	Pre-op.	Post-op.	Total
Sodium	90.8 mEq (5.3 gm.)	37.0 mEq (2.2 gm.)	62.6 mEq (3.7 gm.)
Chloride	86.7 mEq (5.1 gm.)	44.3 mEq (2.6 gm.)	64.1 mEq (3.8 gm.)

TABLE IV. EXAMPLES OF DEVIATIONS FROM THE AVERAGE

	Na:Cl Urine Excretion Ratio. Daily Determinations (mEq/L)							
Case No. 1	Sodium	252	116	140	270	324	247	
	Chloride	118	49	113	62	65	26	
Case No. 2	Sodium	132	148	185	99			
	Chloride	19	10	3	2			
Case No. 3	Sodium	125	91	2	5	5	5	2
	Chloride	71	79	18	38	48	38	70

TABLE V. RELATION OF SERUM POTASSIUM TO BALANCE mEq/L

Case No. 1	Serum K	4.9	2.9		2.7		3.0			
	Daily K Bal.	−3.1	−114.6	−51.0	−26.0	+5.9				
Case No. 2	Serum K		3.2			2.4			5.2	
	Daily K Bal.	−41.0	−15.0	−11.0	−29.0	+29.0	+199.0			
Case No. 3	Serum K	5.8					2.9			4.0
	Daily K Bal.		−41.0	−39.0	−3.0	−27.0	−89.0	+124	+56	

The serious drop in serum electrolyte concentration that follows rehydration without calculating the anticipated dilution and without adequate electrolyte administration to prevent dilution may be exemplified by data from an actual case.

This patient had partial pyloric obstruction for two months. Her general condition was quite satisfactory and dehydration was not apparent clinically. Fluid administration was, in the main, without electrolytes. On the fourth day profound shock appeared. The blood pressure was near zero until Levophed (nor-epinephrine) was given. Such shock has been called "hypochloremic shock" but may well be a more general condition of hypo-osmolarity.

When shock state was discovered, the patient was given 3150 cc. of fluid containing 642 meq. sodium, 656 meq. chloride, 23 meq. ammonia, 33 meq. potassium and 33 meq. phosphate, and after the first liter, Levophed was no longer needed. In such a critical case the usual rule of cautious repletion over a three-day period was deferred and a 3 per cent concentration of sodium chloride was used in the first 500 cc. It may be noted that a normal chloride level was attained in three hours. This case also exemplifies the rise in BUN in the presence of severe electrolyte imbalance.

This elevation may occur as it did in this case in spite of a satisfactory urine volume.

It should be noted that the serum potassium dropped to an even lower level in spite of a positive balance.

The strongly positive sodium balance no doubt is a result of giving too much sodium chloride to correct what should have been mainly a chloride deficit (pyloric obstruction). A hypernatremia of 156 mEq/L approaches a serious level. More ammonium chloride perhaps should have been given, but the ammonia ion is extremely toxic if not immediately converted to urea by the liver. If this concern is justified, then in cases of shock a rapid and safe means of pure chloride administration does not exist.

Blood and extracellular space measurements are more accurately determined by Evans Blue and inulin methods, but the hematocrit is more generally available and is reasonably helpful if anemia or blood loss do not interfere with accuracy.

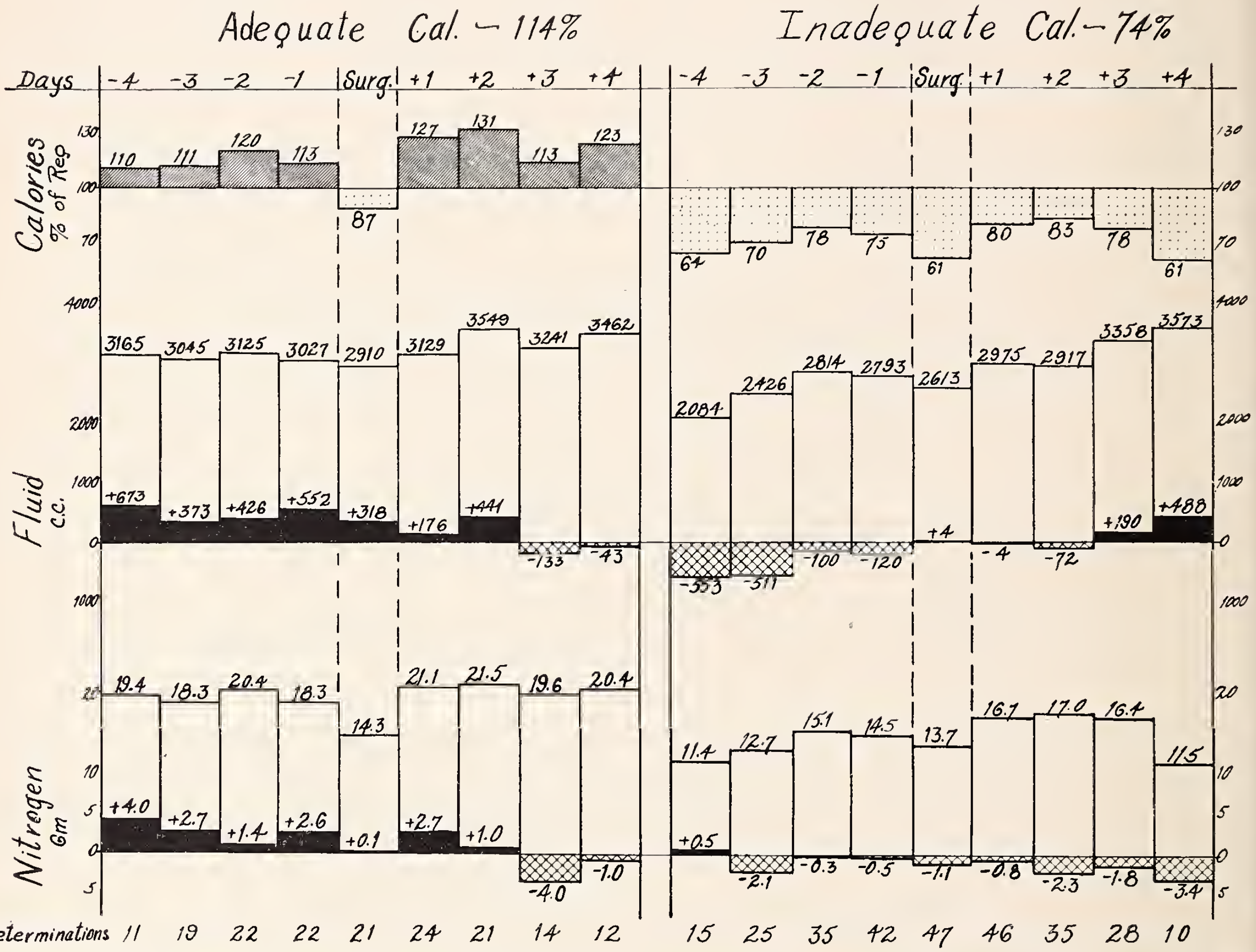

Fig. 1. Intake from zero upward; output from intake line downward. Balance indicated by +black and —cross hatch. Balance studies on 102 patients averaging five days study for each patient. Comparison between the adequate and the inadequate groups reveal a more positive nitrogen balance associated with a better caloric intake. In both groups the catabolic effect of surgery is observed in the postoperative period.

Only the quantity of extracellular ions such as sodium, chloride, etc., are amenable to calculation by this method. Potassium deficit must be estimated by other means.

2. Electrolyte losses that occur from day to day in the hospital must be carefully collected. Urine, gastric suction and bile may, depending upon case urgency, be pooled into one to three day output collections.

Although rather extreme variations occur in gastric suction and particularly in urine electrolyte content, average values are of some use as a starting point.

From several hundred determinations we have compiled the following chart on average electrolyte concentrations in various body fluids and excreta.

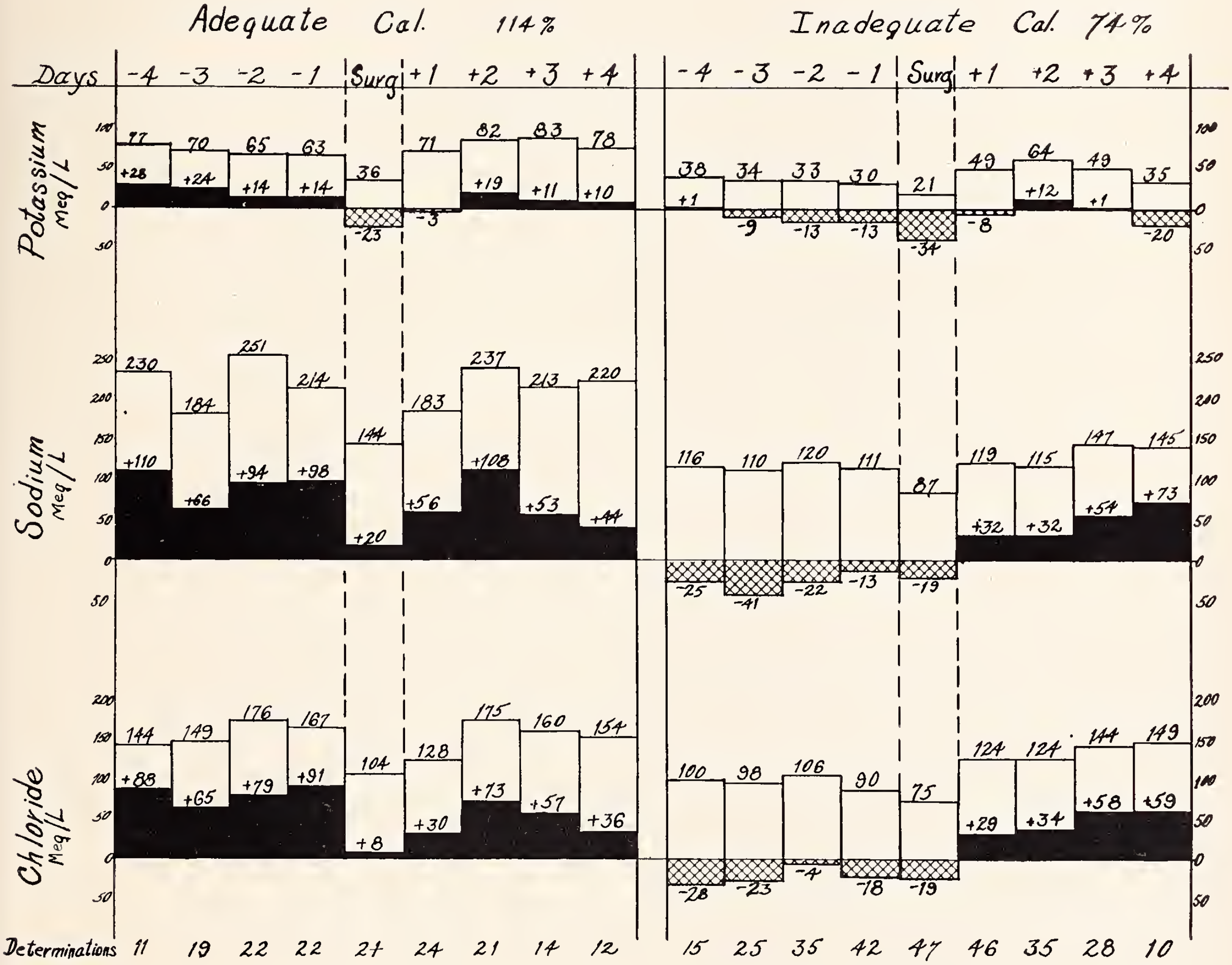

Fig. 2. Intake from zero upward; output from intake line downward. Balance is shown by +black and —cross hatch. Electrolyte balance studies upon the same group of patients in Figure 1. The potassium shift toward negativity from the preoperative to the postoperative period is not as great in the adequate calorie group. The sodium and the chloride ions exhibit an obvious difference between the adequate and the inadequate calorie groups. The intake of these ions does not vary sufficiently within each calorie group, i.e., between pre- and postoperative phase, to explain the variation in pre- and postoperative behavior between adequate and inadequate calorie groups.

In the average surgical patient sodium is excreted in an amount equivalent to the chloride excretion. The simplicity of urinary chloride determinations makes the method quite attractive. However, many physiologic influences may produce extreme variations in any given patient. Therefore, in dealing with an individual case it is unsafe to presume a 1:1 ratio between chloride and sodium. True sodium and chloride determinations should be obtained from the specimen.

The average surgical patient has a urine sodium to chloride ratio of 1:1.

The marked drop in sodium and chloride output in the postoperative period should be noted and taken into consideration in postoperative electrolyte administration.

Marked deviations from the average 1:1 excretion ratio frequently occur both from one patient to another and from day to day in the same patient.

3. Balance data are particularly valuable in regard to the potassium ion. The main concern in surgical patients is directed toward prevention of hypopotassemia and toward correction if it has already occurred.

Since less than 2 per cent of total body potassium is located in the extracellular fluid, a relatively large depletion (up to 50 per cent) of body potassium occurs before the loss is reflected in the serum potassium level. Although clinical symptoms attributed specifically to a low potassium occur only after the serum level dropped at least one milliequivalent below normal, there are many undesirable changes of cellular physiology aready under way. The presence of a strongly negative potassium balance permits a one-day to three-day prediction of drop in serum potassium level.

CHART I. SHEET FOR RECORDING DATA

Name		Age	Wt.		Diagnosis:				
Date									
Fluid cc.	In	Oral							
		Parenteral							
	Out	Urine							
		Suction							
		Insensible							
		Other							
	Fluid Balance								
Calories		Given							
		Requirement							
	Caloric Balance								
Sodium mEq		In							
		Out							
	Sodium Balance								
Potassium mEq		In							
		Out							
	Potassium Balance								
Chloride mEq		In							
		Out							
	Chloride Balance								
Urine		Spec. Gr.							
		Sugar Gm.							
Blood	Hemoglobin (15) Gm.								
	Hematocrit (37-45) %								
	CO_2 (26-28) mEq/L								
	Cl (100-106) mEq/L								
	K (3.8-5.2) mEq/L								
	NA (137-147) mEq/L								
	Sugar (80-120) mg. %								
	BUN (8-20)								
	Protein A/G (6.7-8.4)								

4. Shifts of electrolytes within the body may be detected by studies of the balance data.

For example, a patient who had received an excess of sodium exhibited the following balance findings in a combined three-day study.

Sodium balance + 800 meq.
Serum sodium rose from 158 to 170 meq/L
1/5 (extracellular fluid) × 65 (body wt. in kg.) = 13 liters extracellular fluid.
170 — 158 = 12 meq/L increase of sodium
12 × 13 = 156 meq. in extracellular space.
800 — 156 = 644 meq. of extra sodium located elsewhere than the extracellular fluid space.

Caloric Balance.—An adequate caloric intake appears to have an influence upon the equilibrium of electrolytes as well as nitrogen. It is reasonable to assume that if a patient's weight is maintained while on a sub-caloric diet that a gradual dilution of his system, a water logging, must occur since without meeting caloric requirements tissue loss is obligatory.

Complete balance studies of calories, nitrogen, sodium, potassium, chloride and fluid were recorded on 102 patients, an average of five days for each patient. After correction for urinary loss of calories (sugar), the 102 patients were divided into two groups. One group received 100 per cent or more of caloric requirement and the other group was given less calories. Significance of this material must await completion of statistical evaluation now in progress. However, certain differences between the adequate and the inadequate calorie group are demonstrable in graphic form (Fig. 1).

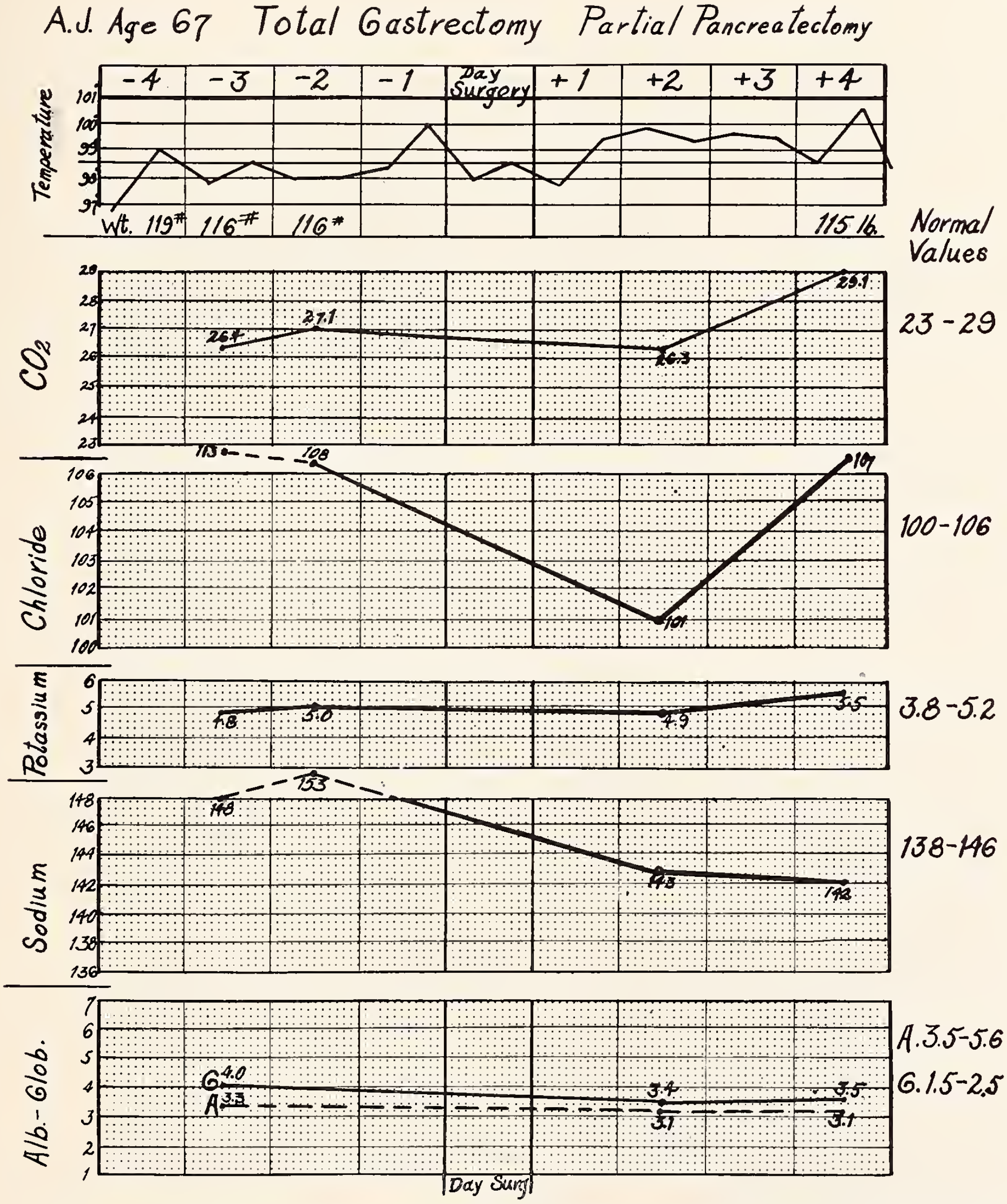

Fig. 3. Blood chemistry studies on a patient operated upon for extensive gastric cancer. Caloric intake averaged 89 per cent of requirement.

An obvious difference in the behavior of the sodium and the chloride ion exists. Even though the intake is not the same in the adequate as in the inadequate calorie group, there is noted a reversal of the preoperative and postoperative behavior from one group as compared to the other. It is doubtful that such a pattern could be explained on the basis of intake differences.

Aside from the expected postoperative excretion increase in potassium we note a proportionately smaller postoperative increase in the inadequate calorie group. One might speculate that a negative caloric balance implies greater cell breakdown to meet obligatory energy demands, thereby releasing more endogenous potassium and thus less need from exogenous sources, hence proportionately greater urinary loss of administered potassium. Statistical evaluation and more accurate conclusions will be the subject of future publications on these data.

In order to achieve equilibrium consistently all possible protein as blood or amino acids must be given. In our opinion all fluid given should contain amino acids and high caloric support.

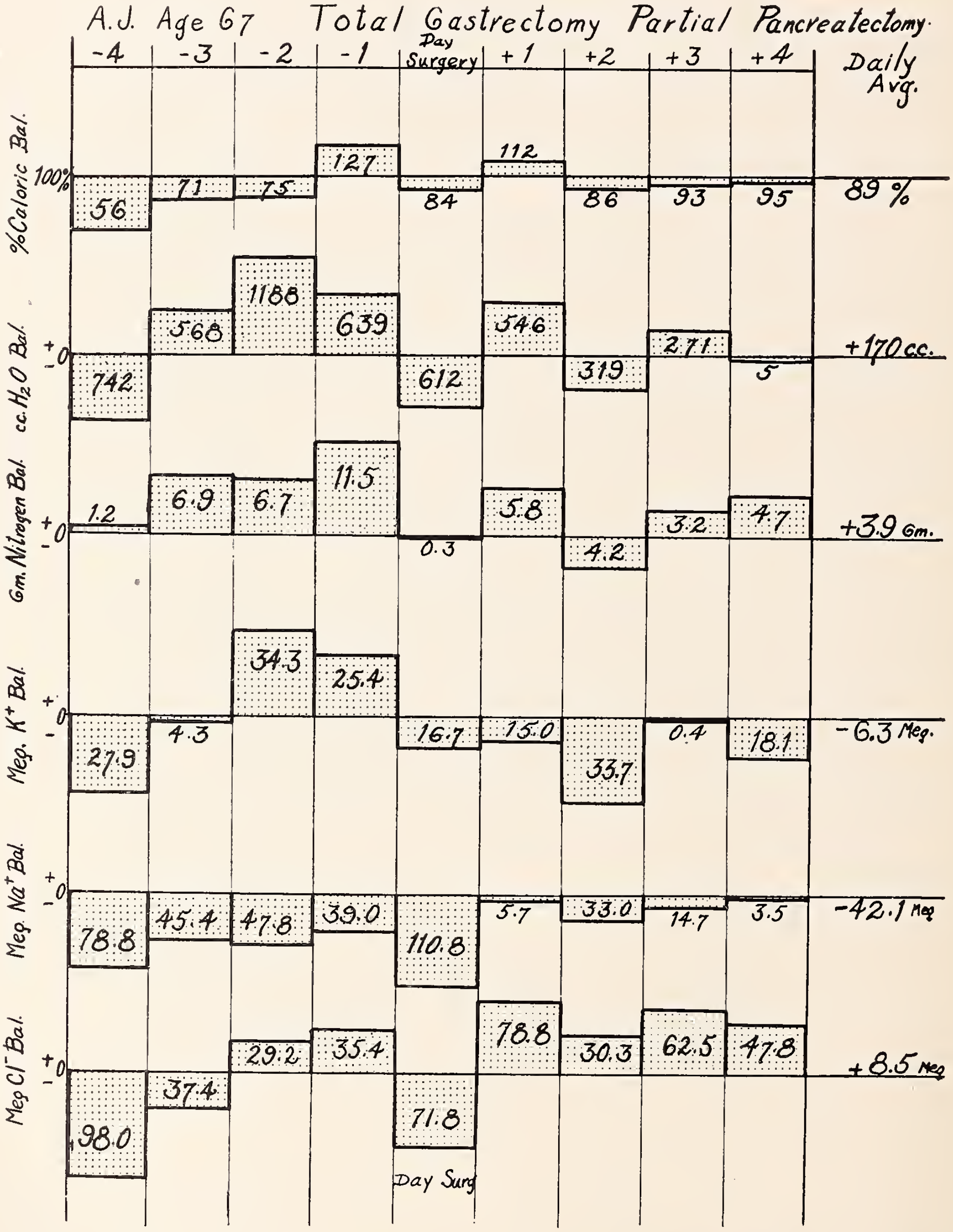

Fig. 4. Balance studies on a patient with gastric carcinoma (same as Fig. 3) reveal all balances close to equilibrium for nine days. The exception of sodium was intentional due to high initial sodium level.

Nitrogen Balance need not be calculated for the practical management of a case. Equilibrium or positive balance is of vital importance and is dependent upon adequate caloric and nitrogen intake.

Case Studies by Graphic Representation.—Figures 3 and 4 represent the blood chemistry studies and the balance calculations on a patient with carcinoma of the stomach. The moderately negative sodium balance was the result of planned therapy since the serum sodium was high at the outset. Hypernatremia is likely to be particularly bad in the face of a reversed albumin globulin ratio. Other factors were maintained very near equilibrium and the patient was discharged from the hospital the eighth postoperative day.

In burn cases, an accurate electrolyte and fluid balance is impractical. Therefore, the blood chemistries must be observed even more closely. Figure 5 and Table VI represent determinations on a fourteen-year-old girl with a 68 per cent body surface burn, 30 per cent of her body surfaces requiring grafting. On a high caloric intake and close electrolyte and fluid adjustment the patient was discharged at 106 days without dressings and returned to school two weeks later.

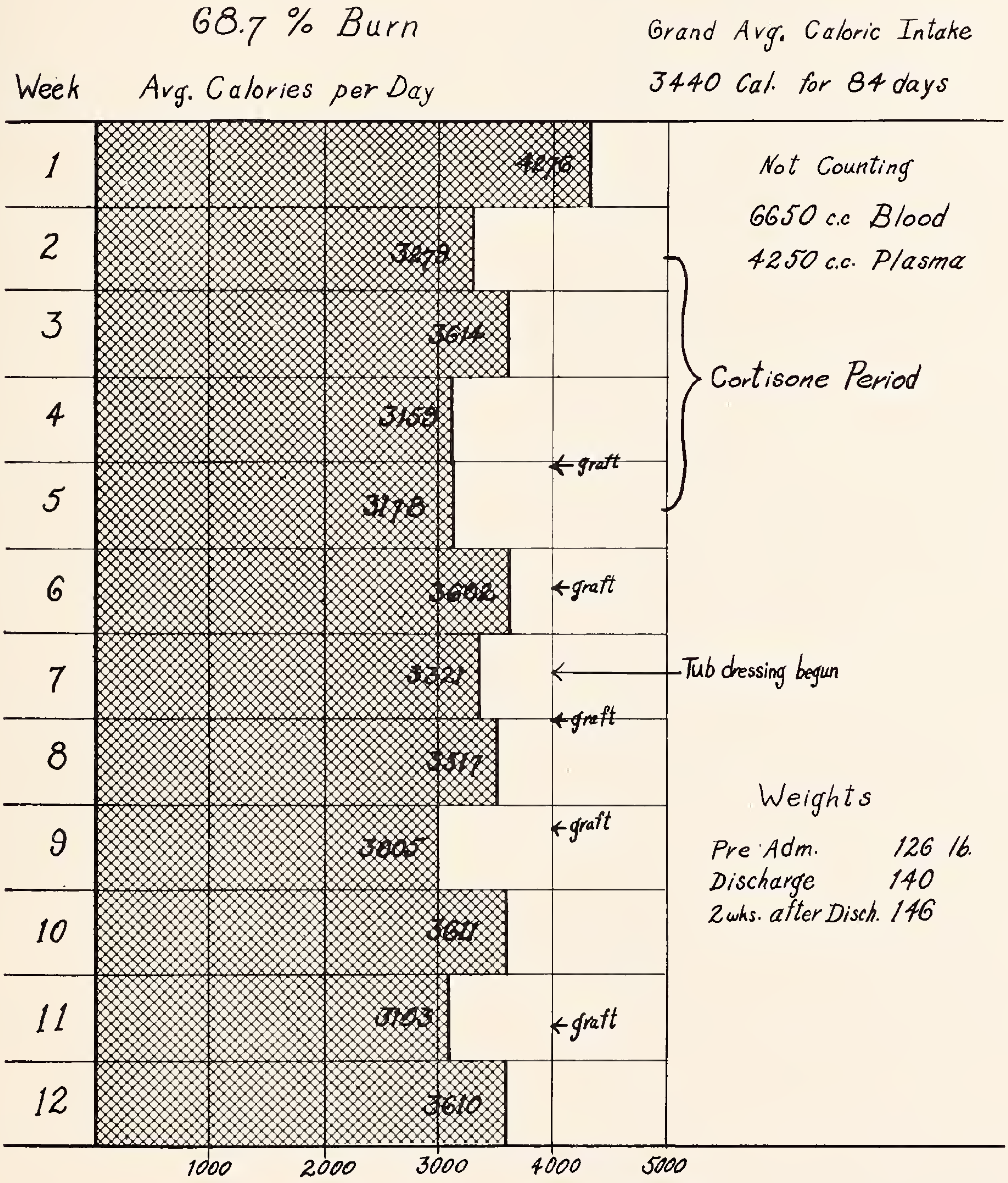

Fig. 5. An extensively burned patient can be maintained on high caloric intake by fluid infusions of 1000 calories per liter solution.

High caloric intake was attained by parenteral supplement (Fig. 5), particularly during the first week, of a solution containing 1000 calories per liter (6 per cent amino acids, 12 per cent invert sugar and 5 per cent alcohol). Cortisone therapy was tried for a three-week period as a further attempt to increase her appetite, but no change was found so it was discontinued. We attribute her survival and rapid healing to her youth, balanced

TABLE VI. BLOOD PROFILE—P.B. 68.7 PER CENT BODY SURFACE BURN

Days Post Burn	Adm.	1	1	2	3	4	5	6	7	8	9	10	11	14	16	20	27	34	38	48	58	63	72	76
Hgb.	16.7	19.8	21.8	20.4	15.5	15	16.1	15.5	16.1	17	16.7	16.7	15.8			16.7	14.7	13.3	13.8	15.5	12.6	10.7	11.7	16.7
Hemat.	47.5	58	65	58	46		42	46	46	49	50	42	47			47		39	43					
Rbc.	5, 17		6, 74																					
CO_2		17			25.5	26.4	29.1	26.4	26.4	24.5			27.7			27.8		29.0	30.0					
Cl.		118		104	99.8	99.1	87.5	99.1	99.1	99.1			99.1			99.1		104	95.7					
Na.		147		137	134			136	136					142	143.6	133								
K		4.3		5.4	5.2			4.2	4.5	4.7					5.0	5.9		4.1	4.5					
BUN		8			12			11	12	14			15			15		9	13					
Sugar		332						158		130			117											
Alb.					2.3			2.4		2.5			2.2			2.8		2.3	2.8					
Glob.					2.3			2.5		4.0			4.2			5.0		4.4	3.8					
Total Pro.					4.6			5.9		6.5			6.4			7.8		6.7	6.6					

Normal Range

CO_2	23-29 mEq/L	Prot. A	3.5-5.6 Gm.%
Cl	100-106 mEq/L	G	1.5-2.5
Na	138-146 mEq/L	T	6.8-8.5
K	3.8-5.2 mEq/L	Sugar	80-120 mg.%
BUN	under 20	Hemat.	40-48%

high caloric parenteral and oral feedings, and to careful attention to blood, fluid and electrolyte administration.

The blood chemistry profile is an invaluable guide to therapy (Table VI). Attention may be called to the rise of hemoglobin and hematocrit at the end of the first twenty-four hours. The blood sugar of 332 on the day after admission is no doubt a reflection of the carbohydrate disturbance secondary to stress. We now believe that insulin should be given at that time as an aid in prevention of electrolyte anomalies. The reversal of the A/G ratio persisted, in spite of high caloric intake, until after skin grafting had been completed. During this period repeated whole blood transfusions were needed to correct anemia.

General Rules and Miscellaneous Suggestions

1. Fluid volume management is best accomplished by accurate daily weights which are checked against output measurements and insensible water loss calculation.

2. Fluid and electrolyte balances should be maintained at equilibrium except in correction of a prior deficit (repletion) or when a previous excess is being depressed (depletion).

3. An average preoperative sodium requirement is 105 mEq (6.2 gm.) and 74 mEq (4.4 gm.) in the postoperative period.

4. Potassium requirement averages 45 mEq (3.4 gm.) preoperatively as compared to 67 mEq (5.0 gm.) postoperative.

5. The electrolyte output in pooled samples of urine, suction, etc., is a valuable tool in balanced management. Output values are particularly useful when correlated with serum levels and state of hydration since deficits are recognized prior to drop of serum levels and onset of symptoms.

6. Water should be restricted on the day of surgery to about 2500 cc. above suction losses since the renal response to trauma is one of diminished water output.

7. Blood sugar levels, in the non-diabetic surgical patient, are very valuable since hyperglycemia is commonly associated with electrolyte disturbances. Insulin therapy aids recovery. The so-called pseudo diabetes of trauma is a very common observation in surgical patients. One unit of regular insulin to each four grams of carbohydrate will substantially aid carbohydrate metabolism. Much larger doses are occasionally required.

8. In hypochloremia associated with hypoproteinemia and edema, salt must not be given since the total body chloride quantity is likely normal. The serum chlorides will rise as protein replacement dispels the edema.

9. Hypochloremic alkalosis usually is pathognomonic of potassium deficiency and sodium chloride administration only increases the alkalosis. Serum sodium and potassium are essential laboratory information.

10. In gastric alkalosis due to loss of hydrochloric acid, there has been a great loss of chloride ion without a proportionate loss of sodium. In such cases give 0.6 per cent to 2.0 per cent ammonium chloride, some chloride as potassium chloride, or 6 cc. of concentrated hydrochloric acid (60 — 72 mEq) in 1000 cc. of 5 per cent amino acid solution as a buffer.

11. Ammonium chloride should not be given in the presence of shock because the ammonia ion is very toxic. If the shock state liver does not rapidly convert ammonia to urea toxicity will rapidly appear.

12. Acidosis, with a normal or high chloride, is best managed with 1/6 molar lactate. Again a fairly normal liver function should exist to insure handling of the lactate radical.

13. Electrolyte replacement is best done by adding the desired mEq of each electrolyte from stock concentrates to each liter of basic fluid, thus avoiding the need to order a liter of this and a liter of that.

14. Finally, a high caloric, adequate protein intravenous fluid diet with the addition of the desired electrolytes affords a more smooth and balanced management of the patient's physiologic needs.

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WANTED—A NEW LOOK AT MEDICAL MANPOWER PROBLEMS

Congressional re-evaluation of the physician draft, with more attention paid to the needs of medical education and research, is advocated by Dr. Dean F. Smiley, secretary of the Association of American Medical Colleges, in an editorial published in the March issue of the *Journal of Medical Education.*

Dr. Smiley warns that the nation's potential supply of medical teachers and researchers will be seriously depleted if the present policy of drafting young physicians is continued in its present form. Since the beginning of World War II, Selective Service has delivered as many doctors to the Armed Forces as they estimated they would need.

The vagaries of Selective Service requirements, he says, in some essential respects have been neither fixed nor fair. Interpretations of rulings which vary from state to state, changes in physical qualifications and other factors have tended to keep young physicians, educators and researchers in confusion.

Military service or the threat of it has made long-term planning almost impossible for these men. In addition, more than 3,000 graduate students in the sciences have had their careers interrupted or terminated by military service. "How long," asks Dr. Smiley, "can this nation continue to ignore the need for medical teachers and researchers before we find ourselves falling behind Russia in our ability both to train new physicians and to carry out important life-saving research in such fields as aerophysiology, antibiotic therapy and radiation therapy?"

Complete Obstruction of the Bowel in the Newborn

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BECAUSE many disturbances in the various systems of the newborn result in symptoms that are common to intestinal obstruction, it is often difficult to recognize specific types of obstruction. Yet if obstructions are not recognized early, the newborn may die. Even if the newborn lives long enough for the diagnosis to be made and corrective procedures to be carried out, every hour, even every minute increases the hazard to the newborn.

By limiting these obstructions to the ones obvious shortly after birth, the congenital anomalies become the most frequent source of obstruction. The incidence of various types of intestinal obstruction seen without any limit to age shows that the congenital anomalies are relatively unimportant (Fig. 1).

At St. Mary's Hospital in Minneapolis from 1942 through 1951 there were 24,594 live births. In this group there was a malrotation of the colon, an ileal stenosis with angulation of the bowel, an ileal atresia, a persistent cloaca with a persistent common membrane, and a meconium ileus. That is five congenital anomalies with complete bowel obstruction in 24,594 cases or close to one per 5,000 live births.

1. *Congenital atresia of the intestine and colon* occurs once in 20,000 births. Although the exact causes of atresia cannot be given, one can safely say they are developmental abnormalities accompanying faulty embryologic events. Ladd and Gross say that such events occur between the fifth and tenth week of fetal life, because before that time and after that time there is a rapid proliferation of epithelium from the pylorus to the ileocecal valve which obliterates the lumen. The formation of vacuoles and then the coalescence of these cystic spaces re-establishes the lumen. Arrest of growth at this period anywhere from the pyloric sphincter to the ileocecal valve may ensue in an atresia. The most frequent site of such an obstruction is in the lower ileum and jejunum. In 392 cases collected by Davis and Poynter, 161 were in the jejunum and ileum.

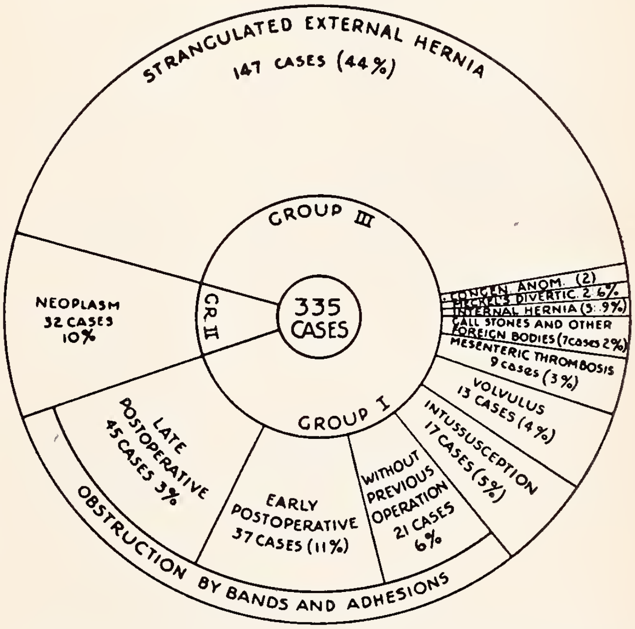

Fig. 1. The incidence of various types of intestinal obstruction, over a ten-year period at the Massachusetts General Hospital, during which time there were 39,936 surgical admissions, 335 cases of bowel obstruction were seen. The high incidence of strangulated hernia in this series is striking. (Reprinted by permission of Wangensteen, O. H., from "Intestinal Obstruction", ed. 2).[7]

Figure 2, taken from Ladd and Gross, shows the usual distribution of sites of obstruction. The obstruction is usually single but occurs in multiple areas in 15 per cent of the cases.

The obstruction may occur in two forms. There may be an internal diaphragm or veil which completely blocks the lumen, or there may be a blind sac of proximal intestine connected to the caudal section by a fibrous band. Above the obstruction, the bowel may be so distended as to rupture. Below the obstruction the bowel is collapsed and ribbon-like, especially in low obstructions.

When the obstruction occurs high in the intestinal tract the child usually has symptoms

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Inaugural Thesis presented before the Minneapolis Academy of Medicine, November 17, 1953, Minneapolis, Minnesota.

early. They always occur within the first day of life. Vomiting which becomes more and more frequent and intense is a major symptom. There may or may not be bile in the vomitus, depending on the level of the obstruction. Obstructions above the papilla of Vater are rare, so that nearly all such infants have bile-stained emesis. The stools are usually scanty in amount and drier than normal. The color is usually not so tarry. The degree of abdominal distention depends upon the level of the atresia. A high obstruction may show little distention because the baby can keep the stomach empty by vomiting. An atresia in the jejunum will show marked and general distention due to gas in the bowel above the obstruction which is not expelled with vomiting. A low atresia often shows intestinal patterning. If fever is present, it is usually due to dehydration and/or peritonitis following rupture of the bowel.

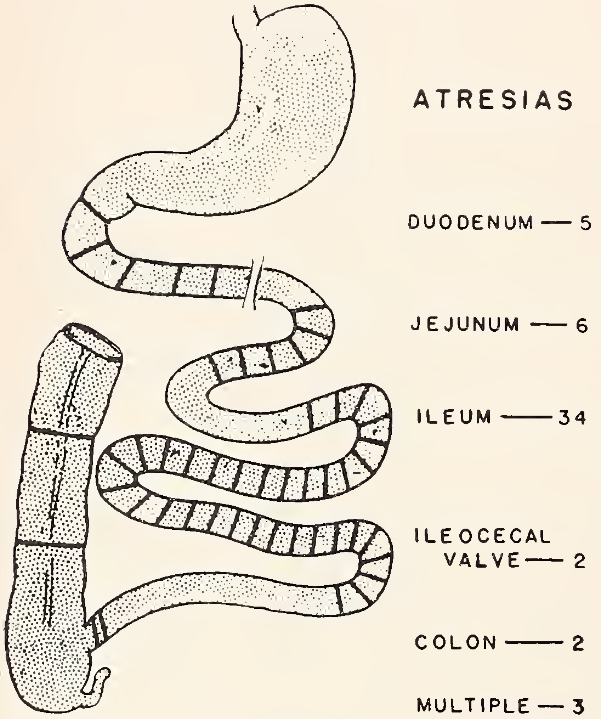


Fig. 2. Diagram showing position of atresias in fifty-two cases. (Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood").[2]

Because of its aid in locating the point of obstruction, roentgen examination is important. Gas in the stomach and duodenum, without gas in the lower intestine, locates the obstruction in the duodenum. A thin mixture of barium or lipiodol orally will locate a high obstruction, and a barium enema will locate an obstruction in the colon. Actually, the use of contrast media is seldom needed, and to avoid the danger of aspiration, lipiodal is preferred.

Farber's test will help one determine if the obstruction is complete or partial, and also if it has been present since before birth. The meconium is examined and the presence of vernix cells (cornified epithelial cells) can quickly be established.

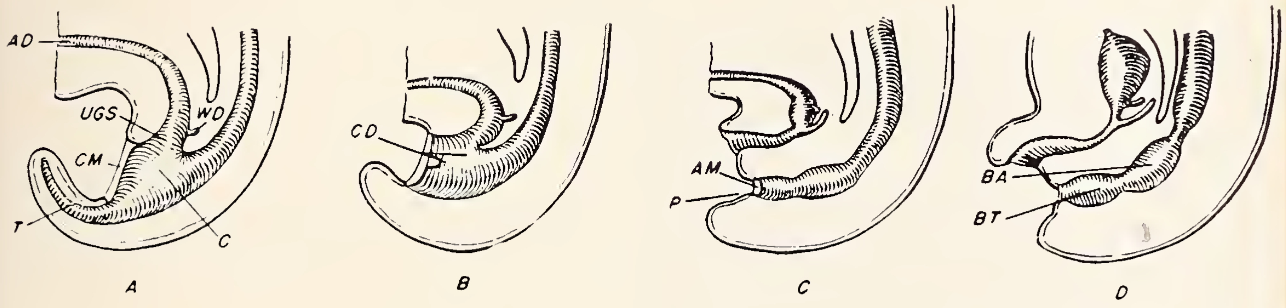
A low bowel obstruction can be differentiated from imperforate anus by external inspection and the passing of a bougie or a barium enema. A high bowel atresia can be differentiated from internal herniation of the bowel or volvulus by absence of bile in the meconium. Also, the abdomen is flaccid and non-tender in an atresia, in contrast to tenderness in internal herniation or volvulus.

2. *Malrotation of the intestine and colon* is much more complicated than atresia as to etiology. To try to visualize it quickly and yet clearly, picture the gut first as a long tube and then as a tube with a "U" shaped piece but still in a single plane suspended by a common dorsal mesentery. Now think what would happen when one section of this tube grows more rapidly than another. It would either have to extend a greater distance, which it can't do because of it's abdominal limitation, or twist to take up the slack. To add to the slack, the "U" shape which is the portion of the intestine protruding through the umbilical orifice now returns to the abdominal cavity. This is at the tenth week of fetal life. The position that various parts of the intestinal tract will take in relation to each other is the result of the intestine rotating in a counter-clockwise manner; and because the cecum is the last part to return to the abdomen, the colon is carried forward over the superior mesenteric artery.

The malrotation may be due to non-rotation of the midgut loop, reversed rotation of the midgut loop, or some other abnormal rotation of the midgut loop. In reversed rotation, the transverse colon lies behind the superior mesenteric artery, giving rise to constricting bands, and there is a lack of attachment of the mesentry along the posterior abdominal wall, predisposing to volvulus.

There are two main features of a malrotated bowel. One is obstruction near the duodeno-jejunal angle occasioned by abnormal adhesions, that is, bands lying directly across the descending portion of the duodenum and obstructing by extrinsic pressure. The other is a volvulus of the small bowel, cecum, and ascending colon or torsion about the superior mesenteric artery. The symptoms are vomiting, abdominal distention, peristolic waves, dehydration, and fever. X-ray will show a gas-filled stomach and duodenum. If there are no bands across the duodenum, there will be an enormously dilated jejunum and ileum. A barium enema will show the cecum in the right epigastrium in an incompletely rotated position. Laboratory findings are of little or no importance. The only change is a change consistent with dehydration. It may be difficult at times to differentiate this from intestinal atresia, but once it has been established that surgery is necessary, this point is not too important.

3. *An imperforate anus* occurs once in 5,000 newly born babies. In the embryo, there is a period when the urogenital and intestinal tracts open into a common cavity, the cloaca. This is closed from the exterior by a cloacal membrane. As the embryo develops there is a cleavage of the urogenital tract from the intestine. The two are entirely separate at the seventh week (Fig. 3). Late in the seventh week the urogenital membrane, the anterior part of the original cloacal membrane, opens; and later the anal membrane (the posterior part) opens. Arrest in development at this stage results in an imperforate anus or a stenosis. There are four types of anal anomalies (Fig. 4). These are:



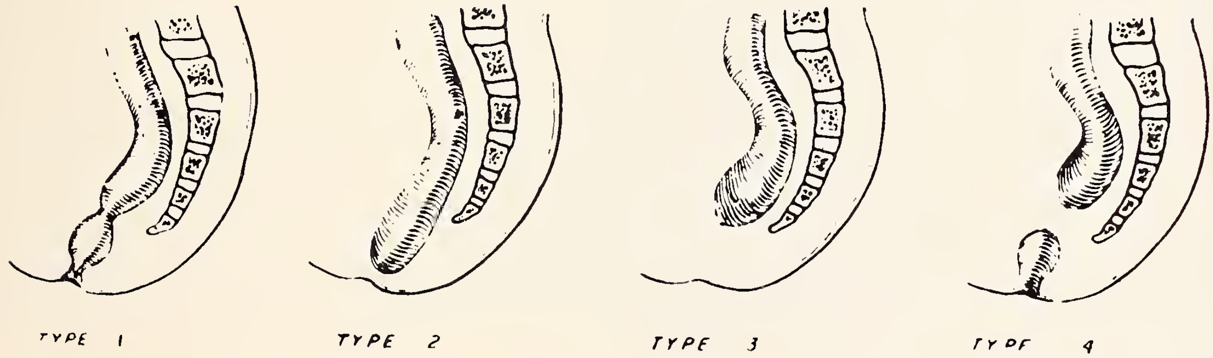


Fig. 3. *(Above)* Normal development of the anus and rectum. A, 7.5 mm. stage. B, 9 mm. stage. C, 22 mm. stage. D, 42 mm. stage.

AD, Allantoic Duct	C, Cloaca	P, Proctodeum
AM, Anal Membrane	CD, Cloacal Duct	T, Tail Gut
BA, Bulbus Analis	CM, Cloacal Membrane	UGS, Urogenital Sinus
BT, Bulbus Terminalis		WD, Wolffian Duct

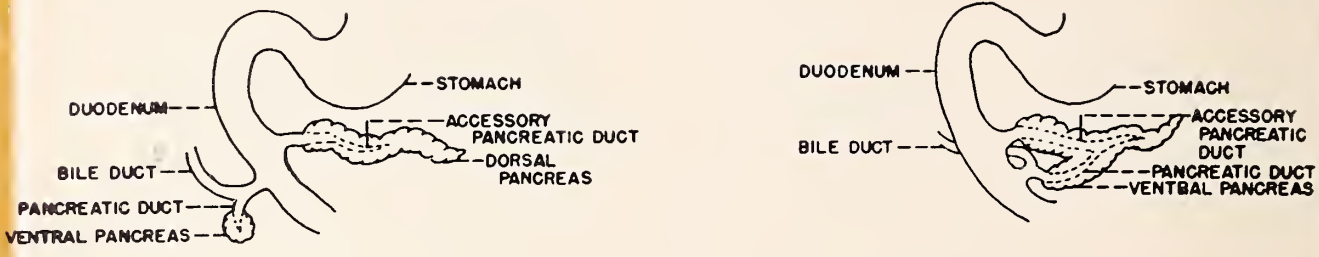
(Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood".)[2]

Fig. 4. *(Below)* Types of anal and rectal abnormalities. Type 1—Stenosis at anus or at a point several centimeters above the anus. Type 2—Imperforate anus. Obstruction due to persistent membrane. Type 3.—Imperforate anus. Rectal pouch ending blindly some distance above anus. Type 4—Anus and anal pouch normal. Rectal pouch ends blindly in hollow of sacrum. (Reprinted by permission of Gross, Robert, from "Abdominal Surgery of Infancy and Childhood.")[2]

1. Stenosis of anus or just above anus,
2. Imperforate anus,
3. Imperforate anus with rectal pouch ending blindly, and
4. Anus normal with rectal pouch ending blindly.

The most difficult to diagnose is type four. The easiest types to treat are types one and two.

No type of bowel obstruction is more easily recognized than the first three types. Clinically a child will take feedings for at least twenty-four hours before regurgitation begins. The external recognition of the imperforate anus is apt to be discovered before any obstructive symptoms develop. The x-ray is a big help in determining the type of obstruction present. The Wangensteen-Rice maneuver is a help in locating the height of the blind pouch. An x-ray is taken of the infant in an upsidedown position. The height the gas ascends will determine the level of the blind pouch. One must not rely on this method before twenty-four hours have elapsed to allow time for the gas to pass through the sticky meconium. About twenty-eight per cent of these children have associated anomalies.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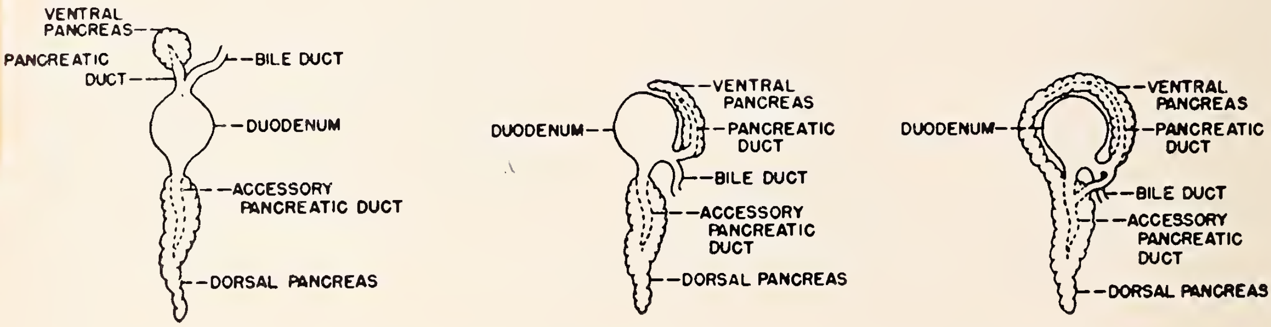
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Fig. 5. *(Above) (left)* Pancreas of human embryo of five weeks (Kollman). *(Right)* Pancreas of human embryo at end of sixth week (Kollman). Courtesy, Gray's Anatomy. (Reprinted by permission of May, Charles D., editor of *Pediatrics*)[4]

Fig. 6. *(Below)* Diagrammatic representation of mode of development of annular pancreas. Free end of ventral pancreatic anlage becomes fixed so that during its subsequent migration, with rotation of duodenum toward dorsal anlage it is drawn out. With fusion as shown at right, it comes to surround duodenum (after Lecco). (Reprinted by permission of May, Charles D., editor of *Pediatrics*)[5]

4. Extrinsic bands, aside from those associated with malrotation of the gut and post-surgical complications, are rare. However, one special type will be discussed, the annular pancreas. This obstruction is due to a complete encirclement of the duodenum by a ring of pancreatic tissue. The majority of individuals with an annular pancreas do not develop symptoms until they have reached adult life, or they may never develop symptoms and the finding is one at the autopsy.

Normally, the pancreas develops as two distinct entodermal diverticuli known as the dorsal and ventral anlage. The dorsal anlage becomes the body and tail of the pancreas. The ventral anlage comes off the primitive bile duct and forms the head of the pancreas. At six weeks in fetal life the two portions fuse. This is the period when the anomaly arises (Fig. 5). To cause obstruction, one of three situations arises. First, if the ventral anlage does not rotate to its normal posterior position there will be pancreatic tissue on both sides of the duodenum. Second, if the ventral anlage becomes adherent to the duodenum, the rotating of the duodenum merely stretches the ventral anlage to form a ring. Third, an unusual overgrowth of one of the ducts may lead to complete encirclement of the duodenum (Fig. 6).

In this condition there is vomiting, upper abdominal distention and visible persistaltic waves, decreased size and frequency of meconium stools, progressive weight loss, and dehydration. On x-ray there is a dilated stomach and dilated proximal duodenum. Barium studies are not necessary.

5. Another congenital anomaly is *agenesis of the myenteric plexus.* These children have vomiting, constipation and abdominal distention during the first four days of life. The picture is more like a lower bowel obstruction. The x-ray will show fluid levels if taken in the upright position. A barium enema will help locate the gas and will show *normal* and dilated bowel. Although the junction between normal and dilated bowel is usually the point of change from abnormal to normal innervation, one cannot accept the roentgenographic findings or the operative findings as evidence of this junction. There may be a considerable segment of dilated bowel that contains no ganglia. The normal or spastic appearing bowel is the area of abnormality (Fig. 7).

6. In an infant with *meconium ileus* the entire intestine is full of tough and inspissated meconium. The end result is obstruction and fairly often a perforation of the intestinal wall. On autopsy, obstruction of the bile duct, the pancreatic duct or both is a finding. Any child that has this probably has cystic fibrosis of the pancreas. The symptoms are the same as in high obstruction—vomiting, abdominal distention and absence of stools.

Case 1.—B.G.H. was a girl born August 21, 1950. She weighed 2.67 kilograms and had a normal delivery. Her postnatal examination on August 22, 1950, was essentially normal, no vomiting or abdominal distention. Vomiting started August 23, two days after delivery. Atropine sulfate was ordered to be given before feedings and subcutaneous fluids were ordered. The child had a meconium stool on August 23, 24 and 25. No more stools were recorded to the date of surgery, August 31.

Fluoroscopic examination on August 28 (seven days after birth) showed a barium swallow progress to the mid-duodenum. The impression was partial obstruction on the basis of a volvulus. Some of the barium passed through. A barium study of the colon showed incomplete rotation of the colon with the major portion of the colon in the left side of the abdomen. An operation, done August 31, revealed the cecum and ascending colon in the left upper quadrant, wrapped around the root of a midgut volvulus in a 360 degree turn.

There were transduodenal bands extrinsic in location across the third and fourth portions of the duodenum. There were a few additional bands over the proximal jejumen. After untwisting the bowels and cutting the bands, the wound was closed. The child is now two years and two months old and doing well.

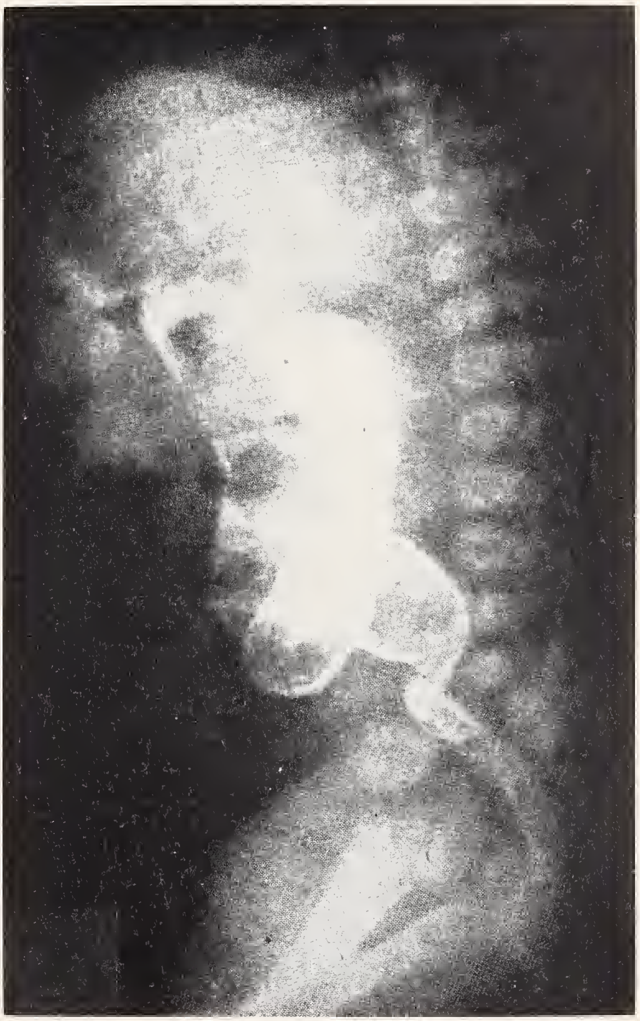

Fig. 7. Small barium enema illustrates narrow spastic rectum and rectosigmoid with dilated bowel above. (Reprinted by permission of May, Charles D., editor of *Pediatrics.*)[5]

Case 2—B.G.T. was a girl born October 15, 1950. The child was cyanotic at birth but pinked up rapidly with oxygen. She weighed 2.14 kilograms. One thing that was misleading to the doctor who saw the case originally is that the child did not vomit apparently large amounts, although the weight loss was 373 grams in four days. The explanation of the peculiar type of vomiting was discovered when the baby was lavaged and 300 cc. of fluid was removed from the stomach. On October 20, 1950, the baby was dehydrated, jaundiced, lethargic and had a distended abdomen. The x-ray showed a marked distention of the stomach with fluid and gas compatible with a high grade obstruction in the region of the pylorus. An operation done the same day, October 20,

(Continued on Page 182)

ACH

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- Sinusitis
- Gonorrhea
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- Mixed Bacterial Infections
- Soft Tissue Infections
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Blood Volume Studies In Gastrointestinal Hemorrhage

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Minneapolis, Minnesota

EVEN THOUGH standardization of diagnostic procedures and therapy is a goal sought after by most of us in the medical profession, it should not be assumed by any of us that similar lesions should be handled in the same manner by all surgeons anymore than we should assume that all surgeons have equal facilities and skill. This fallacy seems to be especially prevalent in the discussions of the management of gastrointestinal bleeding. The procedure of choice in each case, whether in diagnosis or treatment, is that which yields the best results in that surgeon's hands. An attempt will be made here to bring out a point which should better assist one in evaluating the condition of a gastrointestinal bleeder.

We have all seen patients with gastrointestinal bleeding who we know have bled as much as one-third or one-half of their total blood volume and still do not exhibit the classical signs of shock as we know it. On the other hand, why are some patients with considerably less bleeding in profound shock when we see them. The answer, in most cases, appears to be in the total blood volume and red cell mass studies before the bleeding starts and after we see that patient.

TABLE I. NORMAL VALUES FOR A 70 KG. MALE WITH A HEMOGLOBIN OF 16 GM AND A HEMATOCRIT OF 46 PER CENT.

Blood Volume	Weight in Kg. x 85	6000 *cc*
Plasma Volume	Weight in Kg. x 45	3200 *cc*
Red Cell Mass	Weight in Kg. x 40	2800 *cc*
Total Hemoglobin	16x $\frac{6000}{100}$	960 Gms.

46% of 6000—2800 *cc* red cell mass
6000—2800 *cc*——3200 *cc*——plasma volume

TABLE II. VALUES FOR A 70 KG. MALE WHO HAS HAD A SUDDEN LOSS OF ONE-THIRD OF HIS BLOOD VOLUME BY HEMORRHAGE.

Blood Volume	4000 *cc*
Plasma Volume	2133 *cc*
Red Cell Volume	1867 *cc*

Hemoglobin—16 Gms/100 *cc*—640 Gms total.
Hematocrit—46%.

TABLE III. VALUES AFTER EXCHANGE OF FLUID BETWEEN VASCULAR BED AND INTERSTITIAL SPACES.

Blood Volume	5000 *cc*
Plasma Volume	3133 *cc*
Red Cell Volume	1867 *cc*
Total Hemoglobin	640 Gms.

Hemoglobin per 100 *cc*=12.8 Gms.
Hematocrit—37.3%.

Table I shows the normal values for blood volume, red cell mass and plasma volume in a 70 Kg. male with a hematocrit of 46 per cent and a hemoglobin of 16 gm. If this man should suddenly lose one-third of his blood volume by hemorrhage, he would then have remaining the values shown in Table II. This total of 4000 cc. blood volume is only theoretical because we know that along with the vascular constriction that takes place, there is also an exchange of fluid from the interstitial spaces into the vascular bed so that we would have a volume of, say, 5000 cc. The number of red cells is decreased by one-third so that the hemoglobin and hematocrit are lowered.

Table III shows how this affects the plasma volume, hemoglobin and hematocrit. This hemoglobin of 12.8 gm. and hematocrit of 37.3 per cent are not far off the normal and may not cause us much alarm, especially if we think the bleeding has stopped. However, most of the patients with gastrointestinal hemorrhage are still bleeding when we see them and may continue to bleed for some time during our care. Also we know that a good percentage of these patients will bleed again, and each time they bleed there is a further loss of total hemoglobin from the circulating blood.

In a similar manner the circulating proteins will be reduced from a normal figure of 6.0 gm. per 100 cc. blood to a corresponding low after the volume is reduced and then diluted as it increases. Table IV shows the effect on total plasma proteins.

From these figures on a hypothetical case, it can be readily seen that further hemorrhage at this

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Read before the Minneapolis Academy of Medicine, March 16, 1954.

TABLE IV. PROTEIN VALUES BEFORE AND AFTER THE HEMORRHAGE.

Total Proteins ....6.0 Gms./100 *cc*

or $\frac{\text{Plasma Volume}}{100} = \frac{3200 \times 6}{100} = 198$ Gms. total protein.

After the bleeding

$\frac{2133}{100} \times 6 = 128$ Gms. total protein.

After increase of plasma volume to 3133 *cc* the total protein is still 128 Gms. or 4.1 Gms./100 *cc*.

time or even after inadequate transfusions of whole blood will seriously jeopardize this patient's life. This is especially true if surgery is contemplated on this patient because with the added trauma of surgery, anesthesia and surgical hemorrhage, the seriousness of this situation could become alarming.

Of equal importance in the therapy of hemorrhage is the condition of that patient with respect to blood volume and red cell mass before he starts bleeding. These patients may have depleted values in blood volume as a result of nutritional disturbances, heart disease, chronic infection, slow chronic bleeding or malignancy. For purposes of simplification it might be said that this type of patient is suffering from "chronic shock."

This chronic shock has been defined as a syndrome characterized by weight loss, diminished blood volume, increased interstitial fluid volume and an increased susceptibility to shock from anesthesia, surgical trauma, or a physiological shock condition such as hemorrhage.

It is readily apparent from the above example of change in blood volume, red cell mass and plasma proteins that profuse bleeding is much more likely to cause the patient to fall into a shock-like state than a normal healthy individual.

The total protein will be especially affected because during this subpar state the patient is continually using his supply of stored protein and in any emergency he would not have this supply to fall back upon in order to maintain an adequate plasma protein value.

From this, it is quite obvious that the patient with rapid bleeding will show close to normal hemoglobin, hematocrit and plasma protein values. However, in the patient with the same amount of bleeding over a longer period of time, these values are lower than normal. In this type of patient, the plasma volume will be near normal and on receiving transfusions, this plasma volume changes very little. The mechanism of this phenomenon is that there is an exchange of fluid between the circulating blood and the extracellular spaces. In this case the goal should be replacement of the red cell mass and not the blood volume.

The test most used at this time for the determination of the plasma volume is the Evans blue dye method. This involves the intravenous injection of the dye. After ten minutes a specimen of blood is obtained from the opposite side of the body. No tourniquet is used. The plasma from the centrifuged blood is then compared by a colorimetric method with a photo-electric spectrophotometer. This method has been shown to be accurate to within 3 per cent. Another test which has been used more recently is the use of tagged radioactive ions. This is thought to be more accurate than the Evans blue dye method but for all practical purposes the dye method is probably accurate enough.

In conclusion, I would like to leave the following facts to be noted whenever one is confronted with a patient on whom surgery is contemplated and who has experienced hemorrhage either slow or fast or, for other reasons mentioned, presumably the plasma volume or red cell mass is altered.

1. The hemoglobin and hematocrit levels and other clinical signs or lack of signs of shock are not accurate guides to severity of hemorrhage.

2. The red cell mass is a more important determination to know than the hemoglobin and hematocrit in order that a true evaluation of the patient's condition may be known.

3. Because there are shifts in fluids between the plasma and the interstitial space and also at the same time shifts between the interstitial space and the intercellular space, it would be better to defer surgery in many cases until adjustment has been reached.

4. Because the shift of these fluids is not a rapid process but is not complete for at least twenty-four hours, it is probably better to defer surgery until the patient has had time to adapt to his increased blood volume and to his electrolyte fluid balance. Also because of this, transfusion should not be pushed too rapidly unless the patient is in clinical shock. There is always the danger of overloading the circulation and throwing the patient into cardiac failure with pulmonary edema and the subsequent complications.

408 Physicians and Surgeons Building

Gynecological Cancer Detection

Findings at University of Minnesota Cancer Detection Center

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and
MAXWELL M. BARR, M.D.
Minneapolis, Minnesota

IN THE PAST two decades increasing emphasis has been placed on the early detection of carcinoma. The importance of periodic examinations and the recognition of early symptoms have been the basis of educational programs directed at the medical profession as well as the lay public. As a by-product of these efforts, over 133 cancer detection centers have been established in this country since 1937.[1,8] The Cancer Detection Center at the University of Minnesota was established in March, 1948, with the approval of the Minnesota State Medical Association. Examinations have been limited to men and women over the age of forty-five years. The scope of the examinations and the results obtained have been reported elsewhere.[5,7,10]

This report deals with the results of gynecological examinations and special diagnostic procedures performed upon 3,087 women from March, 1948 to October 6, 1953. During this period the following routine has been used for the systematic examination of the female pelvis: (1) A brief history is obtained to elicit the presence of abnormal bleeding or discharge. (2) The lower abdomen is inspected and palpated. (3) The external genitalia are inspected and palpated. (4) Vaginal secretion is aspirated for the vaginal smear. (5) Speculum examination of the vagina and cervix is performed and a cervical smear is taken with an Ayres' wooden spatula. Since 1950 an additional routine has included the insertion of a small piece of gelfoam into the endocervix. If bleeding results the gelfoam is placed in formalin and sent to the pathology laboratory for preparation of sections similar to tissue biopsy. The slide is examined by the pathologist for the presence of abnormal cells in the interstices of the gelfoam as described by Gladstone.[3] (6) Bimanual examination and recto-vaginal examinations are performed.

In addition to the above routine, the following procedures are performed when indicated: (1) Punch biopsy of the cervix is taken when an abnormal lesion or bleeding area is found. When the Papanicolaou studies are reported positive or suspicious, repeated smears are made. If these are positive, biopsies are taken from target areas indicated by Schiller's Gram iodine test. If target areas are not found, biopsies are taken in the four quadrants of the cervix, or a cone of cervical tissue is removed with a Gusberg cone biopsy instrument. (2)) Small cervical polyps are avulsed with Bozeman uterine dressing forceps or the Pratt forceps. When a history of abnormal bleeding is obtained, the patient is referred back to her own physician for dilatation and curettage. (3) When cervical smears are positive but biopsies of the cervix fail to reveal carcinoma, the patient is referred to her physician for dilatation and curettage and either repeat biopsies or knife conization of the cervix. (4) When other lesions or pelvic masses are found, the patient is referred to her physician for further observation or therapy as indicated. (5) Endometrial aspiration smears have more recently been made on patients with abnormal uterine bleeding or discharge in an attempt to improve the screening of patients with endometrial carcinoma. Figure 1 illustrates some of the instruments used for routine and special procedures during the gynecological examination.

The vaginal and cervical smears are stained and

Presented before the Minneapolis Academy of Medicine, April 20, 1953.

This material includes examinations through October 6, 1953, or five and one-half years of operation of the center. The authors wish to acknowledge with thanks the co-operation of Dr. Claude R. Hitchcock, director of the center, in making the records and facilities available for this study. Mrs. Lydia Linsey has been most helpful in assembling data necessary for this report.

The authors have attended the center as gynecological examiners since its inception. The Cancer Detection Center is sponsored by a grant from the Minnesota Division of the American Cancer Society.

TABLE I

	Number	Per cent
Female patients (all over age 45)	3,087	
Total examinations	6,840	
Cervical biopsies	419	13.5
Gelfoam saved	563	
Pap. smears	10,334	
Cervical polyps	193	6.2
Carcinomas found in polyps	2	
Carcinomas found in cervical biopsies	6	
Total—carcinoma of the cervix	8	0.26

TABLE II

	Number	Per cent
Total Pap. smears	10,334	
Total patients	3,087	
Total examinations	6,840	
Class 3 smears	109 smears in 72 patients	
Class 4 smears	14 smears in 10 patients	
False pos. class 3	68 patients	
False pos. class 4	3 patients	
Total false pos.—classes 3 and 4	71 patients	2.3
Total false negative	3 patients	0.1

TABLE III

	Number
Normal	2
Cystic nodule	1
Friable polyp	1
Roughened post. lip	1
Bleeding spot	1
Polypoid tissue	1
Localized granular erosion	1

examined by the technicians in the laboratory of the Cancer Detection Center. Suspicious smears are examined by the pathologist in the University Hospitals for final classification. Technician screening has resulted in a considerable saving of valuable time on the part of professional personnel.

Cancer of the Cervix

Table I shows the number of special examinations performed in the group of 3,087 patients examined from March 1, 1948, to October 6, 1953. The average number of examinations per patient was 2.2 during this five and one-half year period. One hundred and ninety-three cervical polyps were removed, and malignant degeneration was present in two.

Punch biopsies of the cervix were positive for carcinoma in six women. A total of eight carcinomas of the cervix have been detected among women who have attended the center (.26 per cent).

The experience with Papanicolaou smears is shown in Table II. The smears have been classified as follows: Class 1, normal appearing epithelium; Class 2, excessive number of pus cells; Class 3, atypical epithelial cells which are not frankly anaplastic in appearance; Class 4, frankly anaplastic appearing cells.

The relatively high percentage of false positive smears should decrease with increasing experience in reading the smears. Graham and Meigs[4] have shown that their group was able to reduce the percentage of false positive smears from 6.4 per cent to .04 per cent in a period of five years.

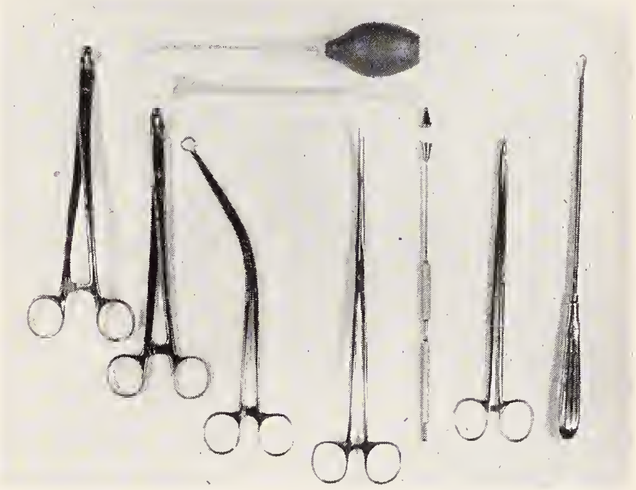

Fig. 1. Instruments used for detection of carcinoma of the internal female genitalia. Left to right are Thoms modification of the Gaylor biopsy punch, Gaylor biopsy punch, single tooth tenaculum, uterine dressing forceps holding gelfoam, Gusberg cone biopsy, Pratt forceps, and a small sharp curette. Above are a glass canula for aspirating vaginal secretion and the Ayres wooden cervical spatula.

The appearance of the cervix in the group of eight women with carcinoma of the cervix is described in Table III.

These findings indicate the lack of any characteristic gross appearance that will definitely identify early carcinoma of the cervix. Two patients with normal appearance of the cervix were detected with Papanicolaou smears graded Class 4. The smear diagnosis was confirmed by biopsy in each case.

Case 1.—M.C.O., aged sixty-two, para 3. At her third visit to the center her vaginal smear was found to be Class 4. Previous smears were Class 1. The cervix appeared normal. The vaginal smear (Fig. 2) revealed a typical anaplastic appearance. A biopsy obtained by her physician revealed early squamous cell carcinoma with no evidence of invasion.

Case 2.—P.F., aged fifty-four, para 2. At her first visit to the center her cervix appeared normal but the vaginal smear was Class 3. Repeat smears over a three-month period revealed Class 3 and later Class 4 smears. Schiller's test was positive around the circumference of

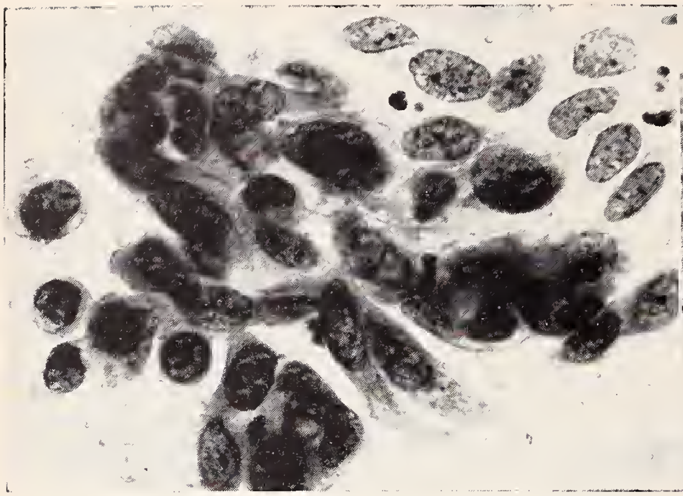

Fig. 2. Vaginal smear obtained from Case 1. Normal appearing cervix. Smear is Class 4. Biopsy taken elsewhere revealed squamous cell carcinoma.

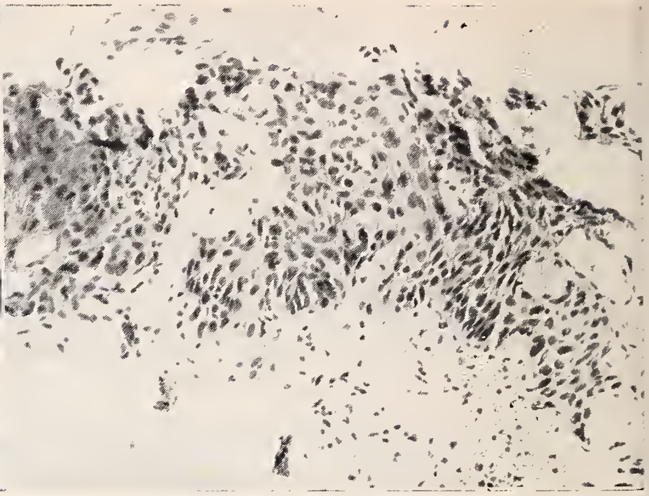

Fig. 3. Biopsy of cervix from Case 2. Normal appearing cervix. Positive Schiller's test after Class 4 smears. Gusberg cone biopsy reveals invasive squamous cell carcinoma.

the vaginal cervical junction. A biopsy was obtained with the Gusberg cone punch and was reported as showing invasive carcinoma of the cervix (Fig. 3).

The remaining six cases of carcinoma of the cervix were diagnosed by biopsy of abnormal lesions of the cervix. However, the routine Papanicolaou studies revealed Class 4 smears in three patients, a Class 3 smear in one patient, and normal smears in one patient. Figure 4 shows a Class 4 cervical smear obtained from a fifty-three-year-old woman, para 2. Biopsy of a small localized granular erosion of the cervix revealed early squamous carcinoma (Fig. 5). In six of the eight patients with positive cervical biopsies no evidence of invasion was found in the tissue examined. The following case is of interest because it was detected only by a set of fortunate circumstances:

Case 3.—E.A., aged forty-five, para 3. At her second visit to the center a biopsy was taken of a small area of the cervico-vaginal junction that bled easily. The tissue was examined by two pathologists, both of whom diagnosed chronic cervicitis. One report noted the presence of a few suspicious looking cells at the margin of the tissue section. Repeat biopsy was requested and was done two weeks later, several small bites being taken from the site of the previous biopsy. This area was located easily because the first biopsy site had been sketched by the examiner. The diagnosis now was definitely squamous cell carcinoma of the cervix (Fig. 6).

The gelfoam biopsy was saved and examined in five of the eight women found to have carcinoma of the cervix. These were interpreted as positive for carcinoma in one patient (Fig. 7), suspicious in three patients, and negative in one. The gelfoam test was not responsble for detecting carcinoma of the cervix but was found confirmatory in four of the five patients in whom the test was used.

Carcinoma of the Endometrium

Two women were found to have carcinoma of the corpus uteri during the first five and one-half years of the detection center's operation. Both presented the history of postmenopausal bleeding and both were referred to their own physicians for diagnostic curettage, which revealed the lesions. In both patients the pelvic findings were normal to bimanual examination and the surgical specimens revealed well localized lesions. Vaginal and cervical Papanicolaou smears were negative in both cases. This experience is similar to that at other centers and has led to the use of endometrial smears as a screening method.[9] Our experience with the endometrial smear is too recent for evaluation.

Carcinoma of the Ovary

Adnexal masses were palpated in 100 women. Of these, forty-five patients are known to have had pelvic laparotomy. Five patients were found to have carcinoma of the ovary. Four of these women were asymptomatic, and one complained of pelvic pain. Cystadenocarcinoma was found in four women, and one had primary adenocarcinoma. On September 1, 1954, four women were alive at four and one-half, three and one-half, three, and two years after surgery, and one patient had expired. Although cure is the excep-

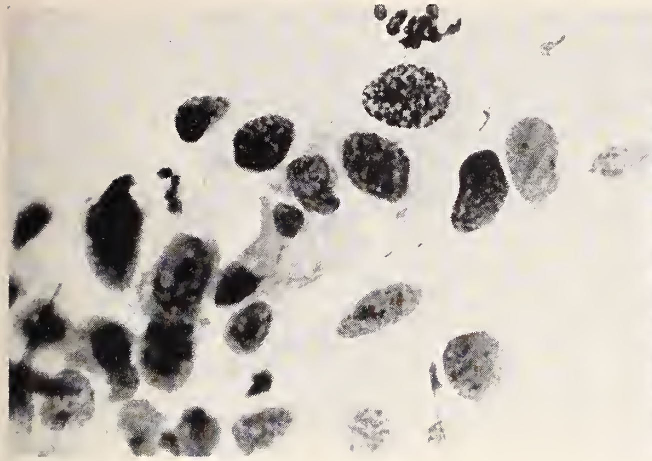

Fig. 4. Cervical smear, Class 4, from cervix with small localized granular erosion.

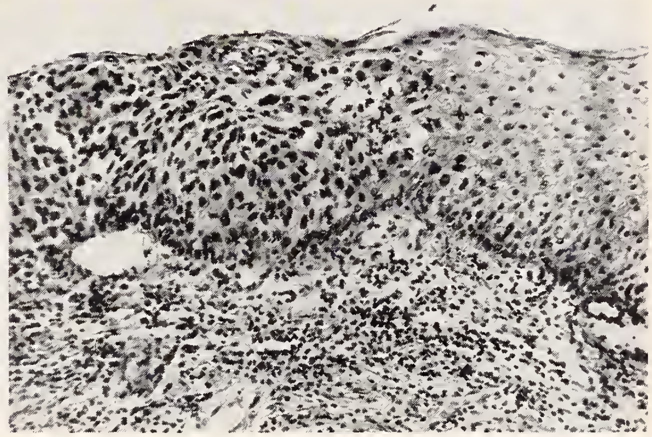

Fig. 5. Biopsy of small granular erosion shows early squamous cell carcinoma. Same woman as in Figure 4.

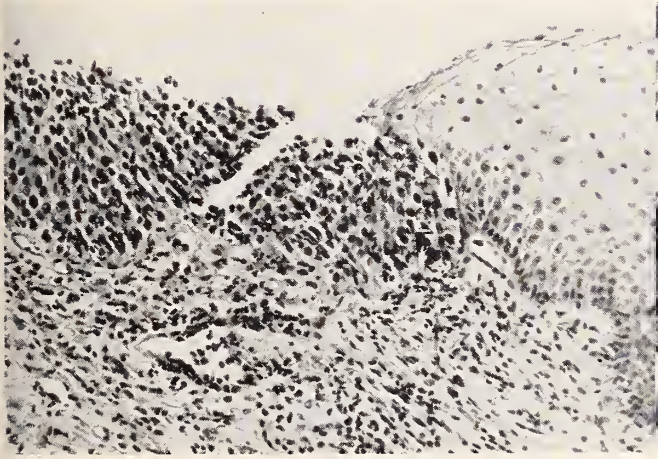

Fig. 6. Biopsy from small bleeding area of cervix repeated at request of pathologist. Squamous cell carcinoma.

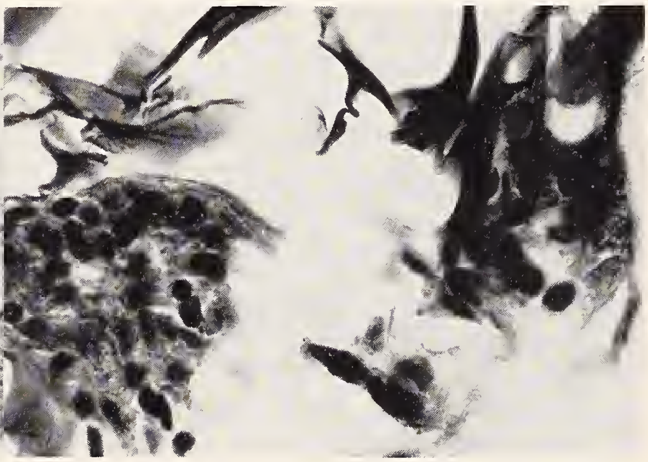

Fig. 7. Gelfoam biopsy of cervix from patient with small cystic nodule of cervix found to contain invasive squamous cell carcinoma.

tion, the early detection of carcinoma of the ovary offers an opportunity for adequate surgical therapy and prolongation of life.

Precancerous Lesions of the Female Pelvis

Probably the only disease process in the female pelvis that is associated with carcinoma in a significant percentage of women is leucoplakic vulvitis. This condition, as its name implies, presents a characteristic white thickening of the vulvar skin which is often confused with an exaggerated aging process of the vulvar skin known as kraurosis vulvae or, literally, shrinkage of the vulva. Kraurosis carries no increased threat of carcinoma while leukoplakia must be observed for the development of ulcers, fissures and induration of the involved skin. Biopsy will reveal the onset of carcinoma of the vulva. Patients with leukoplakic vulvitis have been referred to their physicians with the recommendation that they be examined every six months and biopsies taken as indicated. No carcinoma of the vulva has been detected at the center to date.

Myomata of the uterus may become sarcomatous in a small but definite percentage of women. The incidence of sarcomatous degeneration has usually been reported between 0.5 per cent and 1.0 per cent of all women with myomata.[11] In the general population this figure may be as low as 0.2 per cent. Sudden growth of these tumors, especially after the menopause, should indicate the danger of this complication.

Chronic cervicitis, leucoplakia of the cervix, cervical polyps and urethral caruncles are not

TABLE IV

	Number	Per cent
Skin	18	
Breast	7	
Stomach	5	
Sigmoid	3	
Rectum	3	
Cecum	2	
Duodenum	1	
Thyroid	1	
Lung	1	
Leukemia	1	
Lymphosarcoma	1	
Total Extrapelvic	43	1.39
Total Pelvic	15	.48
Total malignancies 3,087 women	58	1.87

precancerous but may mask or be simulated by the appearance of early carcinoma. Figures available for the first four years of operation of the center reveal that 381 women (17.5 per cent) of the female population attending the center have had hysterectomies. The cervical stump was left in 275 women or 12.7 per cent of the total. Neither carcinoma in the cervical stump nor sarcomatous degeneration of a myoma has been found in any detection center patient.

Extrapelvic Cancer

Carcinoma was detected outside of the pelvis in forty-five women during the five and one-half years of this study. The location and number of these malignant tumors or conditions are indicated in Table IV.

Discussion

During the first five and one-half years of operation of the University of Minnesota Cancer Detection Center, 3,087 women over the age of forty-five years received 6,840 examinations. Eight women were found to have carcinoma of the cervix, two had carcinoma of the endometrium, and five had carcinoma of the ovary. Extrapelvic carcinoma was found in 43 women.

Biopsy of abnormal appearing areas of the cervix was responsible for detecting six of the eight carcinomas of the cervix. Cytological smear study was responsible for detecting two carcinomas of the cervix. Although the Papanicolaou studies have proved to be an important means of screening for carcinoma of the cervix, the current shortage of technicians capable of screening smears makes general usage of this method impracticable except in large centers of population. The gelfoam method of cytological study has not been responsible for detecting carcinoma but has indicated the need for further study in four of five cases. Carcinoma of the endometrium has been detected only by eliciting the history of abnormal bleeding and histologically proving the lesion after dilatation and curettage. Endometrial aspiration smears give promise of effective screening of carcinoma of the endometrium.

The follow-up of women who have attended the Cancer Detection Center is accomplished by contacting all examinees once each year in regard to returning for check-up. In this manner close contact is maintained with greater than 90 per cent of the center population. Up to the time of this report no examinee who was found to be normal on gynecological examination has been found on follow-up to have developed pelvic malignancy, with the possible exception of one patient who was subsequently found to have generalized lymphosarcoma of the abdominal viscera at laparotomy.

The accuracy of the screening pelvic examination to date would indicate that careful periodic examination and biopsy of all suspicious lesions will detect carcinoma of the vulva, vagina, cervix and ovaries in a high percentage of affected women. The Cancer Detection Center is valuable for testing and teaching new methods but cannot substitute for the doctor's office in reaching the bulk of the population.

Conclusions

1. Carcinoma of the cervix can be detected early by careful periodic examination and biopsy of all suspicious lesions. Vaginal and cervical smears are an important adjunct for early detection. Gelfoam biopsies have not proved valuable in this study.

2. Carcinoma of the endometrium can be detected early if women are educated to see their physicians for abnormal bleeding and the physicians follow through with adequate differential curettage. Endometrial aspiration smears offer better promise of effectively screening carcinoma of the endomerium than do vaginal and cervical smears.

3. Carcinoma of the ovary can be detected early only by periodic pelvic examinations and exploratory laparotomy for all newly developed and enlarging adnexal masses.

4. Carcinoma of the vulva is rare but will be detected early by biopsy of all suspicious lesions of the vulva particularly when the lesions are associated with leukoplakic vulvitis.

5. Significant reduction in mortality rates will

(Continued on Page 188)

Ectopic Pregnancy

Diagnostic Analysis of Seventy Cases

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DURING the thirteen and one-half years from January, 1941, to July, 1954, there were seventy patients admitted to the Minneapolis General Hospital with the established diagnosis of ectopic pregnancy. In 1942 Dr. William P. Sadler presented an analysis of the 102 cases of ectopic pregnancy admitted to Minneapolis General Hospital during the nine-year period, 1932 to 1940. The current study analyzes the seventy cases from the standpoint of admission status, symptoms, and preoperative laboratory and physical findings; however, a more complete study, comparable to that of Dr. Sadler, is in the process of formulation.

TABLE I. SEVENTY ECTOPIC PREGNANCIES. MINNEAPOLIS GENERAL HOSPITAL, JANUARY 1, 1941, THROUGH JULY 1, 1954

Age		Para		Race		Marital Status		Amenorrhea	
Under 20	5 patients	0	14	White	58	Married	50	None	2
20-25	12	1	21	Colored	10	Divorced	11	4 weeks	3
25-30	26	2	12	Indian	2	Single	6	5-8 weeks	27
30-35	17	3	10			Widowed	3	8-12 weeks	27
35-40	8	4	6					12 or more	9
40 and over	2	5	5					Unknown	2
		6	0						
		7	1						
		Unknown	1						

The ages of these patients varied from seventeen to forty-two. Table I shows that the greatest number were in the 25- to 30-year age-group (twenty-six patients) with seventeen patients in the 30- to 35-year age-group. The racial distribution was: fifty-eight white patients, ten negroes and two Indians. Marital status was as follows: fifty married, eleven divorced, six single and three widowed. The parity, when known, varied from 0 to 7, with the largest number in the para 1 classification (twenty-one cases), and approximately equal distribution in the groups para 0, 2, and 3.

The record of amenorrhea was quite typical of ectopic pregnancy, with two patients reporting no amenorrhea, three with amenorrhea lasting four weeks, twenty-seven lasting five to eight weeks, twenty-seven lasting eight to twelve weeks, nine lasting twelve or more weeks, and two unknown.

The histories relative to vaginal bleeding prior to admission are shown in Table II. There was no bleeding in twelve cases, only one episode of bleeding in two cases, abnormal menses only in six cases, spotting only in seventeen cases, intermittent moderate bleeding in nine cases, continuous moderate bleeding in eighteen cases, profuse bleeding in seven cases, small clots passed in four cases, large clots passed in five cases, apparent tissue passed in six cases, and no history in one case.

TABLE II. VAGINAL BLEEDING AND PAIN PRIOR TO SURGERY

Bleeding		Pain	
None	12	None	1
One episode only	2	Shoulder pain	24
Abnormal menses only	6	Dull pelvic ache only	5
Spotting only	17	Abdominal cramping	18
Intermittent	9	Sharp pain with fainting	18
Continuous moderate	18	Sharp pain without fainting	41
Profuse	7	Rectal pain and or pain on defecation	17
Small clots	4	Dysuria	6
Large clots	5	Epigastric pain	5
Apparent tissue passed	6	Nausea and or vomiting	43
No history	1	No history	1

Type of pain experienced was also widely variable. The largest group, forty-one, reported sharp abdominal pain without syncope, and a substantial group, eighteen, reported sharp pain with syncope. Abdominal cramping alone or associated with

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Presented before the Minneapolis Academy of Medicine, October 18, 1954.

other pain was reported by eighteen patients. Shoulder pain, in one or both shoulders, and always associated with some other type of pain, was reported by twenty-four patients. Nausea and/or vomiting, associated with pain or bleeding episodes, was reported by forty-three patients.

TABLE III. ECTOPIC PREGNANCIES ADMISSION STATUS

Temperature	Cases	Pulse Rate	Cases	Systolic Blood Pressure	Cases	Clinical Shock	Cases
Below 99° F	27	Below 80	8	Below 80 mm Hg	9	On admission	19
99-100°	34	80- 90	25	80- 90	5	One profound died 20 minutes after admission	
100-101°	7	90-100	9	90-100	2	Shock subsequent to admission	3
101-102°	1	100-110	8	100-110	10	No observation of clinical shock	48
Not recorded	1	110-120	2	110-120	12		
		120-above	10	120-130	17		
		Imperceptible	3	130-140	12		
		Not recorded	5	No record	3		

TABLE IV. ADMISSION BLOOD STUDIES

Hemoglobin	Cases	White Cell Count	Cases	Hemoglobin Drop	Cases
Below 40%	2	Less than 9,000	18	Less than 5%	21
40-50	8	9-10,000	9	5-10	16
50-60	16	10-11,000	2	10-15	12
60-70	21	11-12,000	9	15-20	6
70-80	8	12-13,000	3	20-25	9
80-90	14	13-14,000	7	25-30	3
Not recorded	1	14-15,000	3	30-35	0
Sedimentation rate done	26	Over 15,000	18	40%	2
Elevated	16	Unknown	1	Not recorded	1
Not elevated	10				

Rectal pain and/or pain on defecation reported by seventeen patients, is understandable when the findings of cul-de-sac changes are noted. A relatively small number of patients reported no pain, dull pelvic ache only, pain associated with urination, or epigastric pain.

Abdominal examination revealed tenderness of some kind in sixty-eight instances, rebound tenderness occurring in fifty-eight of these. Abdominal distention was present in fifteen instances, and rigidity in twenty-three instances. Abdominal mass or masses were recorded in eight instances. Pelvic examination revealed adnexal mass or masses in thirty-eight patients. In most of these reports, the masses are described as very tender. Adnexal tenderness without definite masses was reported in seventeen patients. In many patients, masses could not be outlined because of extreme tenderness. Cul-de-sac fullness or masses were described in thirty-four patients, and cul-de-sac tenderness without fullness in nine patients. Painful cervix was reported in forty patients, the most frequent description being extreme pain on movement of the cervix. The cervix was reported as soft and/or bluish in twenty-seven patients. The uterus, when palpable, was described as enlarged in twenty-one patients, and tender in nineteen patients.

Admission temperature was recorded in sixty-nine cases. It was found to be below 90 degrees F. in twenty-seven cases, 99 to 100 degrees in thirty-four cases, 100 to 101 degrees in seven cases, and 101 to 102 degrees in one case. The admission pulse rate, recorded for sixty-five patients, was below 80 in eight patients, 80 to 90 in twenty-five, 90 to 100 in nine, 100 to 110 in eight, 110 to 120 in twenty-two, above 120 in ten patients, and three patients were reported to have imperceptible pulses. Systolic blood pressure was recorded on admission in sixty-seven instances. The pressure was below 80 mm. Hg in nine instances, 80 to 90 in five instances, and 90 to 100 in two instances. The remainder of the admission readings varied from 100 to 140 mm. Hg. Clinical shock was noted on admission in nineteen patients; one patient, admitted in profound shock, expired twenty minutes later. Subsequent clinical shock developed in three patients who were not admitted in shock.

Admission hemoglobin determinations are reported in per cent since that is the method used at the hospital during most of the period covered by this study. Two patients had admission hemoglobin determinations below 40 per cent, eight had 40 to 50 per cent, sixteen had 50 to 60 per cent, twenty had 60 to 70 per cent, eight had 70 to 80 per cent, and fourteen had 80 to 90 per cent hemoglobin. Maximum hemoglobin drop was calculated. Twenty-one patients had less than 5 per cent drop, sixteen had 5 to 10 per cent, twelve had 10 to 15 per cent, six had 15 to 20 per cent, nine had 20 to 25 per cent, three had 25 to 30 per cent, and two had 40 per cent hemoglobin drop.

The admission white cell count varied widely. Eighteen patients had a count of 9,000 or less,

TABLE V. PREVIOUS PELVIC SURGERY AND PELVIC INFLAMMATORY DISEASE

Pelvic Surgery	Cases	Pelvic Inflammatory Disease	Cases
None	42	History only	1
Tubal ligation (Madelaner)	1	Findings only	22
Appendectomy	18	History and findings	19
Tubal pregnancy	6	No history or findings	28
Salpingectomy (indication?)	1		
Dilatation and curettage	4		
Uterine suspension	1		
Bartholins cyst	1		
Vaginal plastic	2		
Cesarean section	1		

TABLE VI.

Diagnostic Tests	Cases	Time Interval Admission to Surgery	Cases	Transfusion Prior or During Surgery	Cases
Posterior colpotomy or colpopuncture Positive findings	20	Less than 3 hours	13	None	28
Equivocal	1	3- 5	8	500 cc. or less	15
Negative	5	5- 7	5	500-1000	13
Culdoscopy	1	7- 9	3	1000-1500	4
Freidman test positive	19	9-11	2	1500-2000	5
		12-24	8	2000-2500	0
		24-36	3	2500-3000	4
		36-48 hours	1	4000 cc.	1
		48 hours to 16 days	26		
		One died prior to surgery	1		

TABLE VII. PHYSICAL FINDINGS

Abdominal		Pelvic	
Tenderness	68	Adnexal mass or masses	38
Distention	15	Adnexal tenderness without masses	17
Rebound	58	Cul-de-sac fullness or mass	34
Rigidity	23	Cul-de-sac tenderness without fullness	9
Abdominal mass or masses	8	Tender cervix	40
		Soft or changed cervix	27
		Tender uterus	19
		Enlarged uterus	21

nine patients had 9,000 to 10,000, two patients had 10,000 to 11,000, nine patients had 11,000 to 12,000, three patients had 12,000 to 13,000, seven patients had 13,000 to 14,000, three patients 14,000 to 15,000, and eighteen patients had a count of over 15,000. Factors influencing white cell count and hemoglobin were dehydration, blood loss and administration of intravenous fluids, plasma and blood.

Twenty-eight patients had had lower abdominal or pelvic surgery previous to the onset of the present illness. There had been one tubal ligation (Madelaner), eighteen appendectomies, four dilations and curettages, one uterine suspension, one excision of a Bartholin cyst, two vaginal plastic operations, one cesarean section, and six procedures for ectopic pregnancy, and one salpingectomy for reasons other than ectopic pregnancy.

History and/or evidence of pelvic inflammatory disease was found in forty-two patients. There were histories and operative findings of pelvic inflammatory disease in nineteen patients, operative findings only in twenty-two patients, and history but no operative findings in one patient. In some cases there was operative description of pelvic inflammatory disease without any pathological confirmation.

There were nineteen positive Friedman tests, and several cases in which the test results were not recorded. Posterior colpotomy or colpopuncture was positive in twenty cases, equivocal in one, and negative in five cases. Culdoscopy was done once.

Dilation and curettage was carried out on twelve patients. Decidual tissue was obtained six times, hyperplastic endometrium twice, proliferative endometrium twice, secretory endometrium once and insufficient tissue for diagnosis once.

The time interval from admission to surgery varied from less than one hour to sixteen days. The interval was less than 3 hours on thirteen occasions, 3 to 5 hours in eight cases, 5 to 7 hours in five cases, 7 to 9 hours in three cases, 9 to 11 hours in two cases, 12 to 24 hours in eight cases, 24 to 36 hours in three cases, 36 to 48 hours in one case, and 48 hours to 16 days in twenty-six cases. One patient expired twenty minutes after admission. Several patients were discharged and re-admitted prior to surgery.

Summary

1. A review of the symptoms and diagnostic findings of seventy ectopic pregnancies has been presented. This series is not to be considered as typical of ectopic pregnancy, but of ectopic pregnancy as encountered in indigent patients at the Minneapolis General Hospital.

2. The percentage of patients admitted in shock was almost identical in the two Minneapolis General Hospital series: 27.1 per cent in this series, and 27.4 per cent in Dr. Sadler's series.

3. Of these seventy ectopic pregnancies, 60 per cent (forty-two cases) were complicated by or associated with pelvic inflammatory disease in some manner. This particular phase of the study cannot be compared with Dr. Sadler's series because he included only those diagnoses of inflammatory disease verified by tissue study, whereas this study also includes diagnoses based on operative findings alone.

4. The high incidence of recurrent ectopics

(six cases) (8.6 per cent) may further illustrate the increased incidence of ectopic pregnancy in the inflammatory pelvis.

5. Study of the data on vaginal bleeding reveals wide variation with no predominant pattern; however, fifty-eight of the seventy patients (83 per cent) presented some type of abnormal bleeding associated with pain.

6. The high incidence of rebound tenderness, distention, cul-de-sac masses, adnexal masses and shoulder pain illustrates the marked pathologic processes in this series.

7. Admission hemoglobin levels and drops in hemoglobin were undoubtedly influenced by dehydration, blood loss and transfusions administered.

8. The long interval between admission and surgery in twenty-six cases illustrates the need for more rapid diagnosis. Culdoscopy might have shortened this interval, but this procedure must be undertaken with great care in the inflammatory pelvis.

Note: Final diagnosis recorded on these patients: ruptured tubal pregnancy, forty-eight; tubal pregnancy with probable rupture, nine; aborting or aborted tubal pregnancy, five; unruptured tubal pregnancy, seven; ectopic pregnancy, probable ovarian, one.

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OBSTRUCTION OF THE BOWEL IN THE NEWBORN

(Continued from Page 169)

revealed that distal loops of bowel beyond the ligament of Treitz were completely collapsed. There was meconium in the colon. The proximal duodenum was distended and the distal duodenum was collapsed. The diagnosis was annular pancreas. A retrocolic duodenal jejunostomy side-to-side anastomosis was performed. The child died November 11, 1950, twenty-two days after surgery. Autopsy revealed malformation of duodenum, pneumonitis, and necrosis of the brain. The anastomosis was functioning all right.

Summary

Six causes of bowel obstruction in the newborn, together with two case reports, have been presented. Various diagnostic points have been given. However, the important thing is not to differentiate the type of bowel obstruction as to etiology, but as to whether it is a complete obstruction, and whether it is a high or low bowel obstruction. The sooner this is done after birth, the better the prognosis for the child.

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Athletic Accident Benefit Plan, 1954-1955

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IN JANUARY, 1952, a summary and explanation of the Athletic Accident Benefit Plan of the Minnesota State High School League, its limitations, administration, and methods of financing, was presented to the members of Hennepin County Medical Society in their *Bulletin*. At the time of this writing an up-to-date review with new revisions in this program is being announced to all of the member doctors of the Minnesota State Medical Association.

In spite of conscientious efforts by H. R. Peterson and by the State High School League, there resulted some controversies between Minnesota doctors and the league, mostly because of an honest lack of knowledge on the part of either the student athlete and his family or the doctor himself. There was a feeling by the parents that the payments by the league were *full* and satisfactory for medical care of the injury involved. There was an unfortunate misunderstanding by some doctors in the state that an attempt was being made by the state league to set up a fee-schedule for athletic injuries and that payments were too low. Fortunately huge strides have been made to eradicate both of these misunderstood aspects in the program.

The Medical Service Committee of the Minnesota State Medical Association has worked for several years in close co-operation with H. R. Peterson, executive secretary of the Minnesota State High School League, and feels that your committee is very close to a satisfactory solution. During this period, Mr. Peterson and his Board of Control have raised a great number of the benefits paid, especially on the common type of athletic injuries. They have devised a completely new method of perforated forms in order to attempt a more certain method of dispersing the information about the "benefit" plan, not insurance, to the student athlete and his parents. Printed on the Parents Permit Signature Card is the following statement "The undersigned further states that the 'Notice to Athletes and Parents' has been received; and understands the Benefit Plan provides only limited financial assistance; and the balance of any bill, if not paid in full, is a responsibility of the pupil or his parents."

The extremely controversial paragraph in the old explanatory bulletin read, "The plan provides limited coverage only: The schedule of maximum allowances *grants reasonable payments for normal services.*" This has been removed from the new forms and only the following paragraph used: "One of the major services provided by the League is the Athletic Benefit Plan which offers financial assistance in meeting the cost of medical, dental, and hospital services resulting from injuries incurred while participating in athletic activities sponsored by the school. The plan is a non-profit mutual benefit program and not insurance. It provides specific limited allowances for various injuries, and the balance of the bills, if not paid in full, is a responsibility of the athlete or his parents". Your committee feels that this should do much to clarify the apparent misunderstanding between the league and the doctors of medicine in the State of Minnesota.

The financing of this plan is done by charging in the past $2 per year to the boy who participates in football and other sports, and $1 per year if he does not participate in football. This fee was known to be insufficient for sound underwriting but many boys in the state from some of the less fortunate families have been unable in the past to afford even this fee. The deficit of the underwriting was made up from profits of the Minnesota State High School Basketball Tournament each year. Fortunately for the success of the plan, the weather and competition have co-operated to make a financially successful basketball tournament. However, realizing that this was not sound and there could be "many a slip 'twixt the cup and the lip", the Board of

(Continued on Page 214)

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Seminar

DIAMOX

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Diamox (2-acetylamino-1, 3, 4 thiadiazole-5-sulfonamide) is a heterocyclic sulfonamide which was developed by the American Cyanamide Company in the search for a powerful carbonic anhydrase inhibitor related to sulfanilamide. In 1940, Mann and Keilin[17] discovered that sulfanilamide and other compounds with a free sulfonamide group inhibit carbonic anhydrase activity. An amino group in the para position was found necessary for the antibacterial action but was not necessary for the inhibitory effect on carbonic anhydrase (Fig. 1). Following the observation that sulfanilamide induced acidosis with an alkaline urine,[21] Höber[14] suggested that alkalization of the urine was due to inhibition of carbonic anhydrase in the renal tubules. Subsequently, Pitts and Alexander[24] showed that sulfanilamide reduced the capacity of the kidney to eliminate acid under maximal stimulation by a phosphate buffer load.

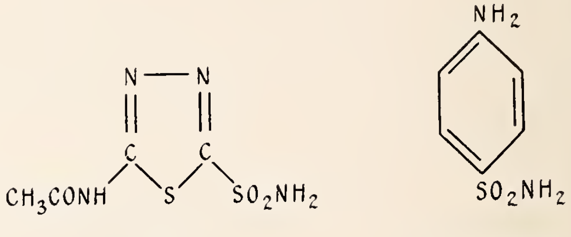
In 1949, Schwartz[26] administered sulfanilamide to three patients with congestive heart failure and observed an increased excretion of sodium, potassium, and water. Although clinical improvement and reduction of edema was noted, the drug was considered to possess too little carbonic anhydrase activity and to be too toxic for continued use.

Chemistry and Pharmacology

Diamox is 40 to 500 times as potent a carbonic anhydrase inhibitor as sulfanilamide. The pharmacological properties have been described by Maren and co-workers.[18,19,20] (Fig. 1.)

Diamox is well absorbed from the gastro-intestinal tract in both the experimental animal and man. It possesses very low systemic toxicity and is excreted largely unchanged by the kidney in twenty-four hours. The renal clearance is approximately two-thirds that of the glomerular filtration rate in the dog. Studies with Diamox and carbonic anhydrase both *in vivo* and *in vitro*, show that the interaction between inhibitor and enzyme is reversible.

Following a single oral dose, there is increased urinary excretion of sodium, potassium, bicarbonate and water. The pH of the urine increases and titratable acid and ammonia excretion are sharply reduced or abolished. This effect lasts about six hours and then reverses with the onset of the recovery phase. This phase lasts about eighteen hours and is marked by urine almost free of bicarbonate and sodium, low in potassium, and with increased titratable acid and ammonia. There is a drop in plasma bicarbonate and pH during the diuretic phase which returns to normal during the recovery period. This effect in the rat is largely independent of the dose in the range of 10-1000 mg./kilo. Dogs given 100 mg./kilo/day for sixteen months showed an initial renal response but then the urinary pattern reverted to normal after the first few days although there was a sustained acidosis characterized by a low blood pH and plasma bicarbonate and elevated chloride. The serum sodium and potassium remained normal. Urinary and plasma electrolytes reverted to normal in three to four days following withdrawal of the drug. If the dog was then challenged with Diamox, the response was the same as with initial treatment. These results were in contrast to those in dogs maintained on 5 to 10 mg./kilo/day where there was a continued renal effect and little or no acidosis. On low daily doses extra base output was effectively countered by extra base conservation during the recovery periods. On high or multiple doses the animal became refractory to the diuretic effect and remained acidotic. The drug is not cumulative with long-term administration and has no preference for any tissue, except red cells, which it appears to saturate at a fairly low level.



Diamox Sulfanilamide

Fig. 1.

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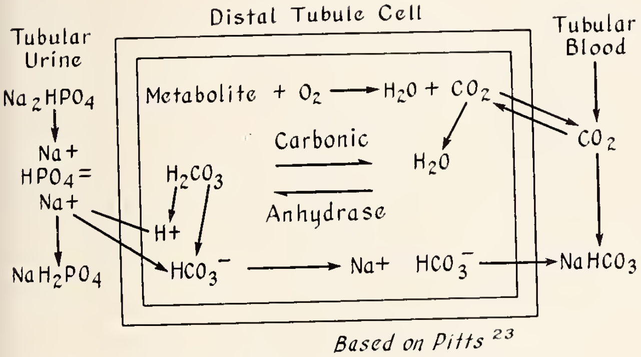
Presented at Medical Grand Rounds, Veterans Administration Hospital, Minneapolis, Dr. E. B. Flink, Chief of Medical Service.

Tomashefski et al[30] and Carter[8] have found after the intravenous administration of Diamox to dogs that the alveolar P_{CO_2} was significantly less than the arterial P_{CO_2}. Shepard et al[28] reported similar findings in two normal human subjects after exercise. These findings are thought to result from interference with the release of carbon dioxide from pulmonary capillary blood after inhibition of carbonic anhydrase.



Based on Pitts [23]

Fig. 2. Process of acidification by renal tubule.

Diamox exerts a marked inhibitory effect on pancreatic secretion in the dog following stimulation with secretin.[5] It has also been shown both in dogs[15] and ulcer patients[29] that Diamox causes a significant inhibitory effect on hydrochloric acid production.

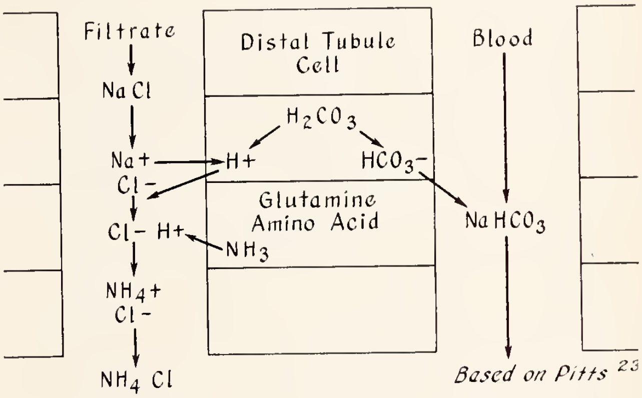
Mechanism of Action

In order to understand better the renal action of Diamox, a brief discussion of acid-base balance by the kidney will be presented. Two hundred liters of plasma are filtered through the glomeruli each day. Each liter of plasma contains approximately 25 mM. of bicarbonate. The total bicarbonate filtered is 5,000 mM., which is five times the body store of bicarbonate.

Obviously the bicarbonate must be filtered over and over again and reabsorbed each time by the renal tubules as only 1 to 2 mM. escapes into the urine each day. Sulfuric and phosphoric acids are formed in considerable quantities in the metabolism of phospholipids and proteins. The excretion of titratable acid and ammonia reverse the process of neutralization and promote the elimination of acid anions minus the base. These two renal mechanisms prevent exhaustion of the bicarbonate stores.

Carbonic anhydrase accelerates the reaction of carbon dioxide and water to form carbonic acid in the tubule cells. The ionization of carbonic acid to form hydrogen and bicarbonate ions provides hydrogen ions which are exchanged for sodium ions of the buffers of the glomerular filtrate, chiefly in disodium phosphate (Na_2HPO_4) and sodium bicarbonate ($NaHCO_3$). The excretion of sodium acid phosphate (Na_2HPO_4) in the urine conserves sodium which returns to the blood. (Fig. 2).

Ammonia (NH_3) is formed from glutamine and other amino acids in the distal tubule cell. Each molecule of ammonia secreted into the tubular urine binds one mole of hydrogen and permits one mole of sodium to be reabsorbed. Thus ammonia is exchanged for base, meq. for meq., although the exchange is indirect (Fig. 3). The rate of ammonia excretion is inversely proportional to the pH of the urine; i.e., the more acid the urine, the more ammonia is excreted, and vice versa.



Based on Pitts [23]

Fig. 3. Process of ammonia production by the renal tubule.

Inhibition of carbonic anhydrase in the kidney leads to the following:

1. Decreased urine acidity due to decreased availability of hydrogen ion from carbonic acid.

2. Increased urinary excretion of sodium due to decreased sodium reabsorption.

3. Increased urinary excretion of potassium due to increased tubular secretion of potassium. Berliner[3] has shown that potassium ions compete with hydrogen ions in the tubules for excretion; hence decreased hydrogen ion secretion leads to an increased potassium output.

4. Decreased ammonium excretion due to limited availability of hydrogen ion for combination with ammonia. This also leads to increased excretion of sodium.

5. Increased total carbon dioxide content of the urine due to increased excretion of bicarbonate. Normally, hydrogen ion secreted by the renal tubule cell reacts with bicarbonate in the tubular urine and the carbon dioxide evolved diffuses back into the blood. When hydrogen ion secretion is blocked by carbonic anhydrase inhibitor this reaction does not take place and bicarbonate excretion is therefore increased.

Clinical Applications of Diamox

Friedberg, Halpern and Taymor[12] studied the intravenous effect of Diamox on twelve hospitalized and ambulatory patients with congestive heart failure of varied etiology and on three patients without evidence of cardiovascular or renal disease. All of the cardiac patients had been treated in the previous few weeks and had little evidence of edema except one patient who had moderate edema of the lower extremities. A single intravenous injection of 0.75 gm. of Diamox was given. Urinary output increased in each case, and the urine contained increased amounts of sodium, potassium, and bicarbonate. Chloride and phosphate excretion were only slightly increased and appeared to be related to the increased urinary flow. The urine became alkaline about thirty minutes after injection and remained so for a period of about six hours. Titratable acid rapidly disappeared. Urinary ammonium was markedly diminished in all cases; however, it continued to be present even when the urine was more alkaline than the plasma. The sodium excretion increased markedly in every instance independent of the presence or absence of congestive heart failure. In two of the control subjects who received 5 to 7 grams of salt a day, the sodium excretions increased four to six times over the control values. In the other control subject on a 500 mgm. sodium diet, there was a tenfold increase in sodium excretion over the control value. No significant effect was noted in plasma concentration of sodium, potassium, chloride, phosphate or creatinine. There was a slight and inconstant tendency towards a mild acidosis as indicated by a decrease in plasma bicarbonate level and blood pH.

Leaf, Schwartz, and Relman[16] performed balance studies on five patients with congestive failure and on one normal subject before and after the oral administration of Diamox in varying dosage schedules. Two of the patients with congestive heart failure and the normal subject demonstrated the usual responses. The other three patients lost no sodium and responded only with increased potassium excretion and reduction in urine acidity. Unresponsiveness to the drug developed rapidly even with intervals of one to two days between doses, and in one case appeared after a single dose of 0.25 gm. They noted that the onset of drug resistance was always associated with the development of hyperchloremic acidosis.

Friedberg et al[13] studied the oral effect of Diamox in twenty-six patients with various types of heart disease and found an increased excretion of sodium and potassium in the urine of all patients regardless of clinical response. There was slight to moderate increase in chloride excretion in sixteen patients and a decrease in eight. Plasma levels of sodium, potassium, and chloride were not altered significantly. The carbon dioxide content fell in most cases, and in some was accompanied by a slight fall in the blood pH. "Excellent" or "good" clinical responses were claimed in eighteen of the twenty-six patients although the mean weight loss was only 4.4 pounds in the hospital group and 3.8 pounds in the clinic patients. Several dosage schedules were employed, but 0.25 gm. every eight hours for two days and repeated every two to seven days or the daily administration of 0.25 gm. were the most effective. Patients who did not respond to smaller doses did not have a diuresis with larger doses nor did prolongation of the course more than two days produce a diuresis in these patients. In the patients who responded satisfactorily to Diamox, diuresis and weight loss continued until "dry weight" was reached which could be maintained as long as the drug was continued.

Relman, Leaf, and Schwartz[25] reported disappointing results following the oral administration of Diamox in twenty-six hospitalized patients with severe congestive heart failure. Approximately half of the patients failed to lose weight and only one of the remainder became edema free, while the others lost 2 to 6 pounds over a seven-day period. All but three patients had a definite pharmacological effect from the drug even when there was little or no diuretic response. Twelve patients had blood nonprotein nitrogen concentrations of 50-100 mgm. per 100 cc. and in fourteen the level was below 50 mgm. per 100 cc. Only three of thirteen courses of treatment in azotemic patients resulted in weight losses of 4 pounds or more; however, seven of fifteen patients without azotemia had this same response. This suggested to the authors the importance of renal function. The explanation of this response is not clear, but it was postulated that carbonic anhydrase is less important for the over-all reabsorption of bicarbonate when the glomerular filtration rate is low and the filtration of bicarbonate is reduced. Another possibility considered was that the azotemia was simply a reflection of severe circulatory insufficiency and therefore of the factors operating to retain sodium. In spite of the disappointing results the authors refer to some encouraging studies[27] in the treatment of congestive heart failure due to chronic cor pulmonale where Diamox appeared to effect large diuresis and significant clinical improvement.

Belsky[2] reported a definite diuresis in all of thirteen patients with congestive heart failure treated with oral Diamox. Eleven of the thirteen patients were kept edema-free with more or less constant weight on a daily dose of 0.5 gm. of Diamox, low salt diet and digitalis. He noted that Diamox alone or in combination with mercurial diuretics had little diuretic effect in the presence of anascarca or renal decompensation in two patients. However, after removal of ascitic fluid, the effectiveness of Diamox appeared to be increased, although supplementary mercurial injections were required.

Nadell[22] reported on the effect of Diamox in two patients with respiratory acidosis and two normal subjects. In three of four patients, the arterial pH fell by 0.1 to 0.2 pH units. After three days, the urine pH and electrolyte values returned to control values and remained unchanged even when the drugs were given for periods of twenty-five to fifty-five days. However, during the entire period the arterial carbon dioxide was decreased 5-10 mEq/1. The control patients remained asymptomatic during the entire period. One of the two patients with respiratory acidosis continued to be alert outside of the respirator on two separate occasions while getting Diamox. When the drug was discontinued, a more marked respiratory acidosis developed. The other patient developed marked drowsiness fourteen days after therapy was begun. This cleared when the dosage was decreased from 10 to 5 mgm. per kilo.

Fishman, Samet, and Cournand[11] found a diminished ventilatory response following an increase in inspired carbon dioxide in patients with pulmonary emphysema with carbon dioxide retention as compared with emphysematous patients without carbon dioxide retention and with normals. The emphysema patients with carbon dioxide retention were given Diamox over a fifteen-month period. Although the arterial P_{CO_2} and alkali reserve returned to normal there was no improvement in the ventilatory response to carbon dioxide.

Bergstrom[4] et al reported on the effect of Diamox in forty-two epileptic persons with intractable seizures. The rationale underlying such therapy is the production of a mild metabolic acidosis similar to that produced by ketogenic diets. Ninety to one hundred per cent seizure control was obtained in four instances and 50 to 90 per cent in four others. None was made worse. The authors concluded that Diamox is useful in only a small number of cases refractory to other measures.

Becker[1] treated nineteen patients with glaucoma with single doses of 0.5 to 1.0 gms. of Diamox. In all cases, the intraocular pressure began to decrease in 60 to 90 minutes. It reached a minimum in three to five hours and returned to initial levels in eight to twelve hours. Because of the high concentration of bicarbonate in the posterior chamber it was postulated that carbonic anhydrase plays an essential rôle in the formation of aqueous humor. It was not determined whether its mode of action is to inhibit the secretory mechanism directly or whether the fall in intraocular pressure is secondary to induced systemic electrolyte alteration.

Diamox has been tried in the treatment of several conditions associated with edema formation. The impaired renal function in chronic glomerulonephritis probably interferes with the effectiveness of Diamox as a diuretic. However, since it had not proven dangerous, it was thought to be worthy of a trial.[9] Equivocal results have been obtained in the treatment of cirrhosis.[6,7]

Toxicity

No serious toxic effects have been reported with Diamox. Paresthesias of the face and hands and drowsiness have been observed frequently and appear to be related to the dosage.[2,4,13] These side effects have been encountered when the daily dose of Diamox was 1.0 gm. or more and were reduced to a minimum or disappeared when the dose was decreased to 0.5 or 0.25 gm. per day. Bergstrom et al[4] reported fatigue, flushing, polydipsia, hypernea, paresthesias, and headache with doses of 10 to 30 mg. per kilo in the treatment of epileptics. but did not state the number of cases involved. A few instances of disorientation and irrational behavior have been observed in cirrhotic patients treated with Diamox.[10] No instances of renal damage, hematologic disturbances* or skin manifestations have been reported.

Summary

Diamox is a potent carbonic anhydrase inhibitor affecting carbonic anhydrase contained in red cells, kidney, stomach, pancreas, and eye. The usual pharmacological response is characterized by the diuresis of an alkaline urine containing increased amounts of sodium, potassium, and bicarbonate and a decreased plasma bicarbonate and blood pH.

The results of Diamox administration in various disease states have been reviewed. The drug is effective both orally and via the intravenous route. The toxicity is low and consists mainly of paresthesias and drowsiness.

Further clinical trial appears warranted.

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*Agranulocytosis following Diamox therapy was recently reported.[22a]

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GYNECOLOGICAL CANCER DETECTION

(Continued from Page 178)

depend on detection of carcinoma at a stage amenable to treatment. The accessibility of the female pelvic organs makes this region nearly ideal for the early detection of malignancy.

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LIMITATIONS OF THE WIDAL TEST

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THERE are three reasons why it is necessary to re-evaluate the Widal test as a laboratory aid in the diagnosis of typhoid fever: (1) knowledge regarding the antigenic relationship of Salmonella typhosa (Eberthella typhosa) to many other Salmonella organisms; (2) the Widal test is essentially of no diagnostic value in the increasingly large number of persons who have had inoculation with typhoid and paratyphoid A and B vaccine; (3) the incidence of typhoid infection in Minnesota is low.

Typhoid O (somatic) and typhoid H (flagellar) antigens are used in the quantitative Widal test. There are approximately 63 Salmonella organisms that possess one or more of the somatic antigens found in S. typhosa. Accordingly, a person who may be infected with and produce antibodies to any one of these organisms gives a satisfactory titer when his serum is tested with O antigen. For example, we have not infrequently demonstrated O antibody titers as high as 1:1,280 in serum from patients with enteric infection and have isolated only S. enteriditis from the stools of these patients. The high O antibody titer in these patients can be easily explained since S. enteriditis possesses two somatic antigens identical to those of S. typhosa.

It is frequently stated that during the early stages of typhoid infection the O titer is higher than the H titer and that a high O titer is diagnostic of typhoid fever. We cannot agree with this statement. The high O antibody titer may be due to an organism with somatic antigens identical to the somatic antigens of the typhoid bacillus. We found that H titers in the great majority of cases were higher than O titers in a study of agglutination reactions in serum from 103 persons reported to be ill with typhoid fever. Also negative results were found in 5.8 per cent of the O tests in this study as compared to negative results in only 1.0 per cent of the H tests. In a study of the agglutination pattern of forty-seven ill patients diagnosed as having typhoid fever and from whom S. typhosa was isolated, the H antibody titer in the great majority of patients was higher than the O titer; in two patients there was no demonstrable O titer, only H agglutinins being detectable.

Thus it would appear that perhaps the H antibody titer would offer more certain aid in the diagnosis of typhoid fever. A study of the H and O antibody titers on serum from 8,945 ill persons who did not have typhoid fever showed that the H antibody titer was consistently higher than the O titer. This was particularly true of serum obtained from 787 persons who had received typhoid vaccine. In this vaccinated group, the results in 43 per cent of the H agglutination tests were positive in dilutions of 1:320 or more, whereas only 2.4 per cent of the O agglutination titers fell in this range. Negative results of H agglutination tests were observed in 24.1 per cent of this vaccinated group as compared to negative results in 55.6 per cent of the O agglutination tests. In a study of serum from 8,075 ill persons who did not have typhoid fever and who gave no history of having had typhoid fever or vaccination, 3.3 per cent of the H agglutination tests gave positive results in dilutions of 1:320 or more, whereas only 0.3 per cent of the O agglutination tests were in this range. Thus it is obvious that we cannot depend on H agglutination as an aid in the diagnosis of typhoid fever.

Perhaps it may be argued that differences in the H and O antibody titers would help in making a diagnosis. Unfortunately no definite single H and O titer is diagnostic of typhoid

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This is the twenty-fifth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

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fever. The higher the titer chosen for the diagnostic dividing line, the more likely is such titer to indicate infection but at the same time the smaller will be the number of cases of typhoid thus diagnosed.

We may try to get some value out of the Widal test as a diagnostic aid by testing serial specimens of blood for H and O antibodies. Again we are frustrated by persons who may have had typhoid fever and by the great many people who have received typhoid vaccine. No diagnostic value can be placed on the H agglutinins in a vaccinated person or a person with a history of previous typhoid fever. Even a considerable increase in titer is of little or no diagnostic importance since this may be due to nonspecific causes. No reliance can be placed on O agglutinins since other organisms share common O antigens with S. typhosa. A blood culture done early in the disease would give specific results and in a shorter period than would Widal tests done on a series of specimens of blood.

Since the Widal test is of questionable value in the specific diagnosis of typhoid fever and of no diagnostic value in vaccinated or previously infected persons, we might ask if it is of value in the detection of typhoid carriers. Serologic studies were done on thirty-seven bacteriologically proved typhoid carriers. Nineteen of these carriers (51 per cent) were negative when tested with O antigen including seven who were also negative when tested with H antigen. Eighteen (49 per cent) of the thirty-seven carriers tested showed both H and O antibodies, but the H antibody titers were higher. Although H agglutinins were present more frequently and in higher titer than were O agglutinins in these proved carriers, the same was found to be true in examination of serum from 229 well persons who gave a history of typhoid vaccination, from twenty-eight persons who gave a history of previous typhoid infection and from 2,355 persons who gave no history of vaccination.

Conclusions

1. No arbitrary titer of either H or O agglutinins can be accepted as diagnostic for typhoid fever.

2. Since Salmonella typhosa shares common O antigens with other Salmonella organisms, the O agglutination test is not specific for typhoid.

3. The Widal test is of no practical value in the diagnosis of typhoid fever in vaccinated persons or those with a history of previous typhoid fever.

Recommendations

1. If typhoid or paratyphoid infection is suspected, culture of blood and stools should be done early in the illness. Blood for culture should be collected during the first two weeks of illness. Examination of stools is the procedure of choice after the first two weeks of illness.

2. Stools should be submitted for culture from all persons having enteric infection suggestive of salmonellosis or shigellosis.

3. Since Salmonella typhosa must be isolated from suspects to prove their carrier status, it is thought that examination of stools from the following persons will yield more productive results than will the Widal test: (1) those giving a history of typhoid fever; (2) those who have resided with or prepared food for any persons who have contracted typhoid fever; (3) those who have symptoms suggestive of gallbladder disease; (4) food handlers who, in addition to any of the conditions just mentioned, may give a history of a recent intestinal upset of any nature.

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The Committee on Medical Motion Pictures of the American Medical Association announces that Booklet 6 of Reviews of Medical Motion Pictures is now ready for distribution. This booklet contains sixty-four critical reviews of medical and health films which were published in *The Journal* during 1954.

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CASE PRESENTATION

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Clinical History

A fifty-three-year-old bartender was first admitted to this hospital for treatment of seborrheic dermatitis and for injection of varicose veins, January 30, 1942. He stated that for six weeks he had been receiving intravenous and intramuscular injections, but did not know for what purpose. His blood pressure was 132 mm. Hg systolic and 80 mm. diastolic. Urinalysis showed no abnormalities. The blood Wassermann test resulted in negative findings.

On April 14, 1951, the patient, now aged sixty-two, was admitted to the hospital complaining of epigastric pain for three weeks. Five weeks prior to admission, he had been awakened by a severe constrictive retrosternal pain which extended into his left arm. He was hospitalized elsewhere for two weeks at bed rest. Thereafter, he had no appetite and had suffered three or four attacks of severe colicky pain in the epigastrium which extended across the right upper abdomen and to the right scapular area. He believed that these attacks were caused by eating fried or greasy foods. At times, he had nausea and vomiting during the attack. There had been no chills, fever, jaundice or darkening of the urine.

For three years, he had experienced substernal pain extending into the left arm on exertion, promptly relieved by rest or nitroglycerin. For a year, he had some dyspnea on exertion but no orthopnea nor paroxysmal nocturnal dyspnea. He had had gonorrhea forty years ago but denied having had syphilis. He had been drinking alcoholic beverages excessively for several years.

His mother had died of "heart trouble" at fifty-eight years, his father had died of a "stroke" at fifty-eight years, and one brother had died of "heart trouble" at forty-two years.

The patient was well developed, well nourished and in no acute distress. The pupils reacted normally. The temperature was 98.6 degrees F., the pulse rate 84 per minute. The lung fields were normal to percussion and auscultation. The heart sounds were of good quality and no murmur was heard. The aortic second sound was louder than the pulmonary sound. The point of maximal impulse was in the fifth interspace at the anterior axillary line. The blood pressure was 170 mm. Hg systolic and 116 mm. diastolic. The abdomen was normal apart from moderate epigastric tenderness. Neurological examination showed no abnormalities.

The hemoglobin was 11.3 grams per 100 ml., the leukocyte count 7,250 per cu. mm. (65 per cent neutrophils, 33 per cent lymphocytes and 2 per cent eosinophils), the erythrocyte sedimentation rate 60 mm. at one hour. Specific gravity of the urine was 1.014, the sediment contained 0 to 3 erythrocytes and 0 to 1 leukocyte per high power field, the test for albumin was 1 plus. A catheterized urine specimen showed 15 per cent excretion of phenolsulphonphthalein in one-half hour. The blood Wassermann test was negative, the Kahn 3 plus, the Hinton 4 plus, the VDRL slide test positive in 1 to 2 dilution. Examination of the spinal fluid on two occasions was completely negative, including total protein, complement fixation and gold curve. There was 7.2 per cent retention of bromsulfalein at forty-five minutes, the cephalin flocculation test gave negative findings, the thymol turbidity 5.2 units, the serum bilirubin 0.1 mg. per 100 ml. at one minute and a total of 0.5 mg. A two-hour urine urobilinogen was within normal limits. The blood urea nitrogen ranged between 16 and 25 mg. per 100 ml., the total protein was 6.9 gm. per 100 cc. (3.9 albumin and 3.0 globulin). Five stools showed no occult blood. The serum amylase was 56 units.

The posteroanterior radiograph of the chest showed a left ventricular cardiac contour, marked dilatation of the ascending aorta, tortuosity of the descending aorta and fluoroscopy confirmed the presence of calcification within the ascending aorta. An upper gastrointestinal series and cholecystogram were negative. Barium enema showed numerous diverticula in the lower colon. Radiographs of the skull showed calcification in the vessels of the circle of Willis. An electroencephalogram showed abnormalities, but with no specific connotation. Electrocardiograms showed left axis deviation, sagging ST and negative T in V5 and V6.

Shortly after admission, the patient experienced an episode lasting for two hours, characterized by numbness and weakness of the left arm. There was definite transient weakness of the left forearm during that time, but no abnormal neurological signs were demonstrated either during or after this episode. Throughout his course, he complained constantly of a severe, generalized headache which was not relieved by codeine. There was anorexia and occasional nausea and vomiting not described as projectile. Within five weeks, he had lost 12 pounds in weight. On the morning of June 24, he was found to be comatose, gasping, cyanotic and promptly expired.

Discussion

Dr. Smith: For three years, this man, now sixty-two, experienced substernal pain which extended into the left

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From the Minneapolis Veterans Hospital; Dr. Gleason, pathologist.

arm on exertion and which was promptly relieved by rest or nitroglycerine. For a year, he had some dyspnea on exertion but neither orthopnea nor paroxysmal nocturnal dyspnea. This, of course, is the classical description of angina pectoris. To me, it means that the man has myocardial ischemia. There is a host of diseases which will cause myocardial ischemia, the most important of which is coronary artery disease, and it is by far the most common cause in our "macro-molecular" human population.

Five weeks prior to admission, he had been awakened by a severe constructive retrosternal pain which extended into his left arm. He was hospitalized elsewhere for two weeks' bed rest. Myocardial ischemia occurring during sleep has a different connotation. Ischemia can occur during sleep if an individual has frightening nightmares or if for some reason a hypertensive patient drops his blood pressure to low levels. Most often, it is due to an increment of coronary artery disease, usually thrombosis. If this ischemia is lasting, usually over fifteen to thirty minutes, some myocardial necrosis will occur, and we clinically call it infarction. I assume that the diagnosis of myocardial infarction was made when this patient was hospitalized elsewhere.

Dr. Samuel Nesbitt: That diagnosis was made.

Dr. Smith: The protocol states that he had gonorrhea four years ago but he denied having syphilis. That is a very circumstantial bit of evidence for a diagnosis of syphilis, but he stated he had been treated for six weeks with intravenous and intramuscular injections, although he did not know their purpose. In 1942, the treatment in vogue, of course, was combined mapharsen and bismuth therapy. It would have been possible to confirm this treatment with an x-ray of the gluteal region to look for intramuscular deposition of bismuth. Possibly even now, the radiologist can aid in this problem. Dr. Nesbitt used the past perfect tense here, "he had been receiving." I assume that this was just a short period before admission, and the serological test for syphilis was presumably negative when seen by his physician then. This causes me to wonder what evidence his physician had in 1942 to start this therapy.

Ordinarily, physical examination will help us somewhat in the differential diagnosis of the cause of angina pectoris. There was no evidence of any valvular disease. He did have hypertension and, of course, hypertension tends to accelerate atherosclerosis. The blood Wassermann test gave negative findings, the Kahn was 3 plus, the Hinton 4 plus and the VDRL slide test positive in 1:2 dilution. We have already some presumptive evidence that this man had syphilis and I will add this as very strong evidence. In late syphilis, this discrepancy is seen fairly commonly—complement fixation tests being negative while macro and micro flocculation tests are positive. If we were seeing this patient today, perhaps treponema immobilization tests would be of value.

The posteroanterior radiograph of the chest showed a left ventricular cardiac contour. There is a discrepancy between the radiograph as just described and the physical examination. I will accept the radiograph in this controversy. I think that physical examination of the heart is a very difficult and sometimes confusing procedure. Further, the radiographic examination showed marked dilatation of the ascending aorta, tortuosity of the descending aorta, and fluoroscopy confirmed the presence of calcification within the ascending aorta. This latter finding I would like to call pathognomonic evidence of lues. It is probably better evidence than a pathologist's examination. A study reported in 1945 by Jackman and Lubert[2] showed that in patients studied at post mortem, in whom the diagnosis of syphilis was possible pathologically, 22 per cent had calcification of the ascending aorta. Two patients with a pathological diagnosis of only arteriosclerosis had similar calcification. In one of these, there was serologic but no pathologic evidence of syphilis. The other patient had very diffuse and extremely severe arteriosclerosis. I think that this is firm evidence. We could pause here and let Dr. Olson show us the radiograph.

Dr. P. A. Olson: The chest radiograph shows the important features which Dr. Smith discussed. There is very distinct linear density in the wall of the ascending aorta which is also dilated to aneurysmal degree. It is sometimes hard to state what is aneurysmal and what is just dilated, but certainly this is wide enough so we would call it aneurysmal. There is calcification in the ascending aorta, so we should certainly consider that lues is present until proven otherwise. The heart as seen in the posteroanterior view shows moderately severe enlargement of the left ventricle. The second shadow, the denser one medial to the left heart border, is the descending aorta. We cannot visualize its right border. Whether he has another aneurysm here is difficult to determine, but the aorta is certainly wider than it should be and is rather tortuous. The gastrointestinal series is normal, save for a diverticulum of the duodenum. This one radiograph of the gall bladder shows that the dye is not concentrated well, but there is no evidence of calculi.

Dr. Smith: The electrocardiogram showed left axis deviation, sagging ST and a negative T in V 5 and V 6. In addition, he has very low R waves across the anterior precordium. I would interpret these changes as subendocardial damage which might have well extended transmurally, anteriorly.

Now, I would like to go back to the reason for his admission. His presenting complaints were in the gastrointestinal system. Examination of the abdomen revealed no abnormalities except for moderate epigastric tenderness. His leukocyte count was normal and the erythrocyte sedimentation rate was 60 mm. There was a 7.2 per cent retention of BSP. The serum amylase was 56 units—I wonder if that was drawn at a time when he was having distress.

Dr. Nesbitt: We do not know.

Dr. Smith: We could see those abdominal x-rays now.

Dr. Olson: We can see marked calcification in this area which is typical of calcified abdominal aorta. The barium enema shows only a few diverticula in the sigmoid and the lower descending colon. There are moderately severe arthritic changes. There is no evidence of bismuth in the buttocks.

Dr. Smith: This brings up the problem of gastrointestinal complaints in patients with heart disease—a discussion about abdominal angina. In a man with a very compromised coronary blood flow, the mere addition of heavy meal may cause distress. One other possibility which has been advanced more frequently recently is that people who have diffuse vascular disease actually have mesenteric artery insufficiency and distress on this basis.

I am unable at this stage to make a diagnosis of hepatic cirrhosis in this man. I do not think there is any strong evidence of intrinsic gastrointestinal disease. Specific gravity of the urine was 1014. He had a 1 plus albumin and 15 per cent excretion of phenolsulphonphthalein in one-half hour. This depression, along with a normal blood urea nitrogen value, is quite a common functional derangement in patients with hypertension with efferent arteriolar constriction.

So now we go to the next complaint. His neurological examination was normal at the time of admission.

He had two spinal fluid examinations completely negative. Did this include dynamics?

Dr. Nesbitt: Yes.

Dr. Smith: A radiograph of the skull showed calcification in the vessels of the circle of Willis. Shortly after admission, according to the protocol, the patient experienced an episode lasting for two hours characterized by numbness and weakness of the left arm. "There was definite transient weakness of the left forearm but no abnormal neurological signs." I think these two statements are mutually exclusive. If he had true weakness, it would have to be listed as neurological, unless it is known to be muscular. I assume it is meant that he had no reflex changes. Throughout his course, he complained constantly of severe generalized headache which was not relieved by codeine. He had vomiting, not described as projectile. He lost 12 pounds in weight. Finally, he was found to be comatose, gasping, cyanotic and promptly expired. Do we know how long before he was found to be comatose that he was last observed?

Dr. Nesbitt: He was seen about an hour before. He was found in distress and he died in a very few minutes after that.

Dr. Smith: This finding of calcification in the vessels of the circle of Willis in a man who has hypertension, is not really very helpful. Camp[2] states that 4 per cent of any given group of people of age sixty would have such calcification. I would like to see those x-rays now.

Dr. Olson: Maybe you can't see it, even from the front row, but there is calcification in the internal carotid artery. There are several other little linear calcified densities in the region of the sella, the exact site of which is not definite but appears to be in vessels. The internal carotid is definitely calcified. The pineal gland is not calcified, so we cannot use it as a landmark as far as shift is concerned. No other abnormality is seen.

Dr. Smith: Well, we have a man with angina pectoris, myocardial ischemia, and luetic aortitis. The problem now is, why does he have coronary artery insufficiency? He did not have aortic insufficiency. Most patients who have ostial involvement of the coronaries have aortic insufficiency. Even when aortic insufficiency is present most patients who have evidence of myocardial ischemia do have coronary artery disease. We have evidence that he has arterial disease elsewhere. I would prefer to think that he has coronary atherosclerosis in addition to his luetic aortitis. He has hypertension, the cause of which we do not know. We know that he was normotensive at the age of fifty-three and that he became rather severely hypertensive thereafter. We do have evidence that this vascular disease is rather far advanced and that he had cardiomegaly otherwise poorly explained; I would prefer to think that his hypertension was of relatively long standing and its cause will not be disclosed. In the kidneys, he should have changes of benign hypertension with or without arteriosclerosis. I mentioned the gastro-intestinal complaints and the fact that I am wavering between abdominal angina and mesenteric artery inadequacy. I think the latter is a possibility when we have demonstrated vascular disease, and he did have, I think, some vascular disease of the lower thoracic and upper abdominal aorta so that there is involvement in the area of the mesenteric artery.

The causes of the cerebral symptoms are very difficult for me to ascertain. There seem to be no localizing findings. We have evidence that the headache was not on the basis of increased intracranial pressure. Since this man was an alcoholic, I would like to consider the possibility of a subdural hematoma. I should think if it were the cause of this man's symptoms, there would be evidence of increased pressure.

Dr. E. T. Bell: Could ischemia of the brain give him headaches?

Dr. Smith: Are you thinking of diffuse ischemia?

Dr. Bell: Yes, I was thinking of a stenotic artery ischemia.

Dr. Smith: Yes, but he has had symptoms for some time, and I'm bothered by ascribing a severe headache to that cause. However, there is the possibility that he had an aneurysmal dilatation of some larger artery intracranially. That could cause headache without increased intracranial pressure. There is really no positive evidence for it and yet I feel that this man's headache has to be explained.

Now can we put all these things together in an Oslerian fashion? As has been done so often in this series, polyarteritis must be raised as a strong possibility. I agree that this is a possibility but I find no evidence for it. For me, it won't explain the headache very well. I think the hypertension is of long standing. It could explain the abdominal pain, but we already have two other reasons for vascular disease, and I dislike raising a third. Dissecting aneurysm should be considered. This could cause pain simulating myocardial infarction and it would give rise to diverse pains and findings elsewhere. I assume that the pulses were palpated and that they were equal. The man already has luetic aortitis. Others have reported that the finding of a dissecting aneurysm superimposed on the luetic aortitis is highly uncommon and actually the former gives some protection against dissection.

Embolization would be one way to put everything together—mycotic aneurysms from SBE or bland emboli from mural thrombus or from arteriosclerotic plaques. I should think, though, that we would have localized findings that are more specific.

I have to state that I am unable to arrive at a definite entity to explain his central nervous system findings, that I would favor an aneurysm of one of the large arteries at the base of the brain which was not seen roentgenographically.

Dr. J. P. Knoedler: Do you think his headache could be due to hypertension?

Dr. Smith: His headache was of an acute onset, his hypertension chronic. I dislike ascribing his headache to hypertension.

Dr. C. N. Sadoff: You said that you prefer to regard the hypertension as a chronic thing and would you then disregard the blood pressure reading in 1942?

Dr. Smith: No, I think that this hypertension probably developed in the interval.

Dr. J. Rothstein: Would you entertain the diagnosis of a slow-leaking cerebral artery aneurysm?

Dr. Smith: I would be worried by the fact that he did not show cerebral spinal fluid changes.

Question from Audience: Would you care to hazard a guess as to the most likely cause of his sudden demise?

Dr. Smith: Yes, I meant to mention it. His hemoglobin had fallen, for reasons not explained, when he came in. I think this amount would be too much for bleeding into the central nervous system. He died very suddenly. It sounds like a cardiac death or death from exsanguination; he is a candidate for either one.

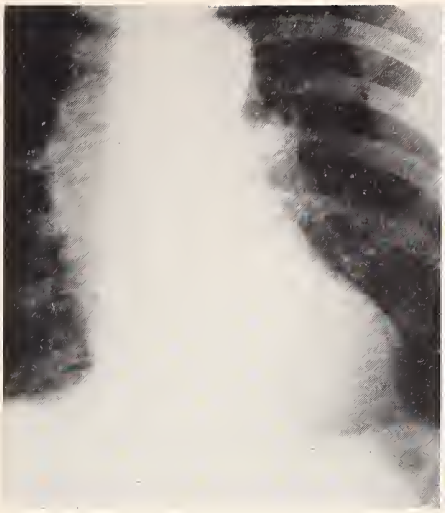

Fig. 1. Chest radiograph, April 16, 1951. Note widened ascending aorta with irregular linear streaks of calcium and second abnormal convex density superimposed behind enlarged left heart border. (Photograph retouched to give more accurate reproduction.)

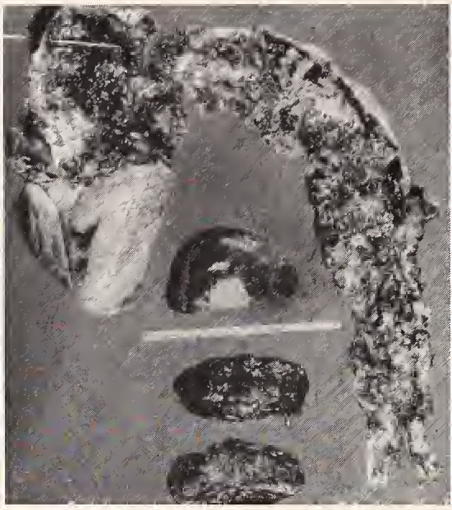

Fig. 2. Heart, aorta and kidneys (one bisected). Severe atherosclerosis of aorta with aneurysmal dilation, ulcerations and mural thrombi. Coarse scars and pitting of kidneys.

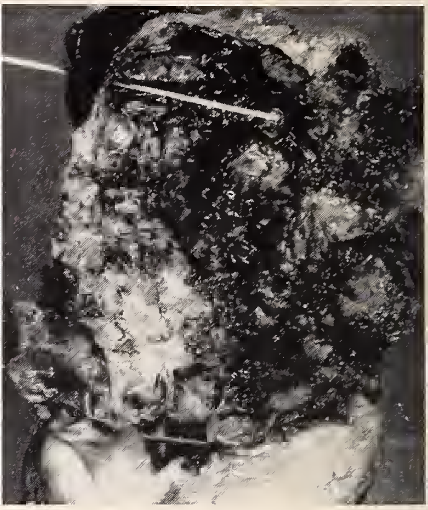

Fig. 3. Heart, aorta, closeup. Severe ulcerating atherosclerosis. Applicator stick in perforation which dissected into pericardial sac.

Dr. Nesbitt: Exsanguination from what?

Dr. Smith: It would be either from the aneurysm in the arch of the aorta or in the descending aorta.

Dr. T. Tamlyn: Do you think that the cerebral difficulties might be explained by meningovascular lues?

Dr. Smith: Well, meningovascular lues should appear relatively early—within five years. If one requires absolute criteria for the diagnosis of vascular lues, he needs to demonstrate the organism. That would be unusual especially since he received some therapy.

Question from Audience: About this radiologic sign of duodenal diverticulum; could that be considered as a perforation into fat rather than a true diverticulum?

Dr. Olson: It's on the lesser curvature of the duodenum. It almost has to be a diverticulum.

Dr. Nesbitt: Clinically, the diagnosis was that of luetic aneurysm. No one hazarded any suggestion on the chart as to the cause of the sudden exitus. The neurologist saw the patient on several occasions and all he offered as the cause of his headaches was "headache following spinal tap," which had been done several times in an effort to determine the cause of his headaches. Shall we read the students' diagnosis?

Dr. E. B. Flink: As a matter of fact, the students' opinions include those diagnoses that Dr. Smith has given. One just diagnosed atherosclerosis and arteriosclerotic heart disease. That's the briefest one. Most of them, though, include syphilitic aortitis with aneurysm. Many felt that the final episode was a cerebral vascular accident rather than cardiovascular.

Clinical Diagnosis

1. Leutic aneurysm.

Dr. Smith's Diagnoses

1. Leutic aortitis and aneurysm.
2. Hypertension, cause unknown.
3. Arteriosclerotic heart disease with coronary arteriosclerosis.
4. Cerebral arteriosclerosis; possible cerebral artery aneurysm.
5. Abdominal angina or mesenteric artery insufficiency.
6. Death from myocardial infarction or exsanguination from aneurysmal rupture.

Autopsy Conference

(October 22, 1954)

Dr. D. F. Gleason: The general autopsy examination revealed significant pathologic conditions only in the heart and aorta. The heart itself weighed 525 grams. The pericardial sac was tensely distended by approximately 750 cc. of blood. The left anterior descending coronary artery was completely occluded by an old, pale fibrotic and calcified process and there was a large area of thinning and scarring of the anterior wall of the left ventricle. The left circumflex coronary artery was approximately 50 per cent narrowed but the right coronary artery showed only minimal atherosclerosis. All of the cardiac valves were normal including the aortic valve. The striking pathologic lesion was in the aorta. The ascending and transverse portion of the arch of the aorta was dilated up to 8 cm. in diameter. This process began a few centimeters above the aortic valve ring. There was a very severe atherosclerosis involving the arch of the aorta and the entire thoracic and abdominal aorta and extending down into the iliac arteries as far as they could be examined. This was a very severe process with ulceration of the intima and patches of mural thrombus from the arch of the aorta to the iliac arteries. The aorta and heart are shown in the accompanying photograph.

From the gross examination alone, as seen in these pictures, we would not be able to make a diagnosis of syphilitic aortitis nor syphilitic aortic valvulitis. There is not a sharp line of demarcation of the process in the thoracic aorta as is usually seen with syphilitic aortitis. The aortic valves are entirely normal and do not show the thickened rolled valve edge nor the separation of the commissures usually found in syphilitic aortitis. However, the changes of atherosclerosis, if superimposed upon leutic aortitis, can obscure completely the changes we require to diagnose luetic aortitis.

The immediate cause of death can be seen in the photograph. There was a small rent in the wall of the aorta some distance from the aortic valve but so located in the ascending aorta that the perforation extended into the pericardial sac. The pericardium contained almost 750 cc. of blood which was partially clotted. The patient undoubtedly suffered an acute cardiac tamponade due to rupture of the aorta into the pericardial sac.

The gall bladder and biliary ducts were normal.

Microscopic examination of sections of the aorta reveals a very severe degree of intimal atherosclerosis which extends deeply into the media and partially destroys it. In addition, there is a perivascular inflammatory infiltrate of lymphocytes and plasma cells around the vasa vasorum in the adventitia of the aorta and occasionally extending a short distance into the media. These are the lesions which are often described as characteristic of syphilis of the aorta but we know that severe atherosclerosis of the aorta can also be associated with such lesions, in the absence of syphilis. Sections of the myocardium confirm the presence of an old scarred infarct due to old occlusion of the left anterior descending coronary artery. In at least one area there are a few chronic inflammatory cells still present, consistent with the clinical duration of the infarct, that is three or four months before death.

Sections of the kidney reveal large irregular areas of atrophic kidney substance related to completely occluded small arteries. Many of these atheromatous lesions contain large "cholesterol clefts." We also see similar occluded arteries in the spleen and pancreas. The kidneys show only mild evidence of hypertension in the form of collagenous intimal thickening of small arteries and arterioles.

Sections of the lung reveal mild acute pulmonary edema and congestion.

Sections of the liver reveal no evidence of cirrhosis.

Gross and microscopic examination of the brain reveals a small area of relatively old encephalomalacia just adjacent to one of the caudate nuclei. This is in a stage of repair which would seem to indicate a duration of a month or two. This may have been partly responsible for the patient's headaches.

Final Anatomic Diagnoses

1. Severe generalized arteriosclerosis.
2. Severe atherosclerosis of the aorta from the ascending portion of the arch out into the iliac arteries.
3. Aneurysmal dilatation of the aorta.
4. Perforation of the ascending portion of the aorta into the pericardial sac.
5. Acute cardiac tamponade due to hemorrhage.
6. Luetic aortitis.
7. Diverticula of the duodenum and cecum and sigmoid colon.
8. Moderately severe arteriosclerotic damage to the kidney.
9. Relatively recent area of encephalomalacia in the brain.

We have to admit that we cannot be certain about the presence or absence of luetic aortitis, but considering all the evidence, we feel that it is probably present.

Dr. Smith: I would like to ask whether the ostium of the superior mesenteric artery was occluded by atherosclerosis.

Dr. Gleason: I am sorry, but that area was not described specifically and I cannot identify it from our gross photographs.

Dr. Bell: I would like to comment upon this aneurysm. When syphilitic aneurysm is overshadowed by massive atherosclerosis like this, we do not know whether syphilis is present or not. We have no criteria to identify it when severe atherosclerosis is present. If you read that some writer diagnoses syphilis or no syphilis in cases like this, I tend not to believe him. I do not believe there is any way to tell them apart. If syphilitic aortitis occurs in a patient without much atherosclerosis, we can make the diagnosis relatively easily. This man has an extremely severe atherosclerosis which has destroyed much of his aorta. That can cause dilation also, but in view of this aneurysm being in the ascending arch and in the presence of positive serology, I believe that the presumption is that this has a syphilitic background to it. Any kind of an aneurysm can rupture.

Dr. Smith: I would like to ask about pain in small cerebral infarcts. I have the impression that the headaches associated with these are not unremitting nor unrelieved by codeine. That bothers me a great deal. I know we do not have an explanation for the headache otherwise. I wish we did.

Dr. Bell: The patient probably had a lot of cerebral ischemia. The headache might be due to that.

Dr. Smith: Other individuals with cerebral ischemia do not usually have headache.

Dr. Flink: Would you say just a word about the causes of sudden death?

Dr. Smith: When sudden death occurs, that is within a few minutes, it is almost always cardiovascular. Death due to lesions of the central nervous system are very infrequently that fast. This patient was observed about half an hour before the comatose state and he died very promptly after that. There are only a few central lesions which will cause sudden death, one being rupture of an aneurysm of the basilar arteries; it is uncommon.

Dr. Flink: Incidentally, one of the students did give rupture of an aneurysm of a basilar artery as the cause of death. We are often faced, in patients of this age, with the problem of explaining an elevated sedimentation rate, in a patient who is otherwise apparently well. Seeing the aorta in this man, I just wonder whether there is not enough thrombotic process and enough ulceration to explain the elevated sedimentation rate. I am sure he had myocardial infarction in the not-too-distant past and he has destruction of tissue in the kidney, but I wonder whether the aorta does not account for the elevation of sedimentation rate in this case.

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President's Letter

TO GIVE WISELY

The problem of personal giving to all the many and varied appeals for funds that come, not only to the doctor's office, but to the home of the average citizen of our state, is one that is becoming increasingly more acute. In scores of emotional appeals, many coming by mail from obscure institutions and faraway places, we are besieged to contribute to every conceivable area of good will and human need.

Americans have a reputation for generous giving, for caring for their fellow men, but this also makes us vulnerable targets for fund campaigns, some of whose natures, purposes, and methods, are questionable. We find it virtually impossible to give to all of the worthy drives.

Because organized fund raising has become big business—money-wise it is the fourth largest industry in the nation—the average citizen wonders if he can depend on his judgment alone to determine the campaign merits. He knows that most campaigns are masterminded by skilled professionals, hired for their scientific know-how about how to open the purse strings. For better or worse, these professionals are a necessity in this modern age, and we all realize this. We know that the future of great humanitarian endeavors hinges on how successfully a campaign is put across.

There is a great overlapping of appeals for funds by various organizations. This is particularly true in the medical field where societies have been formed to establish research funds and to relieve sufferers in almost every conceivable disease. The comedian, George Gobel, recently remarked that it is getting now so that there are hardly any good diseases left. Would it be possible to consolidate all the money raised for research into one fund?

The disturbing problem is pointed out by the following finding in a report from the Campaign Review Council in the city of Duluth. There are thirty-four separate groups now seeking public support in Duluth to aid crippled children and adults. Some of these groups are covered by the Community Chest; most of the others conduct private fund-raising campaigns. Some of the thirty-four groups are concerned only with research; others with rehabilitation; still others with giving personal aid. All are worthy causes, but where does the average citizen draw the line?

As doctors, we are often asked whether the funds raised by the various organizations are well supervised and well spent. I'm sure that we can honestly say that in the great majority of instances, the money received is well spent. However, the very multiplicity of all these campaigns will eventually lead to a point of diminishing returns.

Wouldn't it be possible for each of us in our own communities to work toward some form of consolidation of these various fund drives? In Duluth, for example there is one large fund drive occurring in almost every month of the year, not to mention many of the rather questionable requests for funds that are received from organizations outside our state at or near the holiday season. It is my fear that the very multiplicity of these drives will be the factor that "kills the goose that lays the golden egg."

Arnold O. Swenson

President, Minnesota State Medical Association

Editorial

John F. Briggs, M.D.
Henry G. Moehring, M.D.
Arthur H. Wells, M.D.

TAKING NOTES AT MEDICAL MEETINGS

We have been impressed recently, not only with the tremendous number of note-taking techniques employed at medical meetings, but with the startling revolution our own note-taking has undergone. There was the day when we went to medical meetings with much note-taking paraphernalia including at least two never-fail mechanical pencils, that always managed to fail, and against the failure of which we also carried a freshly filled fountain pen. (In reviewing some of our old notes, however, it appears that a pencil can fail and the writer not become aware of it, for some of our notes appear only as indentations in the paper—no lead, notes taken in the dark.)

Then there was a refinement in note-taking; the flashlight. This required that a flashlight be added to a pocket already bulging with writing gear. When the lecturer demanded that the lights be turned out so he could show his slide, the note-taker grasped the flashlight and its switch in the left hand, his pencil (we assume it is still functioning) in the right hand, and somewhere between elbows, left forearm, knees and anterior aspects of thighs, managed to grip the note-book—and proceeded to take notes. There was always something satisfying in writing prettily under these circumstances; many of our notes were prettier than what we do sitting à la Palmer method at a desk. But a super-refinement came. The combination pencil (or pen) and flashlight. This took the whole note-taking business out of the realms of technique and reduced it to a semi-automatic mechanical process. It left too much time to think about the subject matter and has led directly to the degeneration of our note-taking. Degenerate it did from here on.

As soon as our mind was occupied only with what the lecturer was saying and doing, and the mechanics of note-taking no longer provoked the invigorating thrill of masterly exercise of a technical skill, we found our notes going along well for a line or so, then the words became more scrawly; finally, the words trailed to the vanishing point and were nothing, absolutely nothing. This was not as unsettling as it sounds, for we soon developed a lethargic, contented sort of satisfaction in the technique of dozing in a well-illuminated room without the lecturer or neighbors giving overt evidence of being aware of our sleeping. True, once in a while our technique slipped, and a neighbor was constrained to nudge an interruption to what he would later allege was noisy respiration, but, in a general way, we continued to bask in the self-satisfaction that comes from attending lots of medical meetings, *der ewige Student* type of reputation. Note-taking in itself, you see, had become as effortless as we'd like to have everything else in this life.

Disquieting news reaches our ears now, though. We've been told about people who go to meetings, take notes, rush to their hotel rooms, and transcribe the notes immediately! Now these folks are going to get ahead of us. They will have not only the reputation of going to great numbers of medical meetings, but will be insufferable enough to bring around their well-written notes, and, what's worse, be able to interpret them. This blow has been softened by the underground intelligence that slow decay is tanglefooting the adherents of this school. They have taken to transcribing their notes to the accompaniment of a long, cool drink—after a hot, tiring day in the lecture hall, you know—and the net result is reported to be like that in the system we have; i.e., first line or two is wonderfully written and then the words gradually become as blurred as the thoughts they express.

The other source of disquiet is in line with the reports of the colleagues who take portable dictating machines in their automobiles and dictate patients' histories while en route to or from house calls, weekends, and such like; or the folks who have machines to read tape-recorded medical literature to them while they drive. Insofar as note-taking goes, this type of efficient perfectionist rents a dictating machine at the meeting place, takes notes, rushes to hotel room, transcribes notes onto recording disc or belt, mails record home to his secretary, and has the TYPED notes waiting on his desk when he gets back. This is nothing if it isn't unsporting and, though we are terribly, terribly hurt by this win-regardless-of-cost attitude, we shall simply ignore this school of note-taking.

This brings us back to the good old method requiring no special equipment, available to all of us from the lowest type of specialist to the highest type of provincial physician, namely, alternate dozing and note-taking with a blunt stub of a pencil on the back of a tattered envelope. It gives you just as much satisfaction as any other method, takes just as much know-how, and will be read and referred to just as often and with equal yield as any flossier technique. And what's more, it gives time to observe that alert gent in the front row who listens eagerly, whose eyes fairly glitter with the joy of the game, and who gets up after the talk and, with obvious enjoyment, intelligently punches holes in the lecture and the lecturer.

H.G.M.

THE VALUE OF SPECIALTY BOARDS

With the unprecedented growth of medical science in the twentieth century came the pressing need for specialization to encompass newer and more refined developments in practice. Traditionally, Vienna and Berlin became the mecca for hordes of American physicians seeking postgraduate training. While many of these men were soundly grounded and motivated, the lack of standards and of measured attainment led to a sense of insecurity in the total program. Too frequently the sole evidence of special training was a series of patently meretricious certificates in the physician's office.

American medicine became increasingly aware of the necessity of keeping its house clean. As in other respects of public welfare, recourse to moral suasion rather than legal action was sought. The movement for the regularization of specialty training was initiated in 1916 by the foundation of the American Board for Ophthalmic Examination. Its terms of limitation were expressed as follows: "Each certificate granted or issued does not of itself confer or purport to confer upon any person any degree or legal qualifications, privileges or license to practice ophthalmology. Nor does the Board intend in any way to interfere with or limit the professional activities of any duly licensed physician." In the intervening years, eighteen additional specialty boards have been established, and, as in the case of the American Board of Ophthalmology (revised designation), recurrent expressions of the designs in the certification of specialists have been made. For example, the American Board of Surgery stated, "The prime object of the Board is to pass judgment on the education and training of broadly competent and responsible surgeons, not who shall or shall not perform surgical operations. The Board especially disclaims interest in or recognition of differential emoluments that may be based upon certification."

The Advisory Board for Medical Specialties, established in 1934, has co-operated with the Council on Medical Education and Hospitals of the American Medical Association in guiding the foundation of new boards and in establishing broad, general principles of procedure. However, the individual requirements of the several Boards have been derived without external influence. For example, in the early operation of the American Board of Internal Medicine, the conventional formula for admission to the examination was three years of residency and two years of the practice of internal medicine after graduation from a Class A medical school and one year of internship. After a period of years, the experience of the Board dictated a much more liberal policy, so that currently it is possible for a candidate to be admitted to this examination under one of several plans of preparation. Indeed, twelve years of the practice of internal medicine after the internship without a formal residency is considered a sufficient discipline. The ability of the candidate rather than the strictures of arbitrary terms of sharply defined service establishes his eligibility for certification.

By common consent, the specialty boards have contributed immeasurably to the improvement of the quality of medical service. The standards of specialty training in this country have improved apace as was anticipated when the several Boards were established. In a period when continental medicine deteriorated to an all-time low, the curve of medical education in this country showed an up-swing. In a material degree, this advance can be directly or indirectly traced to the movement for certification in the specialties. With the available facilities, new incentives for professional advancement were afforded a greater number of physicians. Furthermore, direction was lent to graduate training where none had existed before the advent of this program.

An overlooked dividend of certification in the medical specialties is the requirement of basic qualifications for candidates in the sub-specialties. In an earlier day, the acquisition of a gastroscope and a short course in gastroscopy abroad sufficed to qualify a physician in gastroenterology. The purchase of an electrocardiographic apparatus was tantamount to recognition as a cardiologist. Now such candidates must prove their adequacy in internal medicine before they can be admitted to the examination in the medical sub-specialty. By this simple expedient, the standards in the medical sub-specialties have been immeasurably advanced. Never has the American public been so well protected in the insurance of the qualifications of the practitioners in the various specialties of medicine and surgery.

Unfortunately, certification in medicine and surgery has not been an unalloyed blessing. Perhaps its greatest drawback has been the curious psychologic reaction of "working for the Boards." Perchance this response of immature minds is inevitable. Specialty boards set minimal standards of professional adequacy. Any interference with the continuing education and sustained growth of physicians must be met by positive measures. Unfortunately, the yardstick of certification is applied to the appointment and advancement of physicians in various capacities. The most pernicious device in its utilization has been its requirement by certain hospitals for staff appointment in spite of the expressed design of the several specialty boards. Conversely, the lack of certification has been used as a lever to deny staff appointments or to remove proved clinicians from established posts. Such abuses of the medium of certification are as unfair as they are reprehensible. Perhaps the leveling influence of time and experience is required in all good works.

The principle of supply and demand will unquestionably dictate changes in the programs of specialty training. Certification is a means to the end and not the end itself. The mechanism for rating the qualifications of specialists will vary widely among the several branches of medicine and surgery. Any formula that improves the quality of medical service should be encouraged; but the worthy efforts to broaden the base of training and to encourage the continued growth of specialists must not be submerged by the specious and hollow objective of mere certification.

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CHILD PSYCHIATRY

The rapidly growing field of social psychiatry has developed largely from the application of psychoanalytic theory to general behavior. This has changed considerably the nature of psychiatric practice. The psychiatrist, with few exceptions, no longer limits his work to the treatment of the more serious disturbances such as psychosis and psychoneurosis. He plays a more active rôle in the treatment of the many social and emotional maladjustments to life, and these include the broad fields of marital adjustment, delinquency, psychosomatic illness, and the art of living.

Research in the field of human behavior has demonstrated that the way a person feels, believes and behaves in adult life is largely conditioned by the early emotional experiences to which he was subjected. This has placed tremendous importance on the early years of life and has provided a large body of facts concerning the early development of personality.

There has been considerable research recently on the first few months of the infant's life, growing out of the observation that emotional pathology in the young child is due to the absence of a strong positive emotional tie between the infant and his mother. We are beginning to understand early childhood psychosis much better since this research began and we are rapidly developing a treatment program to deal with this condition. The facts gathered from this research have already been extended to other emotional problems of early childhood which are becoming more clearly understood for the first time.

Studies of children, as revealed through child guidance clinic work, demonstrate that children of all ages develop emotional pathologic conditions when there is something wrong in the relationship between them and their parents.

Rarely does one see a child who presents emotional pathologic conditions of any nature—whether it be night terrors, soiling, school failure, inability to get along with other children, hostile aggression, or delinquency—where the emotional relationship between the child and his parents is a healthy one. Almost invariably there is conflict between the parents, growing out of incompatibility, disloyalty, or the many other forms of family pathologic situations.

As a result, in order to effectively treat the emotional conflict or illness of the child, one must work intensively with the parent-child relationship, the problems in the parents themselves, and any problems created within the family. The best way to deal effectively with these problems is for the so-called team of social worker, clinical psychologist and psychiatrist to work together intensively with the child and his parents and all the many emotionally charged situations to which the child is subjected. This task can seldom be accomplished by the child psychiatrist alone; it is too complex and requires the skills of the several disciplines.

Recently, group therapy has been added to the psychiatric treatment of the child and we have found this to be an invaluable aid in the understanding of a child as he relates himself to other children.

The body of information required to carry on psychotherapy of children intelligently is so extensive, and the particular skills in understanding and helping a child are so important that a long period of intensive training is absolutely essential before a psychiatrist is prepared to do psychiatric treatment work with children.

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ANNUAL MEETING

The 102nd annual meeting of the Minnesota State Medical Association will be held in Minneapolis, May 23-25, at the time of the 100th anniversary of the Hennepin County Medical Society.

Business sessions of the Council and House of Delegates will precede the scientific sessions which will begin Monday with meetings in the Minneapolis Auditorium. A tentative program will appear in the April number of Minnesota Medicine, and the official program should reach members about May 1.

THE VALUE OF A SAFETY DEPOSIT BOX

Century after century, down through the ages, the primary concern of man, after providing a livelihood for himself and his family, has been the protection of the surplus wealth accumulated as a result of his labor. Closely paralleling the development of the family unit into an organized society, the protection of the concentrated money of the community has been the responsibility of the banker. With this responsibility arose the problem of creating a depository which would safeguard the money and valuables entrusted to his care.

Ingenious, yet crude were the early methods employed, but from these humble beginnings evolved the modern steel vault, equipped with protective devices and guarded by massive doors weighing many tons, which today make the vault a marvel of safety and convenience, storm-proof, burglar-proof and fire-proof—safe against almost any act of nature or man.

The modern bank, by popular demand, has incorporated in its security function a deposit section containing thousands of safe deposit boxes in varying sizes which are available to the general public. It is not difficult to conceive the value of a safe deposit box for private use when it is realized that the legal evidence of much that mankind possesses, is on paper—paper which if lost, stolen or destroyed, might be impossible to replace. Insurance will help rebuild a home, purchase a new car, furniture or clothing after a catastrophe, but there are many items of intrinsic or sentimental value together with irreplaceable papers and documents which money can never restore.

The use of the safe deposit box is limited only by the imagination of the holder; it is not only a convenience but a means of protection also frequently utilized as an important tool in managing financial affairs in a systematic manner. It is peace-of-mind insurance for the individual, partnership or corporation—one of the most important yet comparatively inexpensive banking services offered.

Rollin O. Bishop
President, American National Bank
Saint Paul, Minnesota

The Dean's Page

UNDERGRADUATE EDUCATION FOR GENERAL PRACTICE

The trend toward specialization in medicine has been discussed with increasing frequency in recent years, and more often than not it has been deplored. Yet medicine's growing complexity suggests that this trend will not be reversed. On the other hand, the well-trained general practitioner will continue to play a vital role in the care of the sick. There can never be an acceptable substitute for the close personal relationship between a patient and his "family doctor."

The principal objective of a medical school is to provide its students with the basic tools essential for general practice. The same tools are equally necessary for the future specialist, teacher, or investigator. To the end of providing their students with a better background for medical practice, a number of schools have recently introduced changes in their curricula, for example, "integrated" teaching, family care plans, preceptorships. Although differing in their forms, each of these programs represents an attempt to give the student a broad view of medicine rather than a series of compartmentalized peeks.

Tradition and the appreciation of socio-economic realities allow us but four years for the formal education of a physician—a period unchanged in more than four decades. Yet today's student must assimilate an infinitely greater amount of information than did the student of 1915, considerably greater even than did the student of 1940. Two closely related conclusions may be drawn from this. First, medical students must be made aware that their education is, in fact, a life-long process and that their years in medical school represent only its beginning; and second, medical schools must plan their curricula with this in mind.

In light of an ever-expanding body of medical knowledge and a fixed period for formal undergraduate training, how may a medical school make best use of the four years during which its faculty is responsible for the education of its students? In our opinion, it must see that before graduation its students acquire the following:

1. A comprehensive knowledge of the basic medical sciences. If a student fails to acquire a real understanding of the basic sciences in medical school, it is most unlikely that he will do so after graduation.

2. The fundamentals of clinical practice in each of the various fields. This means that the student must be taught primarily the approach that is required in solving clinical problems.

3. An appreciation of the role of research in the continuing growth of medicine.

4. A knowledge of how to use medical literature effectively and, along with this, the background and attitude necessary for the critical evaluation of reports of new fundamental, diagnostic, and therapeutic developments.

5. An appreciation of the absolute necessity for integrity and adherence to the ethical code in the practice of medicine and a realization that the physician must accept civic and social responsibilities apart from his professional responsibilities.

6. An awareness of the various paramedical agencies, for example, public health, social service, occupational therapy, which can help him in rendering maximum service to his patients.

If we accept the foregoing as reasonable objectives for the medical school, two corollaries follows: (1) The general caliber of the internship, as an integral part of medical education, must be improved; and (2) The practicing physician will have to make increasing use of the various forms of continuing medical education.

In summary, the best undergraduate teaching program is one which will furnish the student a sound foundation on which he may base his future training. His personal preferences and interests may take him into general practice, specialty practice, teaching and research, public health, or administration. Regardless of his later choice, such a background will make him a better physician.

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Medical Economics

Edited by the
Committee on Medical Economics,
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HEALTH MESSAGE SHOWS LITTLE CHANGE

President Eisenhower's health message to the Congress carried no recommendations that had not been made earlier in his State of the Union or budget messages, except his request for a bigger U. S. contribution to World Health Organization. The AMA congratulated the President on his statement that health proposals "recognize the primacy of local and state responsibility" and would "encourage private efforts with private funds."

His comments and basic outlines for health programs are:

Reinsurance.—The Administration wants Congress to appropriate $25 million to start a federal reinsurance fund for the use of voluntary health insurance groups. The President said reinsurance would be most helpful in three broad areas (a) with rural families, (b) to expand catastrophic insurance, and (c) to bring protection to low-income families in the form of coverage for home and office calls as well as hospitalization. Last year the House defeated a reinsurance bill that followed the same lines but that did not pinpoint certain areas. This was introduced in the omnibus health bill S. 886.

The AMA's official comment on this proposal was that the AMA has been in complete accord with the stated purpose of legislation designed to promote voluntary health insurance and commended the President for his beliefs and efforts in encouraging its expansion. However, the AMA still believes that the proposed reinsurance system will not achieve the desired results.

Medical Care for Indigents.—The President described the present medical care program for public assistance recipients as "far from adequate." He proposed $20 million more in U. S. appropriations, to be matched by the states and placed in a separate fund exclusively for medical care payments. This fiscal year the U. S. is spending about $80 million in this field. The AMA was not prepared to comment on this proposal yet.

Mortgage Guarantee.—The Administration is offering an amended version of last year's Kaiser-Wolverton bill for U. S. guarantee of private loans for health facilities. The President said: "Present methods of financing are not always satisfactory in meeting this problem. Many sponsors and operators are unable to qualify for grants under the recently extended hospital survey and construction act (Hill-Burton). Sponsors of health facilities often find it difficult to obtain private capital for construction." The AMA said it has no opinion yet on this measure.

Health Personnel.—Three methods are proposed to increase the supply of trained personnel, (a) grants to states for training practical nurses, (b) Public Health traineeships for graduate nurses in specialty nursing fields, and (c) establishment of PHS traineeships in public health specialties, including mental health. The AMA commended the President for his support of these proposals.

Mental Health.—Three approaches recommended (a) more aid to states and communities, (b) more money to train personnel for care of mental patients under present authorizations, (c) a new program of project grants aimed at improving the quality of care in institutions and searching out ways of reducing length of stays. The latter point is in recognition of the fact that the care of mental patients is the heaviest financial burden in the medical fields for most states. The AMA agreed with the President on this matter also.

Also included in the President's recommendations to Congress is the extension of the "Doctor-Draft" act. This is incorporated in H.R. 2886, which simply extends the act for two years.

PRESIDENT'S BUDGET IS EXTENSIVE, EXPENSIVE

The Administration has presented Congress its formal request for funds to maintain all programs, including health and medical, for the 1956 fiscal year starting July 1, 1955. The total request for the Department of Health, Education and Welfare is $2 billion, just a slight increase over the current year's estimated spending.

Included in this amount is the request for Hill-Burton hospital construction funds amounting to $125 million. This will be for new facilities and grants to complete hospitals now under construction.

Increased funds were also requested for research projects, some as much as 50 per cent.

VA Medical Care Costs

Medical care costs for the Veterans Administration were set at almost $889 million, compared with $862 million in current spending; hospital construction costs were set at $60 million (mostly remodeling) as compared to the current $50 million.

In his budget message the President expressed concern that the VA patient load would rise 4 per cent next year and that:

> ". . . more than two-thirds of the expenditures will still be for patients hospitalized or treated for ailments not connected with military service. . . . we must bear in mind that government policies designed to assist in the maintenance of a prosperous economy and to support social security, health, and other humanitarian programs are all of value to veterans as well as to other people. Since more than two-fifths of all adult males are entitled to veterans' benefits, expenditures for veterans are a budgetary problem of major interest to the whole population."

STANDARD HEALTH INSURANCE PLAN PROPOSED FOR U.S. WORKERS

The Administration has revised its plan for health insurance for federal employes and is proposing a three-way option for them: participating in (1) a new standard, nationwide plan, (2) existing plans sponsored by their own national employe organizations, or (3) local plans offered in their community or through local employe unions or other agencies.

The standard plan would offer catastrophic or major medical care insurance. It provides:

1. U. S. estimated contribution would be $55 million a year, paying up to one-third of premium costs, with a limit of $1.50 per month for employes without dependents and $4 per month for those with dependents. If the employe chooses the standard national coverage, the cost to him would be $3 per month if he has no dependents and $8 per month if he has dependents. On other types of coverage, the employe's payments would vary with the premiums, but U. S. contribution would still not exceed the above figures.

2. The standard plan would be straight cash indemnity based on a schedule of benefits set by the Civil Service Commission.

3. Major medical cost protection would be restricted. After the policyholder had exhausted the benefits of the basic policy, he would have to meet the next $100 of expenses himself. Then, the standard policy would reimburse him for 75 per cent of the costs up to a maximum of $2,500.

4. After a minimum of fifteen years' civilian service, employes who retire would be permitted to continue under the program by authorizing premium deductions from their retirement checks.

5. Civil Service Commission would be responsible for administration of the entire program, but would contract with a single insurance company to operate the national standard plan. It would not become operable, however, unless 600,000 of the 1,800,000 eligible government workers agree to participate.

COMPULSORY HEALTH INSURANCE AGAIN INTRODUCED

H.R. 95 is a verbatim copy of H. R. 1817 introduced in the 83rd Congress two years ago, providing, among other things, for national compulsory prepaid personal health insurance. Its author is Rep. Dingell of Michigan, author of the previous bill also. It also provides for subsidies for federal aid to medical education, medical research, hospital construction act amendments, special aid for rural and other shortage areas, state grants for local public health units, research in child life and additional grants for maternal and child health and crippled children's services.

M.D. SHORTAGE IS A FADING SPECTER

A recent editorial in *Medical Economics,* the monthly magazine, notes the rapid rise in the number of medical schools in the nation, to help refute the idea that there is an acute doctor shortage in America.

The editorial stated: "For years there's been agitated debate about a national doctor shortage. Medical men couldn't stop it by pointing to the increasing size of graduating classes pouring forth from existing medical schools."

Reasons given were that the population was increasing just as fast. For, between 1940 and 1950 the total number of physicians in this country increased by 15 per cent; but the total population increased 15 per cent too.

But, the editorial notes what is happening today: Two new medical schools (the University of North Carolina and the University of Puerto Rico) produced their first graduating classes within the past year. Eight more new schools (U.C.L.A., Albert Einstein, Seton Hall, and the universities of Miami, Missouri, Mississippi, West Virginia, and Florida) are ready for early training of doctors.

The editorial quotes statisticians as believing then "the rise in the number of medical graduates should be relatively more rapid than general population increases in the course of the next six to ten years."

In conclusion, the *Medical Economics* editorial says:

> "This heartening break-through doesn't solve *all* our doctor-supply problems. There's an unquestioned shortage in certain local areas and in certain key specialties. But at least the specter of a *national* doctor shortage should begin to fade."

BUSINESS HAS AN INTEREST IN HEALTH

According to Benson Ford, president of the Board of Trustees of Detroit's 650-bed Henry Ford Hospital, business has a substantial interest in health and health problems throughout America. Mr. Ford is also vice president of the Lincoln-Mercury division of Ford Motor company.

In a speech at the 56th annual convention of the American Hospital Association, quoted in the December, 1954 issue of *Hospitals,* Mr. Ford said that "of the 43 million Americans who take part in Blue Cross, a large majority are covered by industrial group plans. Industry also foots part of the bill for millions more Americans in other insurance plans. Add to that the cost of private industrial medicine, insurance against industrial accidents and illness, and it becomes clear that business has a substantial interest in the whole health system of our country.

Mr. Ford outlined the broader problem of union demands on industry, the aging population and other problems which affect industry's interest in health matters. But he stated:

> "Up to this point, I seem to have painted a picture of a pretty sadly-confused health situation. That would hardly be a true or fair picture. Most of our present difficulties are inherent in the nature of the private enterprise system. Ironically, it is the very high quality of our private medicine and our partial success in developing private means of financing medical care that make it possible for us to enjoy some of these worries I've been talking about."

Suggests Permanent Health Commission

Mr. Ford suggested a solution to the national problems of health matters in a health commission representing all the major segments of the health profession, to examine our major health needs and to recommend how best these needs can be met.

"I'm not suggesting a super-lobby to fight socialized medicine or a 'politburo' to dictate hospital and medical practices," Mr. Ford continued. "I have in mind a top-level professional group adequately staffed to study the developing pattern of wants and demands and to establish standards and practices for the guidance of all our services, including hospitals, clinics, medical schools and medical research centers."

In conclusion, he said, "I think we must all recognize the existence of a broad public desire for a kind of health care commensurate with the vast wealth and resources of our nation." He declared:

> "If you, the professionals of health, can supply the drive and leadership to get that job under way, I am sure you will win the earnest gratitude and also the eager co-operation of industry, of labor, of government and of all the American people in helping you solve a great public problem by private action."

Public Health

DUTIES OF THE PHYSICIAN IN REGISTERING BIRTHS AND DEATHS

The physician is the first person to take official note of a birth or a death. On him, therefore, rests the responsibility for prompt and accurate reporting of these events occurring in his practice. Laws regarding the reporting of vital events are changed from time to time. For the help and guidance of physicians, the Minnesota State Board of Health herewith presents the current requirements.

The Vital Statistics law requires the physician to:

(a) File a birth certificate for every live birth he attends.

(b) Certify the cause of death when he is the physician last in attendance, unless the case is under coroner's jurisdiction.

Birth Certificates.—The Vital Statistics law requires the attending physician to file, within five days, with the local registrar of the district where birth occurs, a birth certificate for every live-born infant he has attended.

Exception.—A birth certificate for a child *born out of wedlock must be filed directly and promptly with the state registrar,* 469 State Office Building, St. Paul 1, Minnesota. (Local registrars are clerks of the District Court, except in cities which by ordinance maintain a primary registration district. In such cities, of which there are twenty-two in Minnesota at present, the health officer is the local registrar.)

Reportable Births.—Live births and stillbirths are reportable after twenty weeks' gestation.

Live Births.—The standard definition of a live birth is "an infant which either breathes or demonstrates heart beat or movement of voluntary muscle after it is completely outside the body of the mother." Live births are to be reported on the *Live Birth* certificate; stillbirths on the *Stillbirth* certificate.

Hospital Births.—A birth record is a legal record and is of great importance to the person throughout his life. When hospital personnel prepare birth records for the physician, he is nevertheless responsible for their legibility, completeness, and verity. He must sign them in his own handwriting. They must also be dated. If the physician is not satisfied with their appearance or content, he should require that they be made out again before he signs them.

Stillbirths.—If a funeral director takes charge of the body of a stillborn infant, the law requires him to prepare a certificate and to present it to the physician for certification of the causes indicated. If there is no funeral director, the hospital or the physician may take the initiative in preparation of the certificate.

Death Certificates.—The funeral director is required to present the death certificate to the physician last in attendance, for certification of the cause of death, unless the coroner has jurisdiction, in which event the certificate must be presented to the coroner. Physicians and coroners should promptly certify the cause of death and immediately thereafter return the certificate to the funeral director so that he can file it and obtain the required permits before burial or other disposition of the body.

Cause of Death.—Cause of death should be certified in full (abbreviations should not be used) according to directions set forth in the World Health Bulletin, Medical Certification of Cause of Death, obtainable from the Section of Vital Statistics, Minnesota Department of Health, 469 State Office Building, St. Paul 1.

Autopsy Pending or Diagnosis Deferred.—If autopsy is pending or diagnosis is deferred for some other reason, the physician or coroner should so state in the death certificate. He should date and sign the certificate and return it at once to the funeral director. At a later date, the state registrar will obtain a statement of the cause of death from the physician or the coroner.

It may be of interest to physicians to know that Minnesota rated 99.9 per cent completeness in the registration of births in the 1950 birth registration test conducted by the Bureau of the Census in co-operation with the National Office of Vital Statistics and the Vital Registration Executives of the United States. Minnesota was tied with Rhode Island for second place among the states, exceeded only by Connecticut with a rating of 100 per cent. Credit for this achievement was due to the physicians, hospital personnel, and local registrars of Minnesota who have most faithfully fulfilled their legal obligations regarding birth registrations. During the test period studied, only two Minnesota physicians failed to report. Constant co-operation between local and state officials is necessary if the high standard of vital reporting in this state is to be maintained.

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Current Concepts

DIET AND ATHEROSCLEROSIS

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The theory that the diet may affect the development of coronary heart disease is not new. For years, the balance between pro and con teetered under the accumulation of indecisive evidence from animal experiments, often having little relevance to the real problem in man. Recently. however, the increasing dominance of coronary heart disease in our national health picture has stimulated much more research, and some of this has been more systematic and more directly concentrated on man himself. The result is that we can now see some of the outlines, if not the details, of a major relationship between the habitual diet and the tendency towards atherosclerosis and resultant coronary heart disease.

Perhaps the most crucial evidence is that human populations who subsist on different diets show great differences in the prevalence of clinical coronary heart disease, in the age-specific mortality ascribed to coronary or degenerative heart disease, and in the incidence of atherosclerosis seen at autopsy. The old impression that coronary heart disease may be rare in some populations, such as the Chinese, has been replaced with conclusive data. It is now clear, for example, that middle-aged men in the United States have perhaps ten times as much atherosclerosis, and die far more frequently from coronary heart disease, than do South African Bantu, or Japanese of the same age. It is not simply that white men are more afflicted than men of the Mongol or Negroid races, because there are also notable differences between white men. We may contrast Minnesotans with Italians or Spaniards, for example, and point to differences between white men in the same country, such as rich versus poor Spaniards, or Italians in Naples versus Italians in Bologna. It is most significant that the indicated differences between populations in regard to coronary heart disease appear to be parallel to differences in the average concentration of cholesterol in the serum in samples of these populations.

The unifying concept that brings order into an otherwise mystifying picture is that the proportion of fat in the diet has a powerful effect on the serum cholesterol level and so on the development of atherosclerosis. The dietary cholesterol itself is unimportant, but whether different neutral fats in the diet have different effects is as yet undecided. The important point is that all of the data —and they come from Guatemala, Sweden, and England as well as from Spain, Italy, South Africa, Japan and the United States—show a remarkably regular and direct relationship between the total fat content of the diet and the prevalence of coronary heart disease at given age. Further, the experiences of World War II and after, when many countries changed their diets, are in close agreement and indicate that dietary changes may have large effects on mortality in populations in a year or so.

It is significant, then, to note that our average American diet is the world's highest in fat and that the proportion of our dietary calories supplied from fats has been steadily increasing for a long time. Contrast our present more than 40 per cent of calories from fats with the 20 per cent figure for the Neopolitan or the 8 per cent figure for the Japanese!

As yet, there is little evidence that dietary manipulation will greatly change the prognosis of the coronary patient, though time may change this picture, but it is already reasonable to hope for real progress towards prevention through dietary means. Obviously, we need much more research. particularly systematic studies on populations which can tell us things not to be learned from our laboratory animals, but the future ahead looks bright. We can now be sure that age alone does not inevitably bring atherosclerosis and coronary heart disease.

It is clearly evident that our public health problems of the future will be concentrated among older persons, among whom chronic disease and disability are more prevalent; their social and economic overtones will have an impact on almost every family. Our plans must encompass the degenerative diseases, the long-term illnesses, and the disabling conditions of older age groups. This does not mean that we can relax our guard against communicable diseases, nor lessen our campaign to reduce infant and maternal mortality, nor let up on our crusade to eradicate tuberculosis. Environmental sanitation is as important as ever and requires constant vigilance to keep the air, water, food, and milk free from pollutants including those spawned by atomic fission. Herman E. Hilleboe, M.D., *New York State Journal of Medicine,* December, 1954.

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

National

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American College of Gastroenterology, southern regional meeting, Hotel Peabody, Memphis, Tennessee, April 24, 1955.

American College of Surgeons, sectional meeting, The Fort Harry, Winnipeg, Manitoba, Canada, April 25-26, 1955.

American Goiter Association, annual meeting Skirvin Hotel, Oklahoma City, Oklahoma, April 28-30, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

American Radium Society, Hotel Shoreham, Washington, D. C., April 21-23, 1955.

Gill Memorial Eye, Ear and Throat Hospital, twenty-eighth annual spring congress, Roanoke, Virginia, April 4-9, 1955.

Industrial Health Conference, Memorial Auditorium, Buffalo, New York, April 23-29, 1955.

Society for the Prevention of Asphyxial Death, New York Academy of Sciences, New York, New York, March 24, 1955.

International

Inter-American Congress of Radiology, Shoreham Hotel, Washington, D. C., April 24-29, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

INTERNATIONAL COLLEGE OF SURGEONS

The United States and Canadian sections of the International College of Surgeons, through the generosity of the Women's Auxiliary, are offering two opportunities to young Canadian or American surgeons.

One is a scholarship of $3,000 to pay for transportation and living expenses for one year for a young surgeon who wishes to study abroad. The doctor selected for the scholarship will be expected to spend at least ten months of the year as a resident or fellow in a teaching center in Europe or South America.

The other is an invitation to two residents or fellows in training in surgery to attend the annual meeting of the college and present a clinical or scientific paper. Transportation and living expenses during the meeting, not to exceed $300, will be provided by the college. The next annual meeting will be in Philadelphia from September 12 through September 15.

Further information concerning both offers can be obtained from the Scholarship Committee, International College of Surgeons, 1516 Lake Shore Drive, Chicago, Illinois.

AMERICAN INSTITUTE OF DENTAL MEDICINE

The next annual meeting of the American Institute of Dental Medicine will take place at the Desert Inn, Palm Springs, California, October 23 to 27. The faculty will consist of Maury Massler, D.D.S., M.S., University of Illinois, Chicago; Valy Menkin, M.D., Temple University Medical School, Philadelphia; Hans Selye, M.D., University of Montreal, Canada; Reidar F. Sognnaes, D.M.D., Ph.D., Harvard School of Dental Medicine, Boston; Wendell L. Wylie, D.D.S., University of California, San Francisco.

All seminar lecturers will participate in a round table forum discussing the application of their subject to the practice of dental medicine. Applications and full information may be secured from the executive secretary, Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

COURSE IN CLINICAL PATHOLOGY AND PATHOLOGY OF PARASITIC DISEASES

A short, intensive course on the laboratory diagnosis and pathology of parasitic infections will be presented August 15-27 at the Louisiana State University School of Medicine in New Orleans.

The course is designed primarily for pathologists and technologists. However, general practitioners, internists, pediatricians, gastroenterologists and physicians engaged in the practice of public health and tropical medicine who are interested in the laboratory diagnosis of parasitic infections are welcome to attend. The instruction and training will be of assistance to pathologists who are preparing for board examinations, to pathologists and physicians who are responsible for the diagnosis of parasitic infections in their laboratories and to technologists engaged in this specialty.

Registrants should bring their microscopes, equipped with mechanical stages, and their microscope lamps. A limited number of places will be available. The fee for the course is $50.

Persons interested in attending this course may write to: Dr. Clyde Swartzwelder, Department of Microbiology, Louisiana State University School of Medicine, 1542 Tulane Avenue, New Orleans 12, Louisiana.

PAN AMERICAN MEDICAL ASSOCIATION

The next and tenth Inter-American Congress of the Pan American Medical Association will be held in Mexico City. The scientific sessions will begin on March 25, 1957. The Congress will be held in sections covering all branches of medicine and surgery. There will be medical moving pictures, panel discussions and scientific and technical exhibits. The association has forty-two medical sections including the new Section of General Practice.

Four days will be devoted to scientific sessions, and the next three days will be spent in sight-seeing, with visits to Cuernavaca, Taxco and Acapulco. The following week medical meetings will be held in Guatemala City in conjunction with the local chapter there.

The president of the Association is Dr. Pedro A. Gutiérrez Alfaro, Minister of Sanitation and Public Welfare of Venezuela, and the executive director is Dr. Joseph J. Eller, 745 Fifth Avenue, New York, New York.

SURVEY OF POSTGRADUATE MEDICAL EDUCATION

The Council on Medical Education and Hospitals of the American Medical Association has completed a comprehensive survey of postgraduate medical education in the United States. The material collected during the study has been worked into a final report and is being published as a series of special articles in the *Journal of the American Medical Association*. The first study titled "The Scope and Extent of Postgraduate Medical Education in the United States," was published in the February 26 number. Others will appear as follows: March 12, "The Physician as a Lifelong Student. March 26, "The Objectives and Content of Postgraduate Medical Education." April 9, "Educational Methods in Postgraduate Teaching." April 23, "Time and Place Arrangements of Postgraduate Courses for Practicing Physicians." May 7, "Sponsorship and Administration of Postgraduate Medical Education." May 21, "Financing Postgraduate Medical Education." June 4, "The Future of Postgraduate Medical Education."

ART AND HOBBY SHOW AT STATE MEETING

An exhibit of art and hobby projects is to be shown at the Minnesota State Medical Association annual meeting, scheduled for Minneapolis on May 23, 24 and 25. The exhibition will be more inclusive than ever before; not only paintings in various media, photography and sculpture are being shown, but also any hobby projects which are suitable for this type of exhibit. Complete information and entry blanks will be available in April from the State Office, 496 Lowry Medical Arts Building, St. Paul. Any Minnesota doctors doing any of the above type of art or hobby work are encouraged to exhibit. The showing will be appraised by competent judges, and numerous awards will be given. Co-chairmen for this event are Dr. J. S. Milton and Dr. F. A. Zinter, Minneapolis.

PUBLIC HEALTH RADIO-TV SERIES

A thirteen-week series of programs on public health will be presented on stations KSTP and KSTP-TV, Twin Cities, beginning March 9. The programs, which will consist of interviews of public health officials by Bee Baxter, radio and television personality, have been planned in co-operation with the section of public health education of the Minnesota Department of Health.

The interviews will be carried on television (KSTP-TV) on Wednesdays at 1:30 p.m. and on KSTP radio on Thursdays at 11:30 a.m.

Persons to be interviewed on the series include Dr. R. N. Barr, Dr. Helen Knudsen, Dr. Henry Bauer, Dr. Dean S. Fleming, Alberta Wilson, Dorothy Hagland, Dr. A. J. Chesley, Dr. Carl Lundeberg, Dr. A. B. Rosenfield, Frank L. Woodward, Herbert Bosch, Dr. Dale C. Cameron, Marie Ford and Dr. Gaylord Anderson.

MINNESOTA HEART ASSOCIATION

Minnesota institutions and individuals desiring consideration of Grants-in-aid and Fellowships in support of cardiac research and study from the 1955-56 research budget of the Minnesota Heart Association must present their requests on or before May 1, 1955.

Approved grants will extend from July 1, 1955, through June 30, 1956.

Further information may be secured by writing the Minnesota Heart Association, 532 Endicott on Fourth, Saint Paul 1, Minnesota.

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

F. H. Magney, M.D., Secretary

License of Arthur F. Sether, M.D., Suspended for Three Years

On February 4, 1955, the Minnesota State Board of Medical Examiners suspended for a period of three years the medical license held by Arthur F. Sether, M.D., of Ruthton, Minnesota, because of his violation of the terms of a stipulation which Dr. Sether signed following his personal appearance before the Board on May 18, 1951, in response to a citation for the revocation of his license. In that stipulation, which Dr. Sether entered into with the Board on August 23, 1951, in addition to other provisions he agreed to refrain from the personal use of the so-called barbiturates and alcoholic liquors in every form. It was because of the violation by Dr. Sether of this portion of his agreement with the Board that his medical license was suspended.

Dr. Sether, who was licensed to practice medicine in Minnesota in 1935, was born in St. Paul, Minnesota, on November 5, 1905. After taking a one-year internship at Minneapolis General Hospital, Dr. Sether graduated from the University of Minnesota with an M.D. degree in 1936. In the following year Dr. Sether opened an office for the practice of medicine at Ruthton, where he has practiced since that time.

Woman's Auxiliary

MID-WINTER MEETING WELL ATTENDED

Mrs. L. Raymond Scherer

The Mid-Winter Board meeting of the Woman's Auxiliary to the Minnesota State Medical Association was well attended despite below-zero weather.

The meeting was held February 10 at the Town and Country Club in St. Paul and was attended by county and state auxiliary officers and committee chairmen.

Members were welcomed by the auxiliary president, Mrs. Peter S. Rudie of Duluth. This was followed by the pledge to the flag and the pledge to the auxiliary, led by Mrs. John Dordal of Sacred Heart.

Regular reports of the secretary and the treasurer were read and approved, followed by a report of the president on the meeting of presidents and presidents-elect in Chicago during November of 1954. Mrs. Rudie stated that the meeting offered an excellent chance for an exchange of information with other state auxiliary presidents, and she reported on the many excellent panel discussions, including such subjects as organization, program, publications, *Today's Health,* the American Medical Education Foundation, public relations, legislation, civil defense, mental health, allied medical careers, and finances.

Following Mrs. Rudie's report, members heard a report from the president-elect, Mrs. H. H. Fesler of St. Paul, and the second vice president, Mrs. V. J. Schwartz of Minneapolis. Then followed reports from regional advisors in attendance: Mrs. O. M. Heiberg of Worthington, Mrs. Ralph Creighton of Minneapolis, and Mrs. J. C. Buscher of St. Cloud who reported on activities of auxiliaries in their districts.

State committee chairmen reported on various activities of their committees; reports were heard on Bulletin, legislation, organization, program and health education, public relations, American Medical Education Foundation, health days, newsletter, school of instruction, medical and surgical relief.

County auxiliary presidents in attendance told of individual county auxiliary projects. Reports included were from presidents of the following county auxiliaries: Hennepin, Park Region, Ramsey, Renville-Redwood, St. Louis and Stearns-Benton.

Following the completion of the business meeting, auxiliary members attended the luncheon meeting, presided over by the president. Mrs. Rudie introduced Mrs. V. J. Schwartz, Minneapolis, second vice president, who in turn, introduced Miss Marguerite Breen, Director of Public Relations for the Minnesota Tuberculosis and Health Association. Miss Breen thanked the state auxiliary for its many years of participation in her organization's program of promoting tuberculosis essay contests among high school and junior high school students in Minnesota. She exhibited the annual plaque and trophy given to the best essay on tuberculosis by a junior and senior student each year. The auxiliary sponsors this project jointly with the tuberculosis and health group, and Mrs. Schwartz served as a judge for the 1954 awards. Miss Breen then presented Barbara Sitzman, senior division winner, and Judy Lewis, junior division winner, who presented their prize-winning essays. Both girls are from St. Agnes High School in St. Paul. Auxiliary members agreed that their essays were of excellent quality and fully deserving of the awards they won.

RAMSEY COUNTY AUXILIARY REPORTS ACTIVITIES

Mrs. L. T. Simons

The State Medical Auxiliary Mid-Winter Board meeting was held on February 10, 1955, at the Town and Country Club, St. Paul. Mrs. W. H. Von Der Weyer and Mrs. B. E. O'Reilly were in charge of hospitality. Ramsey county members present were: Mrs. W. P. Gardner, president, Mrs. H. O. Peterson, president-elect, Mrs. Charles Waas, Mrs. C. W. Froats, Mrs. J. L. Benepe, Mrs. H. H. Fesler, Mrs. Mark Ryan and Mrs. John Ryan. Mrs. P. K. Arzt, chairman of state and Ramsey county legislation, presented both her own report and Mrs. E. M. Hammes' concluding report on the Dr. William A. O'Brien fund.

The Auxiliary took part in the Inter Club Council's "What's Cooking?" program held in the Lowry Hotel on March 28, as its public relations project for the year. Mrs. J. F. Madden and Mrs. J. R. Meade, auxiliary representatives, organized the medical scholarship material used on the education panel. Dr. Charles Rea, St. Paul, a member of the panel, spoke on medical scholarships and their allied fields. Mrs. O. I. Sohlberg served in an advisory capacity on the planning of the legislation panel.

Mrs. R. O. Burmeister, philanthropic chairman, reported a total of 175 cancer dressings. The Auxiliary assists this committee by making dressings during and following the board meetings.

The Medical and Surgical Relief Committee, Mrs. H. F. Schroeckenstein, chairman, packed 29 cartons of supplies in January. These were sent to the Episcopal Mission Orphanage in Osaka, Japan.

Mrs. R. F. Sturley, nurse recruitment chairman, reported her committee has been busy arranging for transportation of nurses to Murray, Roosevelt and Mahtomedi High Schools during February. These nurses talk to junior and senior girls who are interested in nursing as a career.

In Memoriam

ALOYSIUS STEPHEN FLEMING

Dr. A. S. Fleming, formerly of Minneapolis, died January 19, 1955, at Madison, N. J., where he had been living since his retirement in 1950. He was eighty-three years old.

Dr. Fleming was born in Eau Claire, Wisconsin, in 1872. He attended the University of Wisconsin for two years and got his medical training at the medical college of Hamline University. For fourteen years, he practiced in Wheaton, Minnesota, before coming to Minneapolis in 1913.

He was a former member of the staff of Hillcrest Hospital and was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association. He was also a "Fifty Club" member of the Minnesota State Medical Association.

Surving are three sons, Noel C., Madison, N. J.; Dr. Dean S., Hopkins, and Allan W., Duluth; a daughter, Helen Fleming, Madison, N. J., and four grandchildren.

CHARLES HARRY GHENT

Dr. C. Harry Ghent, St. Paul physician, died January 31, 1955, in St. John's Hospital, St. Paul. Dr. Ghent had been ill for four months. He was sixty-six.

Dr. Ghent was born in Centralia, Illinois, in 1888. He graduated from the Northwestern University Medical School in 1911 and interned at Passavant Hospital in Chicago. He practiced in Dawson, Minnesota, and White Rock, South Dakota, before moving to St. Paul in 1916.

He had been a member of the staff of St. John's Hospital since 1916 and was also on the staff of Mounds Park Hospital. He was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Ghent is survived by his wife, Florence; a daughter, Mrs. John B. Rutledge of Chicopee, Massachusetts; a sister, Mrs. John B. Hamilton, Carbondale, Illinois; and three grandchildren.

CHARLES ALBERT HALLBERG

Dr. Charles A. Hallberg, Minneapolis physician and surgeon, died suddenly January 30, 1955, at St. Barnabas Hospital. He was sixty-seven.

Dr. Hallberg was born in Stockholm, South Dakota. He received his medical degree at the University of Illinois in 1911 and interned at University Hospital in Chicago.

In 1917, Dr. Hallberg went to the Mayo Clinic, Rochester, Minnesota, on a fellowship. Except for a period of service during World War I, he remained there until entering private practice. He became an associate in medicine on the Mayo Clinic staff and took his specialist training in surgery there. He entered private practice in Minneapolis in 1926.

A staff member of St. Barnabas and the Northwestern Hospitals, Dr. Hallberg was a fellow of the American College of Surgeons, a member of the American Medical Association, the Minnesota State Medical Association and the Hennepin County Medical Society and the Minneapolis Surgical Society.

He is survived by two sisters, Mrs. Alma Darud, Seattle, Washington, and Mrs. Amanda Oestrich, La Bolt, South Dakota.

RAY GEORGE JOHNSON

Dr. Ray G. Johnson, Stillwater physician, died January 14, 1955. He was fifty-nine years old.

Dr. Johnson was born in Waltham, Minnesota, in 1895. He obtained a B.S. degree from Carleton College in 1920 and his M.D. degree from the University of Minnesota Medical School in 1924.

Following his internship at Mounds Park Hospital in St. Paul, Dr. Johnson practiced in St. Paul until 1938. In 1939, he became resident physician at the Minnesota State Prison at Stillwater.

He was a member of the Washington County Medical Society, the Minnesota State Medical Association, the American Medical Association, St. John's lodge No. 1, and A. F. and A. M.

He is survived by his wife, Ida May; two daughters, Barbara and Janet Rae; one brother, Vaughn, of Terry, Montana; two sisters, Mrs. Howard Crowther, Saint Paul and Mrs. Mabel Jordre of Oberon, North Dakota.

GEORGE P. KIRK

Dr. George P. Kirk, physician of East Grand Forks, Minnesota, died on January 20, 1955, at the age of eighty-one.

Dr. Kirk was born in Minnesota in 1874. He graduated from the University of Minnesota Medical School in 1896.

In 1949 Dr. Kirk, a member of the Red River Valley District Medical Society, became a life member of the Minnesota State Medical Association. He had been a member of the State Association for forty years.

During his active life, Dr. Kirk had been a health officer and city doctor. He was on the staff of St. Michael's and Deaconess Hospitals.

Dr. Kirk is survived by his wife, Anna; two sons, Cyrus of Minneapolis and Paul of Edmonton, Alberta, Canada; one daughter, Mrs. Edward Kuppich of Fargo; a brother, John H. of Glendale, California; a sister, Mrs. Ernest Freivogle of Richfield, Connecticut; and seven grandchildren.

Of General Interest

On February 3, two hundred Winnebago citizens turned out to honor the forty-seven years of service of **Dr. M. D. Cooper.** Dr. and Mrs. Cooper were guests of honor at a testimonial dinner sponsored by the Winnebago Community Club in the Presbyterian Church.

"This Is Your Life" theme was carried out, with many citizens and friends giving short tributes to the doctor's contribution to the life and welfare of the community. **Dr. Ralph Armstrong,** president of the Blue Earth Valley Medical Society, commended Dr. Cooper's long years of service.

In accepting the many honors, Dr. Cooper pointed out that when he arrived in Winnebago it was a thriving town, that there were several industries, three hotels and a college. "However," he said, "it was not these things that brought me here. It was the people."

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Dr. Philip S. Hench, Rochester, presented a talk entitled, "X Marks the Spot," at a program in St. Paul on February 17, sponsored by the Rochester Area, Women's Association, Minnesota Historical Society.

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Principal speaker at a meeting for heart fund volunteers in Anoka on February 16 was **Dr. Morley Cohen,** Minneapolis, who spoke on "Recent Advances in Treatment of Heart Disease in Children."

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Dr. R. H. Puumala, of Cloquet, attended a postgraduate course in internal medicine at the University of Minnesota Continuation Center in mid-February.

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At the annual meeting of the Fosston Hospital Association in Fosston on February 8, **Dr. George Sather** gave a talk on different types of hospital and medical insurance policies.

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It was reported early in February that the Westbrook Commercial Club was hoping to locate and persuade another physician to open a practice in or near Westbrook. The community now has one physician.

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Dr. George S. Allen, Mineral Springs Sanatorium, Cannon Falls, attended the Veterans Administration Army-Navy Conference in Atlanta, Georgia, early in February. Subject of the conference was the chemotherapy of tuberculosis.

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Dr. and Mrs. Edward D. Detjen, Bigfork, spent the first week of February in Texas, visiting relatives and absorbing sun.

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Principal speaker at the annual meeting of the Citizens Mental Health Association at Minneapolis on February 18 was **Dr. Adelaide M. Johnson,** Rochester, who discussed problems of bringing up children in modern society.

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Dr. Walter A. Fansler, Minneapolis, took part in a symposium presented at Detroit on February 9 before members of the Michigan Academy of General Practice and the Wayne County Medical Society. Subject of the symposium was problems of the general practitioner.

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Dr. Tauno Ketola, staff member of the Cokato Hospital since September, 1954, was called into service with the Air Force at Montgomery, Alabama, at the end of January.

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Dr. William S. Chalgren, Mankato, spoke at a meeting of the Windom Kiwanis Club on February 8 on the subject, "A Psychiatrist's Approach Towards Juvenile Delinquency."

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Dr. and Mrs. A. B. Rosenfield, Minneapolis, and **Dr. and Mrs. C. L. Sheedy,** Austin, left on February 11 for a six weeks' vacation on various Caribbean Islands. At San Juan, Puerto Rico, both physicians presented papers before medical groups.

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On February 15, **Dr. A. J. Lenarz** left his practice at Browerville for a residency in surgery at St. Joseph's Hospital, St. Paul. He expects to return to Browerville on July 1.

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Dr. Harris P. Hinderaker, Bird Island, was the principal speaker at a meeting of the Bird Island Parent-Teachers Association on February 7. He spoke in connection with the local heart fund drive.

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A radio talk was given by **Dr. F. H. Koenecke,** Lakefield, over station KWOA, Worthington, on February 6. His talk was on "Premature Babies" and was part of the program entitled, "Your Health Hour," sponsored by the Southwestern Minnesota Medical Society and Auxiliary.

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After two years in the Air Force **Dr. R. J. Lindeman** returned to his practice at Paynesville in mid-January. While in service he was stationed at Colorado Springs, Colorado.

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Dr. M. J. Schirber, Grand Rapids, was the principal speaker at a meeting of the Itasca County Historical Society in Grand Rapids on February 7. He discussed medical instruments and methods used by pioneer physicians in Itasca County.

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Dr. L. G. Flanagan, Austin, attended a continuation course in ophthalmology at the University of Minnesota during the first week of February.

Dr. Nicholas H. Zeller, staff member of the Worthington Municipal Hospital since April, 1954, left in mid-January for a two-year tour of duty in the Army.

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Dr. Dean S. Fleming, director of disease prevention and control for the Minnesota Department of Health, has announced that more than 100,000 children in Minnesota may be vaccinated for poliomyelitis this spring. The decision about the vaccination program depends upon the results of a report on the Salk vaccine to be issued at the University of Michigan about April 1. If the report is favorable, Minnesota would accept a supply of the vaccine from the National Foundation for Infantile Paralysis and make it available to physicians throughout the state.

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Dr. Frederic Kottke, head of the department of physical medicine and rehabilitation at the University of Minnesota, was the principal speaker at a meeting of the Minneapolis District Multiple Sclerosis Society on January 18. Dr. Kottke discussed the university's rehabilitation center.

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Dr. C. J. Swendseen, Graceville, arrived in Starbuck in mid-January to take charge of the practice of **Dr. A. F. Giesen**, who was hospitalized at Northwestern Hospital, Minneapolis, due to injuries suffered in a fall.

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The Swedish consul general in Minneapolis announced in mid-January that **Dr. Henry P. Linner** had been decorated by King Gustav of Sweden. Dr. Linner was made a knight of the Order of North Star. He was one of four Twin Cities men honored for achievements in their fields and for their contributions to Swedish-American relations.

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Dr. I. L. Oliver, Graceville, was the guest speaker at a meeting of the Graceville Nurses Association on February 10. He spoke on new methods in heart and lung surgery and new types of anesthesia and sutures.

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The former Mayo Institute of Experimental Medicine, located near Rochester, has been renamed the **Institute Hills Farm.** The change was made because most of the research laboratories of the institute have been moved to the Medical Sciences Building in Rochester. The farm facilities now are used primarily for breeding and holding animals for research.

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The main speaker at a meeting of the Exchange Club in Cloquet on January 24 was **Dr. Kenneth W. Douglas**, superintendent of Sandstone State Hospital. He discussed the problems of alcoholism and narcotic addiction.

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Dr. and Mrs. H. L. Smith left Rochester on January 31 for a trip to Europe. They plan to return in May. Dr. Smith is a retired member of the Mayo Clinic staff.

Dr. Shohei Shirai of the Mesaba Clinic, Coleraine, spoke on the subject of personal health at a meeting of the Women's Club at Bovey late in January.

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Among the speakers at a conference of the Twin City Preschool Education Association at the University of Minnesota on January 22 was **Dr. Edwin Burklund**, St. Paul pediatrician, who spoke on the value of nursery schools.

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Dr. Lewis Hanson, Frost, was elected chairman of the Faribault County Public Health Nursing Advisory Board at the annual meeting of the board in Blue Earth in mid-January.

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Dr. John E. Hildebrand, Bemidji, suffered a severe fracture of one leg when he fell while crossing an icy street in Bemidji in mid-January. His practice was conducted during his convalescence by his associate, **Dr. J. K. Hartjen.**

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Main speaker at the January meeting of the Mankato Association for Retarded Children was **Dr. John Olive**, pediatrician at the Mankato Clinic. His talk was primarily on the Rh factor and kernicterus.

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Dr. Mario Fischer and **Dr. Edward Zupanc**, both of Duluth, were among the new directors appointed to the board of the Duluth Mental Hygiene Clinic at a meeting late in January.

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Common illnesses and practical ways of dealing with them were discussed by **Dr. Viktor O. Wilson**, Rochester-Olmsted County health director, at a public meeting in Rochester late in January. The talk was one of a series on family health being given in the community. Other lecturers in the series included **Dr. R. M. Wilder** on nutrition and **Dr. George Williams** and **Dr. R. L. Faucett** on the development of mental and emotional health.

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Dr. James J. Coll, Duluth, was guest speaker at a meeting of the Arrowhead Society of Medical Technologists at Duluth on January 28.

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Three Twin Cities physicians took part in a discussion of "Surgery of the Heart" on a television program on WCCO-TV on January 30. They were **Dr. Richard Varco, Dr. J. Arthur Myers, Dr. John Briggs** with Mr. Thomas Mulrooney.

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Dr. H. C. Otto, Frazee, who was operated upon at the University of Minnesota Hospitals in November, returned to his home in Frazee late in January. He was not expected to resume his practice for some time.

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"Health and the Child" was the title of an address by **Dr. John K. Butler**, Cloquet, at a meeting of the Parent-Teacher Association in Carlton on January 18.

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The Truman Public School Board has appointed

Dr. M. J. Lester, Truman, to fill the unexpired term of a late member of the board. The term runs until July 1.

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Dr. Russell M. Wilder, emeritus member of the Mayo Clinic staff, Rochester, was honored at a meeting of dietitians and nurses in Rochester late in January. Dr. Wilder recently won the American Medical Association's Goldberger award in clinical nutrition. He is best known for his studies of diabetes mellitus, carbohydrate metabolism and a program for vitamin-enriched bread and flour.

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Dr. S. T. Normann, Waseca, has been named coroner of Waseca County, replacing **Dr. Orvie J. Swenson,** who held the office for twenty-six years.

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Among the speakers at a meeting of the Minnesota Tuberculosis Nursing Council in St. Paul on January 14 was **Dr. F. F. Callahan,** St. Paul, chief of the division of medical services of the Minnesota Department of Public Welfare. He discussed proposed tuberculosis legislation in the state.

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At a meeting of the Dennison village council in January, **Dr. A. M. Nielsen,** Northfield, was named official physician for the village.

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Dr. Ivan D. Baronofsky, associate professor of surgery at the University of Minnesota Hospitals, has joined the Navy for a two-year tour of duty. He reported for service in February with the rank of lieutenant-commander.

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Dr. Royden H. Belcher, Wheaton, was the guest speaker at a meeting of the Graceville Nursing Association at Graceville in January. He spoke on new treatment methods in various types of epilepsy.

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Little-known medical services in the community were described and explained by **Dr. G. M. B. Hawley** at a meeting of the Red Wing Kiwanis Club in January. Among the subjects he discussed were the grievance committee of the Goodhue County Medical Society, surgical conferences, pathology examinations, and hospital services.

* * *

The Duluth Chapter, Minnesota Academy of General Practice, held a special refresher course on "Fluid and Electrolyte Balance" at St. Luke's Hospital, Duluth, on February 18. Four Minneapolis physicians presented talks during the afternoon and evening sessions. **Dr. John LaBree** and **Dr. Richard J. Frey** discussed "Electrolyte Balances in Cardiac and Renal Disease," and **Dr. W. P. Eder** and **Dr. Richard Raile** talked on electrolyte balance in regard to the surgical and pediatric fields.

* * *

Dr. H. F. Helmholz, Rochester, emeritus member of the Mayo Clinic staff and president of the Olmsted County Community Council, was the principal speaker at the annual dinner meeting of the Rochester Community Chest on January 19.

One week after **Dr. John W. Ekblad,** Duluth, retired as St. Louis County coroner on January 1, he fell down an outside stairs while calling on a patient and sustained a hip fracture. During his sixteen years as coroner, which often required strenuous activity, he was never injured. Dr. Ekblad is eighty years of age.

* * *

Dr. Sidney Esensten, Minneapolis, has received a commendation medal for meritorious service while a prisoner of war in Korea from November, 1950, to September, 1953. The commendation praised Dr. Esensten's efforts to render effective aid to sick and wounded comrades in spite of the difficulties imposed by his North Korean captors.

* * *

Dr. George R. Smith has returned to Hutchinson after two years of active duty in the Army. He has resumed his medical practice with **Dr. Elmer W. Lippmann** and **Dr. Dan W. Huebert.**

* * *

Dr. T. S. Eberley, Benson, sustained a severe leg fracture when his car skidded on an icy highway near Benson about the first of the year and overturned, hurling him from the car. He was taken to Miller Hospital in St. Paul for treatment.

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Dr. William B. Halme, who closed his practice in Wadena nearly two years ago to enter the armed forces, has leased a recently vacated building in Wadena. He expects to be discharged from service before June 1 and plans to resume his practice at Wadena immediately.

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Early in January, **Dr. F. W. Calhoun,** Albert Lea, was honored on his seventy-eighth birthday at a surprise party given by friends. Dr. Calhoun is now in his forty-eighth year of practice, all in the Albert Lea area.

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It was announced in January that **Dr. Herman Miller,** Grand Meadow's only physician, had been called into service with the Air Force.

* * *

Dr. John F. Briggs, St. Paul, has been elected to the Horseshoe Club of London, England, an organization whose object is to foster friendship between American, Canadian and English men and women interested in the cure and prevention of disease.

Dr. Briggs has also been asked to act as a collaborator for *Medico-Surgical Annals,* a new magazine which will stress diseases of the chest and thoracic surgery. The magazine will be published in Spain.

* * *

Dr. Gordon R. Kamman, St. Paul, spoke on "Psychological Evaluations in Medical Practice" at a meeting of the Sixth District Medical Society at Bismarck, North Dakota, on February 22.

NEW LOCATIONS

Dr. Carl Anderson, formerly of Miles City, Montana, has been appointed resident physician at the Minnesota State Penitentiary at Stillwater.

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Dr. Carl Heinzerling, formerly of Watertown, has opened offices for the practice of medicine in Chaska.

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Dr. A. L. Ourada, Ceylon, has opened an office at Fairmont. He now maintains office hours in Fairmont in the morning and in Ceylon in the afternoon. He expects to move to Fairmont in June.

* * *

Dr. Edward Menefee, who recently completed a residency in pathology at Rochester, is now associated with the Naeve Hospital, Albert Lea, as a pathologist.

* * *

Dr. John Meyers, formerly of Chicago, has become associated in practice with **Dr. L. J. Monson** in Canby.

MINNESOTA BLUE SHIELD-BLUE CROSS

Blue Cross News

During the month of December, 1954, 18,870 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of December 31, 1954 to 1,000,051 participant subscribers.

The amount of hospital care benefits received by Minnesota Blue Cross subscribers during 1954 amounted to $18,272,129.50, an increase of $2,822,556.55 over the amount provided in 1953.

During the past year, 165,989 Blue Cross subscribers were hospitalized for a total of 1,003,552.3 days of care. Average length of stay in the hospital for a Blue Cross patient was six days.

The four major causes for hospitalization of Minnesota Blue Cross subscribers during 1954 were: pregnancies, injuries and accidental poisonings, respiratory ills and digestive illness. There were 28,579 pregnancy cases of which 5,612 were for pyelitis of pregnancy, toxemias, ectopic, et cetera, and 22,967 were deliveries amounting to over 2½ million dolllars in benefits. There were 25,306 injuries and accidental poisonings amounting to over one million dollars in savings to subscribers. Over 1½ million dollars in benefits was provided for 22,703 subscribers who had respiratory illnesses. 21,996 subscribers had digestive ailments of which 11,600 were surgical cases. A total of approximately $3,200,000 was paid in benefits for digestive diseases alone.

Since 1933 when Blue Cross was first started here in Minnesota, 1,729,994 subscribers have spent 10,479,006.6 days in hospitals and have received benefits amounting to $110,949,752.

Blue Shield News

In the year 1954, payments to doctors for services rendered to Blue Shield subscribers totaled $4,970,000. This amount marks a new high in the annual amount of money paid to doctors. For the year 1954, payments have been over $700,000 more than was paid in 1953. Further analysis shows that the payments made in 1954 are 16 per cent greater than the amounts paid in the preceding year, 1953. The dollar increase in 1954 was due principally to the increased benefits which became effective on January 1, 1954, but also to the increase in the number of services rendered by doctors of medicine.

Another interesting factor in the growth of Blue Shield is revealed in the compilation of data which discloses that during 1954, 169,445 services were rendered by doctors for which Blue Shield provided allowances. This can be compared with a total 148,561 compensable services rendered during the year 1953.

Internal changes have also been made in the Blue Shield organization in order to provide more time to disseminate information to members of the medical profession regarding the Blue Shield contract and procedures. These changes have been of some help in the doctors' offices and have led to greater efficiency in Blue Shield. In this particular field, many of the problems which have formerly existed were eliminated through professional relations activities in 1954. This program is continuing and is being expanded as rapidly as possible.

Blue Shield enrollment as of December 31, 1954, totaled 670,757 participant subscribers. Enrollment as of December 31, 1953, was 609,474 participant subscribers. This represents slightly more than an 11 per cent increase in enrollment throughout the year 1954.

ATHLETIC ACCIDENT BENEFIT PLAN

(Continued from Page 183)

Control approved an increase to $3 per year for the boy who participates in football alone or football and other sports, and $1.50 per year for the athlete not playing football. It is hoped and intended that this will take care of the increased payments.

Your medical service committee and H. R. Peterson realize that there are a number of problems still to be solved and that there always will be administrative and public relations items to be considered. I assure you that utmost co-operation can be expected from the league and its medical advisor, and improvements will continue to be made. Comments and discussions from the physicians of Minnesota are more than welcome at any time and at all times.

Appreciation is expressed at this time to Dr. J. A. Bargen, chairman of the Medical Service Committee of the Minnesota State Medical Association, and his committee for their energetic help and advice in the 1954-1955 revision of this program.

Original Contributions

Polypoid Rhabdomyosarcoma of the Bladder: Sarcoma Botryoides

BAXTER A. SMITH, JR., M.D.
Minneapolis, Minnesota

AT the risk of seeming heretical, I would suggest that the clinical term "sarcoma botryoides" replace the more scientific pathologic term "rhabdomyosarcoma." If the latter designation is employed, the adjective "polypoid" should precede it, since sarcoma botryoides of the bladder appears to be a clinical entity. This stand is taken in the expectation that the clinical appearance is typical enough to promote early diagnosis and proper treatment. However, I am in complete accord with McFarland,[9] who showed it to be one of the manifestations of dysontogenetic tumors of the urogenital region; he found 119 different names used for this tumor in 516 references. Rhabdomyosarcoma of the prostate is no doubt the same lesion with a different appearance because inadequate room for expansion prevents the pronounced edema and the polypoid appearance.

Botryoid sarcoma is a polypoid, usually diffuse, edematous-appearing, grapelike tumor, involving the vesical submucosa. It arises in the region of the trigone or internal meatus and tends to produce obstruction early. It spreads through the submucosa to the adjacent bladder, posterior urethra and ureters. It is not feasible grossly to determine the margin of the tumor involvement. Infiltration in depth occurs considerably later. Local extension occurs and distant metastasis (regional lymph nodes, liver and lungs) is fundamentally a late manifestation. Once the lesion escapes the confining viscus, because of surgery or infiltration, rapid, and massive extension or local recurrence is the rule. Its "seeding" tendency seems pronounced.

Based on the behavior just described, the potential prognosis is good. In rhabdomyosarcoma of the adult bladder, the lesions seem to be single and pedunculated and may arise at a distance from the trigone; by description, they are not botryoid but no doubt have the same origin. Sarcoma botryoides occurs in the vaginas of little girls, resulting quickly in protruding polypoid masses. It occurs in the cervix in the middle years and the uterus in later years. The lesion may involve the soft palate also.

The microscopic picture shows edematous loose myxomatous stroma, in some areas fairly dense. The typical cell is spindle-shaped with acidophilic cytoplasm showing longitudinal fibrillae and cross striations. These latter may require phosphotungstic acid or iron-hematoxylin stain for identification; if searched for they usually are present and diagnostic. Mostofi and Morse[10] describe large spherical cells with a small amount of acidophilic cytoplasm and multinucleated giant cells as noted by several pathologists. As McFarland pointed out, the cells may be of mixed types and in varying stages of differentiation.

The bladder tumor is presumed to be of Wolffian-duct origin in the region of the ureteral orifices and trigone and is dysotogenetic, though the fibers of the external urethral sphincter or the prostatic capsule are postulated as the origin by some. Swinney[12] described a small pedunculated narrow growth protruding from the posterior urethra into the bladder at cystotomy. The recurrence was typical, diffuse botryoid sarcoma, and the initial description is likely that of a very early lesion. Cervical and vaginal origin could be from the duct of Gartner, a Wolffian-duct structure, or it may be from the Müllerian duct; most tumors in this location originate in the proximal vagina or the anterior wall.

The incidence of this tumor is much greater than reported cases would indicate, although the

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Presented before the Minnesota Academy of Medicine, February 10, 1954.

lesion is uncommon. In addition to the two cases here reported, I have knowledge of another seen in Minneapolis. Grier and Phillips,[4] in 1953, stated that twenty-two cases had been reported but did not include at least twenty-eight reported since their article was submitted for publication. Many tumors have been reported as myxosarcoma or myxoma,[2,5,9,11] but the clinical findings fit sarcoma botryoides and the striated muscle cells have not been sought or seen. McFarland collected seventy-seven cases of dysontogenetic bladder tumors; in sixty-one the patients were less than twenty-two years of age and almost all of the lesions were very soft. Higgins[6] reported a series of four cases.

Though Youngblood and associates[13] stated that the sex incidence is even, it would seem that males are afflicted in a frequency of 3:1. The age varies from that of the newborn, as in Hunt's[8] case, in which complete retention occurred at the age of five days, to twelve years, but most patients are less than four years of age. Adults have had rhabdomyosarcoma of the bladder, but apparently these were not sarcoma botryoides.

The prognosis has been given as uniformly bad. Only two children with such tumors have been reported as well two years after diagnosis. One of these, a boy, was well seven years after cystectomy, according to Flocks[3]; the other, described by Higgins, was a girl who was well two years and nine months after cystectomy. Higgins and associates[7] describe a "myxoma" which prolapsed through the urethra of a girl at birth; a ligature was applied. Cystoscopy at four weeks showed a scar at the region of the left ureteral orifice. Some "vesicles and thickening" were fulgurated upon two occasions. Four years later, the patient was well, and cystoscopy gave negative findings. Subsequently, the tissue was re-examined and found to be typical of rhabdomyosarcoma. Most patients are dead or dying of recurrence within a year, though Swinney's patient did not have recurrence until twenty-five months after definitive surgery. However, many authors have pointed out that the potential cure should be great, and it is probable that if the disease is understood, successful therapy will not be such an exception. It is imperative that those seeing the relatively few cases be familiar with the disease.

Symptoms are those of urinary obstruction, infection and hematuria. Obstruction occurs early. As one of the "grapes" tends to prolapse, giving obstruction, hematuria usually occurs at some time in the disease. In female patients, the masses may prolapse, and occasionally some "grapes" may be passed. Spasmodic pain from the obstruction to urination and prolapse of an edematous mass may be intense; pain may be due to secondary infection. The patient may have intermittent or continuous evacuation of urine or may have complete retention.

A mass is almost always present and is usually the distended bladder. After catheterization, the mass usually persists on bimanual palpation. It is moderately soft and somewhat movable, depending upon the state of the disease. The kidneys may be palpable due to ureteral obstruction. As already mentioned, there may be prolapse of a "polyp" through the urethra in the female patient, and, if the disease is extravesical, rapidly enlarging, edematous masses may protrude from the vulva or vagina. In a case described by Canard and Rivarola,[1] the urethra in a two-year-old girl had been so dilated by a prolapsing "polyp" that digital examination and biopsy of the bladder were feasible. The urine may be normal but usually shows red blood cells and pus. Fever may be present, and renal function is likely to be impaired in variable degree. The patient may be cachectic and show evidence of obstruction to venous and lymphatic return from the lower extremities.

X-ray examination should be performed by excretory urography, if the renal function allows. This will show the status of the upper urinary tract and, if function is adequate, give a satisfactory cystogram. If not, retrograde cystography will show a picture typical, but not well-known, in the roentgenologic or pediatric literature, as determined by my scanty perusal thereof. This appearance is of multiple filling defects, usually smooth and involving the base particularly and the prostatic urethra frequently. The lesions are sometimes described as "lacunar." As in Case 1, others have confused the filling defect picture with "rectal gas."

Cystoscopy is usually feasible in contrast to sarcoma of the prostate. Interpretation may be difficult because of the presence of bleeding and infection and the use of small instruments, but the picture should be diagnostic. Multiple, pearly white, semitranslucent polypoid structures are seen. The region of the trigone and internal meatus almost always is involved. The appearance has been likened to hydatidiform mole, and Canard and Rivarola compared the bladder to the inside of a hydatid (echinococcus) cyst filled with daughter cysts. Transurethral biopsy should be done to verify the clinical diagnosis so that one will have pathologic backing to bolster one's courage for the necessary undertaking in a young child. The bladder should not be opened. The grapelike masses may be cut or broken off without fear of untoward bleeding and are diagnostic pathologically.

The differential diagnosis should include polyps of the verumontanum. If there is a question of the lesion's nature, resection rather than fulguration only should be done so that material for biopsy may be obtained and an early lesion discovered. Edema from adjacent processes such as an appendiceal abscess may be confusing. An ureterocele, which may prolapse through the female urethral meatus, or which may be inflamed, edematous and located intravesically, must not be mistaken for botryoid sarcoma.

Report of Cases

Case 1.—A fifteen-month-old white girl was hospitalized March 12, 1948. "Vaginal prolapse" was first noted in December, 1947; in January, 1948, "prolapsing bladder mucous membrane" was partially excised and the remaining portion reduced. Two days prior to admission, there was prolapse of the rectum and what was believed to be uterus; again "bladder mucosa" protruded. No complaints referable to voiding were noted, but the rectal prolapse was probably due to straining to urinate.

Examination revealed a large firm purplish mass protruding from the vulva. The next day an observer stated the mass had increased no less than four times in size. Blood urea nitrogen was 24 mg. per 100 cc. Catheterized urine was normal except for 1+ pus cells. Excretory urography showed bilateral hydronephrosis and hydroureter and deformity of the bladder base believed due to rectal gas.

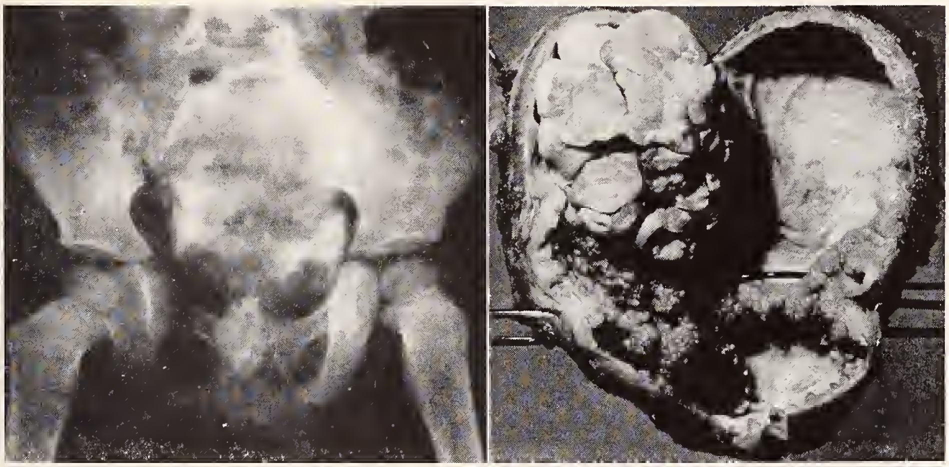

Fig. 1. *(left)* Case 2. Excretory cystogram. Note smooth filling defects in the bladder, particularly at the region of the internal meatus, and the dilated posterior urethra with filling defects.

Fig. 2. *(right)* Case 2. Frontal section of the bladder, fixed in distention. The ureteral catheter identifies the position of the ureteral orifices. Note tumor nodules in the prostatic urethra.

At cystoscopy, the urethra was found posterior to the vulval mass; the meatus was not dilated and no mass protruded from it. There was marked edema of the anterior bladder wall at the internal meatus. Trabeculation was present in the fundus. The right ureteral orifice was identified, but not the left. The vulval mass seemed attached by a broad stalk of tissue going anterior to the urethra. There was thickening noted on rectal examination. The vesical changes were interpreted as secondary to extravesical tumor, and biopsy of the mass showed malignant mixed tumor, predominantly myxoma. The retrograde cystogram, in retrospect, was quite typical of botryoid involvement near the internal meatus.

On March 19, the large lesion anterior to the urethra was dissected upward, displacing the bladder posteriorly. The tumor fanned out in a sheet across the anterior bladder wall; biopsy at the line of excision resulted in a diagnosis of myxosarcoma. The excised mass measured 3.5 x 3 x 2 cm. and the bladder was then felt to be 4 x 3 x 2 cm. with thickening out of proportion to hypertrophy. Botryoid sarcoma was then believed to be the diagnosis; since involvement extended beyond the bladder, total excision was not deemed feasible. It is probable that the lesion spread to the retropubic area from the vulva rather than by penetration of the urinary tract.

On April 6, 1948, transverse colostomy was done by Dr. David State, and the bladder was estimated at 5 x 3 x 2 cm. One month later, he removed the bladder, uterus, vagina and vulva transplanting the ureters to the sigmoid. Two peritoneal nodules were present over the bladder, and several nodes were involved with tumor.

The specimen showed a large fungating tumor composed of grayish-yellow, somewhat rubbery tissue. Penetration occurred through half of the bladder wall. There was a tumor nodule measuring 2x1x1 cm. in the vagina. Microscopic examination showed spindle cells of fairly uniform size in a stroma that in some areas was fibrous and in some areas myxomatous.

The girl was discharged on May 18, 1948, and was readmitted in August, 1948, because of prolapse of the colonic stoma. Recurrence was noted on rectal palpation; the child died in February, 1949.

Case 2.—A two-year-old white boy was first seen on January 16, 1953. Two months previously, he had mild hematuria, and an excretory urogram was presumably negative. From that time on, he had variable but progressive difficulty in voiding, and at the time seen he was dribbling almost continually. At times, pain seemed rather intense and he awakened from sleep in pain. He would rub his penis and cry. Fecal incontinence occurred with straining to void.

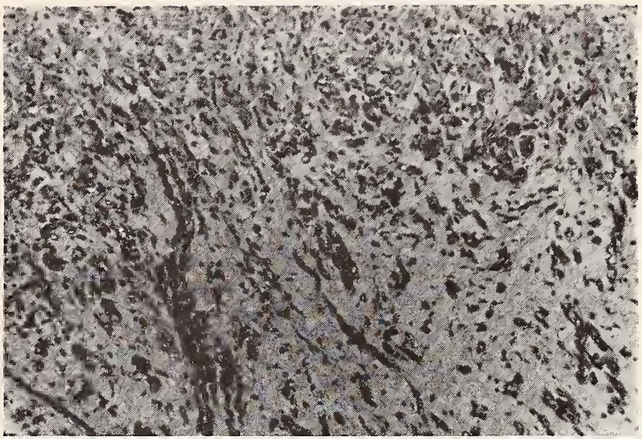

Fig. 3. *(above)* Case 2. Low-power view of the tissue, myxomatous below and more cellular above; acidophilic bands are prominent.

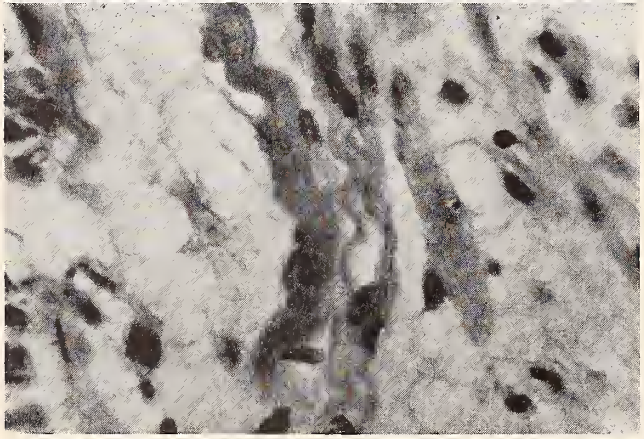

Fig. 4. *(below)* Case 2. High-power view showing myofibrillae and cross striations.

Examination revealed a palpable tender bladder. The urine contained 2+ albumin, a few red blood cells and packed fields of leukocytes. Excretory urography demonstrated slight dilatation of the lower ureters and a typical cystogram (Fig. 1).

At cystoscopy, landmarks were difficult to recognize. The typical white grape-like masses were present, mainly in the anterior wall and surrounding the internal meatus. There was trabeculation and cellule formation in the fundus and base. Several small masses were excised for biopsy. A moderately soft mass extending well toward the umbilicus was present on bimanual palpation.

Microscopic examination showed a markedly edematous stroma and chronic inflammatory exudate in the submucosa. In this stroma were thick pink-staining bands with cross striations in some.

On January 23, 1953, prostatovesiculocystectomy and regional lymphadenectomy were performed. The urachus was severed at the umbilicus; the urethra was divided 2 cm. below the prostatic apex. The ureters were transplanted to the rectosigmoid in a modified Leadbetter manner, leaving a rather long loop of sigmoid proximal to the anastomosis so that terminal colostomy might be performed later. The child's bladder is more intraperitoneal than the adult's, facilitating cystectomy somewhat

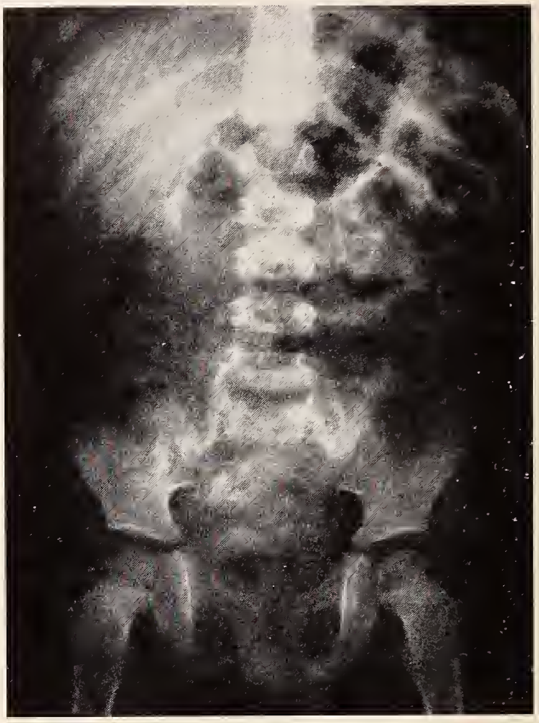

Fig. 5. Case 2. Excretory urogram 6 months after operation.

Pathologic examination showed diffuse involvement of the bladder and posterior urethra (Fig. 2) and one nodule in an intramural ureter. Sixteen identified nodes showed no evidence of malignancy. The microscopic picture was similar to that of the material removed for biopsy (Figs. 3 and 4). Invasion of muscle was not seen.

Convalescence was uneventful. An excretory urogram on the fourteenth postoperative day was normal, and the patient was dismissed the next day.

The boy was seen June 17, 1953. Blood chemical values and the urogram were excellent (Fig. 5), and the only suspicious finding was a small nodule at the site of one of the stay sutures. This was excised and a stitch abscess with a bit of silk was found. Bimanual examination and palpation of the urethral bulb gave negative findings. The grandparents reported that in January, 1954, the boy was well and prospering. Terminal colostomy is planned later.

Comment

In almost all instances of this disease, the urinary tract has been opened prior to definitive surgery, and the speed and magnitude of local recurrence are impressive. Renal function was destroyed or the disease had extended prior to seeking medical care at times. A high index of suspicion is essential to early diagnosis. This disease seems to be a clinical entity, and from the cystogram, cystoscopy and palpation under anesthesia, diagnosis should be accurate. Cystoscopic biopsy is feasible, but suprapublic biopsy is contraindicated.

Prompt cystectomy, including the urethra in female patients, the prostate, seminal vesicles and a liberal portion of adjacent urethra in male patients and a segment of adjacent ureter in both, should be performed. In female patients, the uterus and at least the upper half of the anterior vaginal wall probably should be excised. The method of urinary diversion is another subject. Irradiation is ineffectual and is thought by some to stimulate the tumor to more rapid growth; fulguration has the same reputation.

Summary and Conclusions

1. Two cases of sarcoma botryoides have been reported: One patient died, whereas the second patient was well eleven months following radical cystectomy.
2. Sarcoma botryoides or polypoid rhabdomyosarcoma of the bladder in children seems to be a clinical entity and the descriptive former term is suggested as perhaps more appropriate in stimulating earlier recognition of the disease.
3. Familiarity with the nature of the lesion should lead to early diagnosis.
4. Opening the bladder or cutting into the tumor usually is followed quickly by massive local recurrence.
5. Radical cystectomy should be undertaken forthwith, including a liberal proportion of the adjacent urethra and ureters.
6. The prognosis of the tumor should not be as bad as the statistics to date would indicate.
7. It is believed that sarcoma of the prostate is the same lesion with a different gross appearance.

Addendum

The second patient (Case 2) was well when last seen, June 30, 1954.

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RETREATMENT OF CERVICAL CANCER

Two Mayo Clinic radiologists have published a review of results in *retreatment* of cervical carcinoma.

Their conclusion: "The impression that prognosis is largely determined at the time of primary diagnosis and treatment is partially refuted."

The two doctors are Martin Van Herik and Robert E. Fricke, from the section of therapeutic radiology at the Mayo Clinic. Their review and report appears in the March issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

One hundred and ten patients with recurrent cancer of the cervix, treated by irradiation, during 1940 to 1947 were studied. Of the entire group: twenty-five patients (22.7 per cent) lived more than three years following treatment for the recurrence; eighteen patients (16.4 per cent) lived more than five years.

"This result is far from excellent, but it is sufficient to refute partially the impression that the prognosis is largely determined at the time of primary diagnosis and treatment," report Drs. Van Herik and Fricke.

"Furthermore, detailed study of the records of our patients who did not survive at least three years following treatment for recurrence reveals that in a great many cases a worthwhile palliation was achieved," they add.

The Southern Minnesota Medical Association

WILLIAM C. BERNSTEIN, M.D.
Saint Paul, Minnesota

A PRESIDENTIAL address which is to be delivered as an after-dinner speech must of necessity be conceived and written with the proverbial two strikes against it. I have listened to many presidential addresses at this and at other meetings, and I am well aware of the necessity for brevity and alacrity in order that the speaker remain in the good graces of his audience. I shall try to remain in your good graces.

This has been a most pleasant year for me. To have served as your president and to have had the privilege of working closely with the other officers and committee members has been an honor and a pleasure which I shall long cherish. I completed my internship at the Ancker Hospital in Saint Paul and then began the practice of medicine at New Richland in Waseca County in 1928. In 1929, I became a member of the Southern Minnesota Medical Association and read my first paper at the annual meeting which was held in Mankato. I am therefore, in 1954, celebrating my silver anniversary as a member of this organization by speaking to you as your president.

The Southern Minnesota Medical Association has played a most significant role in my professional career. It provided me with the opportunity through the years not only to listen to many outstanding medical speakers, but to read papers, report cases and participate actively in the affairs of this dynamic medical society. It has given me the inspiration, which is so vital in a young medic's life, to work towards higher goals. What membership in this organization has done for me, it can do for every young physician who practices in southern Minnesota. This society actively solicits the interest and participation of its younger members. The officers of this society are most happy to have papers submitted by all of its members. The program committee works zealously to build a program that will interest everyone and which will stimulate others to submit papers for future meetings. I urge our membership to be active members in the society. Our organization, since its inception, has been a vital force in Minnesota medicine, and from the quality of the program which was presented here today, there can be no doubt as to the future status of this organization.

It may be of interest to point out a few important historical facts concerning the Southern Minnesota Medical Association. Dr. Monte C. Piper of Rochester delivered an address on this subject at the meeting in 1935, and I have selected a few interesting details from his paper. We are meeting today in the city which witnessed the birth of this society on July 28, 1892. The letters inviting the doctors in this area to an organization meeting were sent out by a group of Rochester physicians. Thirty-five physicians attended, and the association was founded on that day. The committee on permanent organization consisted of Drs. W. T. Adams of Elgin, W. J. Mayo of Rochester and J. B. McGaughey of Winona. The dues were fifty cents a year. The first paper was presented by Dr. McGaughey on "Cerebrospinal Meningitis"; the second paper was by Dr. W. J. Mayo on "Less Common Forms of Surgical Tuberculosis."

Twelve years before the organization of the Southern Minnesota Medical Association, the Minnesota Valley Medical Association was organized at Le Sueur on December 1, 1880. This society adopted the by-laws of the Ramsey County Medical Society as a foundation for its government. Dr. Otis Ayer of Le Sueur was honored by being chosen the first president.

At a joint meeting of the Minnesota Valley Medical Association and the Southern Minnesota Medical Association held in Rochester in 1911 the two societies amalgamated, and the Southern

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Presidential address presented at the annual meeting of the Southern Minnesota Medical Association, Winona, Minnesota, September 13, 1954.

Minnesota Medical Association as its exists today came into being. The Minnesota Valley Medical Association which had existed for thirty-one years contributed 105 members, while the Southern Minnesota Medical Association which was nineteen years old contributed 115 members. Dr. L. A. Fritsche of New Ulm was elected president of the amalgamated societies.

As the Southern Minnesota Medical Association grew in scope and influence, rumors arose that this society was a threatening rival to some of the larger medical organizations. Talk of further expansion and competition with the Minnesota State Medical Association and the Tri-State Medical Association was put to an end by Dr. W. J. Mayo in 1919 when he stated, "Dr. Andrews has said that the association is equal to, if not greater than, the state association. I want to say to you, members of the Southern Minnesota Medical Association, that this should not be true, that every man who is here is potentially a member of the state medical association, and he should attend the meetings of the state medical association as the representative body of the state. After all, we are a local society, but we have great traditions."

I believe that time has proved the wisdom of Dr. Mayo's admonitions. This society is a local, nonpolitical organization which fills a definite need in the professional life of the physicians in southern Minnesota. We encourage the preparation and presentation of scientific papers; we encourage higher scholastic attainments in medical school by our annual awards to the most outstanding senior. We encourage scientific exhibits at the annual meeting of the Minnesota State Medical Association by awarding a medal for the best scientific exhibit, and we are an organized unit to enhance the quality of medical and nursing care in Minnesota.

Anyone who has listened to the wide variety of excellent papers presented at our meeting today must be impressed with the facts that we are now discussing many subjects almost undreamed of twenty years ago, such as, "Radio-isotopes in Diagnosis," "Cortisone Therapy," "Pulmonary Hypertension" and others. Also, of course, it is no longer possible for one person to be well informed on all phases of medicine. Dr. Aura E. Severinghaus, writing in a recent issue of *The Journal of the American Medical Association*, said, "The first half of this century will always be known as 'a scientific age.' Among the major accomplishments of science, history will record the conquest of disease. Progress in this area has been spectacular beyond all expectation. With it has come, however, an almost staggering expansion of our scientific knowledge. More than ever before it has become necessary for those who wish to keep professionally informed to increase their scientific information. More than ever before it has become obvious that no person should even attempt to encompass the entire body of facts. This has naturally increased the emphasis on science in the future physician's education and resulted as well in a high degree of specialization in today's medical practice. The end is not yet in sight. I am reminded of a friend who recently hailed a taxicab in front of the Bureau of Archives in Washington. As the cab drew up he was still repeating audibly the inscription over the front entrance, 'The Past is but a Prologue.' 'Do you know what that means?' he said to the driver, and, as is characteristic of Washington cab drivers, the driver quickly replied, 'Yes sir, that means you ain't seen nothin' yet.' "

I would like now to make some comments relative to the practice of medicine in Minnesota in general and in southern Minnesota in particular. It is well to take inventory once in a while to evaluate our own assets and our own liabilities or shortcomings. I am certain that no one would contradict me when I state that the standards of medical practice in this section of our country are of the very highest. I know of no area where medical practice, be it of the general practitioner in the small community or of the specialist in the larger city, is on a higher plane. I know of no place in this entire country where postgraduate courses, tailored to fit the needs of the physicians practicing in the state, are made so readily available. Is there a reasonable explanation for this situation? The answer, I am sure, lies in the presence in our midst of two of the world's greatest institutions of medical teaching and research, namely, the University of Minnesota Medical School and the Mayo Clinic with its associated Mayo Foundation. Those of us who practice so close to these institutions of world renown begin, after a while, to take them for granted and to disregard the enormity of the roles played by them in medical progress. Need I mention more to you than the contributions of Hensch and his co-workers at the Mayo Clinic in the development of cortisone, and the epoch-making advances in cardiac surgery recently announced at the University of Minnesota Hospitals, to remind you of the atmosphere in which all of us live and practice? We are sometimes blinded by our proximity and, to use a trite expression, we fail to see the forest for the trees. We need but to travel to other states or other countries to be impressed with the overwhelming stature of these two institutions. How often do we hear or participate in derogatory discussions concerning these institutions? Would anyone have to stretch his imagination to believe or to admit that medical practice has attained its high level here because of the influence of our great medical centers? Could one dare to imagine what would happen to the standards of medical practice in this area were we to lose one or the other —or both—of these institutions? We who practice here are the beneficiaries of one of the richest medical legacies in history. Let us not treat our legacy too lightly.

The practice of medicine in Minnesota is different in other ways, too, from the practice of medicine in some other locales. We hear too often of contending factionalism and of bedeviling provincialism in medical practice. Fortunately for us, we experience few of these evils in Minnesota. The spirit of co-operation at hospital staff and county society levels is everywhere apparent. I believe that few states can boast of better records in this regard.

I would be amiss in my responsibilities, however, if I did not point out one of our most flagrant shortcomings. It is a well-known fact that Minnesota ranks high in the incidence of malpractice suits. It is paradoxical that this should be true. Our state society has had a hardworking committee assigned to this problem for many years. The situation has shown little improvement thus far, and our medical malpractice insurance rates continue to rise. Everyone seems to be agreed that most suits are initiated or encouraged by misdirected remarks on the part of other physicians. This problem can and must be corrected if we are to maintain a position where we can use our initiative in treating patients without having the threat of malpractice forever dangling over us.

One of the great problems facing the medical profession here and elsewhere is the shortage of nurses. It is interesting and gratifying to learn that the situation in Minnesota is not as bad as it is in many other states and that our schools of nursing are turning out more nurses in all categories than in previous years. Although Minnesota ranks eighteenth among the other states population-wise, it ranks fifth in the number of nurses it turns out. Hospitals are learning that many duties can be delegated to nonprofessional personnel and that the nursing team consisting of a registered nurse, a practical nurse and several nursing aides can function well as a unit in caring for large numbers of patients. We are fortunate in having progressive and mature leaders to encourage and educate large numbers of young women for nursing careers. The outlook for the future is brighter than it has been for many years. This society has a very active committee on nursing which has made some valuable contributions to the nursing program.

In conclusion, may I once again urge the younger men to participate actively in the affairs of this society and may I impress upon you the privilege which is yours of practicing medicine in an area where the light of medical learning and of high standards of practice and ethics shines so brightly upon all the members of our profession.

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Puerperal Psychoses

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ACUTE psychotic symptoms develop as a complication after pregnancy about once in every 600 deliveries. The frequency varies slightly with different authors, due to different criteria for diagnosis. Prior to the advent of obstetrical asepsis and antisepsis and the development of antibiotics, mental illness occurred much more frequently, due to the high incidence of toxic delirium associated with puerperal sepsis. In the present era toxic reactions are rare, and the clinical picture in the psychoses after childbirth is that of a depression, schizophrenia or occasionally a manic reaction. As a result, the clinical course of these illnesses in modern times is prolonged but the mortality is low. In the puerperal toxic psychoses of an earlier day the patient recovered or died very promptly. Today the mortality results from an occasional suicide, or more rarely from catatonic or manic exhaustion.

There is a tendency to drop the term "postpartum psychosis" from current literature. Modern psychiatric textbooks devote little or no space to it, apparently preferring to classify these illnesses with the usual functional psychoses. This we feel is a mistake. The terms postoperative and involutional psychosis are commonly and properly used, and the diagnosis postpartum psychosis should be likewise preserved. We feel that there are differences between the postpartum and other psychoses which warrant their being classified as an entity. The illness carries a distinct threat to the newborn infant and, in addition, differs in its course, prognosis and frequency of recurrences, in comparison with other "functional" psychoses. These differences should be emphasized, and can be done so only if the postpartum psychoses are classified in a separate category.

The term postpartum psychosis should be reserved for those cases which meet all the following three criteria: (1) The onset of symptoms is during the first six weeks after the termination of a known pregnancy—the common obstetrical definition of the puerperium. (2) The patient has no past history of other psychotic reactions. A previously schizophrenic or manic-depressive patient who has a relapse after delivery cannot properly be classified in this group; however, recurrences of the disease after succeeding pregnancies can occur and are consistent with the diagnosis. (3) The patient must have an actual psychosis. This eliminates the more common, often transient, exhaustion syndromes and neurotic reactions which occur at this period.

We have reviewed our experience over the last several years with forty-three cases of this condition, in an attempt to find some indication which would make possible the detection during pregnancy of the woman who will develop a postpartum psychosis, as a step toward prevention of this complication. Our patients were compared with two groups of controls. The first consisted of psychotic married women of the same age distribution, whose mental illnesses developed unrelated to pregnancy and delivery. In comparing our patients with this group, no differences could be found in the age of marriage, age of childbirth, family history of psychoses, position of the patient in her sibling pattern, prepsychotic personality traits, educational background or frequency of environmental stresses. In each group there was a family history of mental illness in about 30 per cent of the patients. In both the postpartum group and this control group, about one-half of the patients had a prepsychotic personality considered to be normal and well adjusted by their families; the balance were described variously as "excessively nervous," withdrawn or overly meticulous and perfectionistic.

The second control group consisted of a series of married women in the same age range, who had no psychotic symptoms. These were for the most part patients with organic neurologic disease.

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In comparison with this group appreciable differences were seen in the family history of psychoses and in the personalities of the patients. Only four of these forty-three control patients had a positive family history of mental illness, and twenty-nine were considered to have stable and well-adjusted personalities.

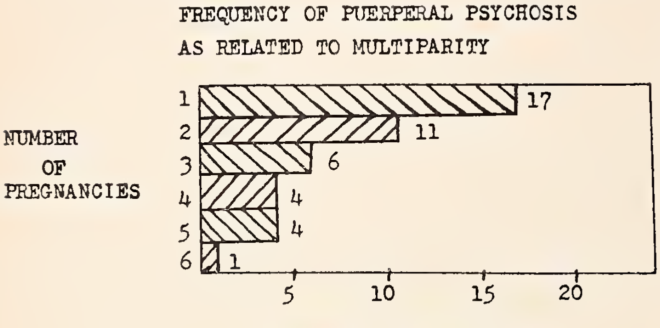

Fig. 1.

The patient who is destined to have a postpartum psychosis is more likely to have a history of mental illness in her family and a past history of an unstable personality than is a non-psychotic woman. But these factors occur with about equal frequency in women who develop psychoses not related to parturition. Thus one can predict a postpartum psychosis in a pregnant woman with about the same degree of accuracy as one can predict a functional psychosis before it occurs. This agrees with the statement of Strecker et al[1] that the puerperal period may precipitate a psychosis, especially if there is evidence in the patient's personality makeup of either manic-depressive or schizoid tendencies.

Puerperal psychoses occur rarely following abnormal pregnancies or premature deliveries. In thirty-nine cases the pregnancy and labor were entirely normal. One patient had a cesarean section and one a postpartum hemorrhage following which the patient's hemoglobin dropped to 40 per cent. Toxemia of pregnancy occurred in the two other complicated cases. Of these one occurred antepartum and was mild. The other patient had severe eclampsia with convulsions immediately postpartum, and the associated psychosis was toxic in type. In each of the 49 psychotic episodes which occurred in these 43 patients the pregnancy resulted in a living, healthy child born at full term. There was no instance in this group of a postpartum psychosis developing after an abortion, miscarriage, premature labor, stillbirth, or after the delivery of a deformed or abnormal baby. These forty-three women have had a total of 126 pregnancies. Of these, twelve have ended tragically with a dead or deformed infant without a single instance of puerperal psychosis. The remaining 114 pregnancies, which all resulted in apparently normal children, were followed in forty-nine instances by mental illness. While the series is too small to permit definite conclusions statistically, it does suggest that death or deformity of the child does not predispose to the development of a puerperal psychosis and may actually decrease the possibility of a mental illness after delivery.

The likelihood of a puerperal psychosis is greater in primiparas and early multiparas than it is in women who have had several pregnancies. Of the forty-three women, seventeen were primiparas, and thirty-four developed their first psychosis after one of the first three pregnancies. There was one patient whose mental illness did not develop until after the sixth pregnancy (Fig. 1). The various types of psychoses occur in about the same ratio as do the usual functional psychoses in this age group. The implication is that whatever role the pregnancy and delivery may play in precipitating the illness, the type of mental reaction which results is determined by the patient's own personality makeup and by other factors unrelated to the pregnancy. Our patients included eighteen with depression, one with a manic psychosis, fifteen with schizophrenic reactions, eight with disorders which were classified as schizo-affective, and one with toxic delirium. As a group they showed a pronounced tendency toward suicidal impulses and toward homicidal threats against the newborn infant. Eight of the patients were considered suicidal risks, and six expressed definite desires to harm their babies. These two factors place a great responsibility on the obstetrician in the early recognition of psychotic symptoms and the prompt protection of the mother from harming herself or her child.

Results of Treatment and Follow-up

On the whole the prognosis for recovery from puerperal psychoses is gratifying and better than the average for the usual functional psychoses in this age group. Five of the patients were not hospitalized, either because the family refused it or because the illness was mild enough to permit out-patient treatment. Of these five, four made steady improvement to recovery and one a social remission. A two- to five-year follow-up of four of these patients shows that three are well at the present time and the other is at home in partial remission. The remaining patients concerning whom we have treatment and follow-up data had long and stormy hospital courses, often with frequent relapses but with good ultimate prognoses.

We have been able to follow twenty-eight patients for from one to eight years after their illnesses. Three of these (all schizophrenic) are still in hospitals and must be considered permanently mentally ill. Seven patients are able to be at home and to carry on a partially normal life, but are still having mild symptoms. The remaining eighteen patients recovered and have maintained their recovery to the present time (Table I).

The question of recurrence of mental illness in patients after having once had a puerperal psychosis is invariably asked by the patient and her family. Of the twenty-eight patients we have followed, only ten have had more children, and one of these had a second postpartum psychosis. Two additional patients have had subsequent psychotic reactions unrelated to pregnancy and both recovered.

Reviewing the entire reproductive history of the group, we find thirty-seven women who had two or more pregnancies. Of these, six have had postpartum psychoses on two occasions. Thus there appears to be about one chance in six of the disease reappearing in a subsequent pregnancy. Compared with the average incidence of once in 600 pregnancies, the chances of a postpartum psychosis are about 100 times greater in a woman who has been psychotic following an earlier pregnancy than they are in a woman without such a history. Obviously great caution must be taken in advising a patient about further children after she has had such an illness.

TABLE I. FINAL RESULTS
1 to 8 Year Follow-up

Type of Psychosis	Recovered	Improved (Home)	Unimproved (in Hospital)
Depression	8	2	
Manic	1		
Schizophrenic	7	3	2
Schizo-affective	2	1	1
Toxic		1	
Total	18	7	3

Conclusions

Puerperal psychoses are most likely to occur in women with family histories of mental illnesses and past histories of unstable personalities. The disease occurs most commonly following the first three pregnancies, but may not appear until after the sixth pregnancy. An uneventful pregnancy, with normal labor and the birth of a healthy child, is no protection against the development of puerperal psychosis. The clinical picture is that of any of the usual functional mental illnesses. The disease carries a distinct threat of suicide and of homicidal impulses against the newborn infant. The prognosis for recovery is good, usually after a prolonged and stormy hospital course. The chance of a recurrence in subsequent pregnancies is about one in six.

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BENIGN STOMACH ULCERS

The actual incidence of benign ulcers of the greater curvature of the stomach may be much higher than the reported cases—despite statements that this benign type of ulcer is rare in comparison to malignant ulcers in similar location.

This is the suggestion of Dr. Frank S. Browne, radiologist from Wichita Falls, Texas, in the March issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

He reported a patient in whom most of the characteristics associated with carcinoma were present, yet microscopic examination revealed a benign peptic ulcer.

The ulcer itself had been discovered during an x-ray examination of the gastrointestinal tract.

"Verbal reports from numerous observers would indicate that the actual incidence of benign greater curvature ulcer is much higher than reported cases," concludes Dr. Browne.

Psychiatric Illness Following Gastric Resection

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THE role of emotional factors in the etiology of peptic ulcer has been the subject of much theorizing and of some objective research. Investigators from the time of Beaumont have made observations on changes in gastric function under a variety of emotional stresses. Despite these observations the precise mechanism of ulcer production following stress has not been formulated. However, it seems to us that the studies of such investigators as Alexander,[1] Szasz,[4,5] and Mahl[3] suggest a basis for proper dynamic treatment of peptic ulcer patients and point the way toward research which can throw additional light on the problem.

TABLE I.

Patient	Age	Sex	Diagnosis	Indication for Surgery	Adjustment Prior to Resection
1	47	F	Psychophysiological gastro-intestinal reaction	Peptic ulcer	Inadequate
2	49	F	Drug addiction. Psychoneurotic reaction, mixed	Peptic ulcer	Poor
3	56	M	Depressive reaction	Peptic ulcer	Adequate but markedly hypochondriacal
4	37	F	Depressive reaction	Peptic ulcer	Adequate
5	54	M	Depressive reaction	Peptic ulcer	Nervous breakdown, age 40. Good recovery
6	38	M	Depressive reaction	Peptic ulcer	Adequate
7	57	M	Depressive reaction (psychotic)	Peptic ulcer	Adequate
8	41	M	Depressive reaction	Peptic ulcer	Poor
9	50	F	Paranoid state	Esophagitis	Adequate
10	59	F	Catatonic reaction	Esophagitis	Adequate

We are concerned here with ten patients who were seen on the wards of the University of Minnesota Hospitals for psychiatric treatment or consultation and who gave a history of previous gastrectomy. These are consecutive unselected cases collected during the past two years. So far as we can determine from a review of the literature, there is only one report which considers the results of this surgical procedure in terms of total social and emotional adjustment. That is, Browning and Houseworth's[2] study of sixty duodenal ulcer patients, thirty of whom were gastrectomized and thirty of whom were treated medically. The gastrectomized group showed a reduction of ulcer symptoms but a marked increase of psychosomatic and psychoneurotic symptoms. In the medically treated group the reduction of ulcer symptoms was less dramatic but there was no increase in psychosomatic or psychoneurotic symptomatology. In addition long term follow-up demonstrated an increase in psychogenic disturbances and organic difficulties in the post-gastrectomy group as compared to the non-gastrectomized group. In light of total ability to get along in the world, the thirty gastrectomized people were less well off than the thirty who were managed medically.

The ten patients we are reporting had gastrectomies by several surgeons in various hospitals from three days to nine years before we saw them (Table I). Of the total, eight patients had psychiatric difficulties which from history seemed clearly related to the gastric resection. Of the eight, six were depressive reactions, one was a catatonic reaction and one a paranoid reaction. The six depressive reactions occurred in patients who had gastrectomy for peptic ulcer. The catatonic reaction occurred in a woman who had a gastric resection for severe esophagitis and the paranoid reaction in a woman who had a gastric resection for esophageal obstruction. Two of the ten patients had emotional illness which was apparently unrelated to the gastrectomy. One of these (Case 2) had a history of longstanding, severe neurotic difficulty including dependence on drugs and the other (Case 1) was a Latvian refugee who had a history of intense,

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early deprivation, plus repeated exposures to severe emotional trauma during the occupation of her homeland by hostile forces.

The Minnesota Multiphasic Personality Inventory was administered to nine of the patients. All profiles were abnormal. Six profiles showed patterns commonly found in people with depressive reactions, *i.e.,* primary elevations on the left side of the profile with the apex at D and a secondary elevation on the right side with the apex at Pt. Figure 1 shows the average profile of the depressed group. These profiles were on the patients who had gastric resections because of peptic ulcer and are no different from profiles of patients with depressive reactions unassociated with gastrectomy. Two of the profiles have in common a V configuration on the first three scales which is characteristic of neurosis with hysterical features (Fig. 2). These profiles occurred in the two cases (1 and 2) who had peptic ulcer, long histories of severe neurotic difficulties and in whom the psychiatric illness did not seem related to the gastrectomy. The profile of the patient who had a paranoid reaction was one which is usually considered psychotic. No profile was obtained on the patient who had the catatonic reaction.

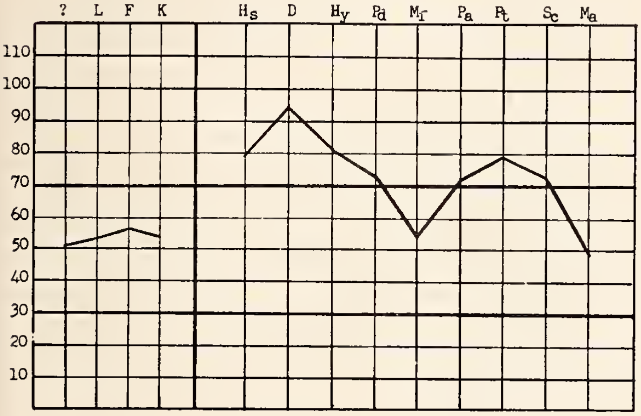

Fig. 1. Average MMPI profiles of the six cases with depressive reactions related to gastrectomy. (Cases 3-8, inclusive)

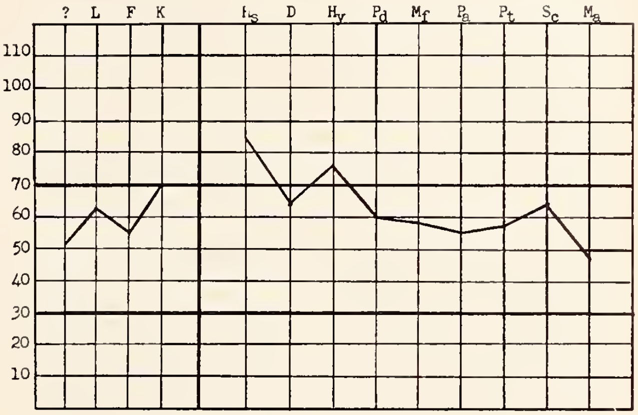

Fig. 2. Average MMPI profiles of the two cases in whom psychiatric illness was not related to gastrectomy.

Summary

We have reported ten patients who had psychiatric illness and gave a history of previous gastric resection. In eight of the cases, psychiatric illness seemed clearly related to the resection. Six of these were depressive reactions and occurred in patients who had resections for peptic ulcer. The two non-depressive psychotic reactions followed resections for conditions other than peptic ulcer. Two patients had psychiatric illness seemingly unrelated to gastrectomy.

Conclusions

1. It appears to us that gastric resection bears a psychiatric morbidity which is generally unrecognized and which to our knowledge has not been reported previously.

2. From this admittedly small group of cases, it seems that the psychiatric reaction to this surgical procedure is different in ulcer and nonulcer patients.

3. The evaluation of gastric resection, as well as other surgical procedures, in terms of subsequent emotional and social adjustment, is an area insufficiently investigated and deserving of further study.

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Medico-Legal Relations

A Joint Report

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At the request of President Wallace Ritchie, the attention of the Legislative Committee of the Ramsey County Medical Society during 1954 was directed to a study of medico-legal matters, especially those pertaining to expert opinions in personal injury suits.

The President of the Ramsey County Bar Association, Mr. Frank Hodgson, was asked to appoint a committee from the Bar Association to meet with the committee from the Ramsey County Medical Society, and during the summer a number of joint meetings were held.

The report of this Joint Committee is presented herewith.

THE Joint Committee on Medico-Legal Relations has held three evening meetings to discuss topics of common interest proposed by the medical members and lawyer members at separate meetings. In addition, the full committee has considered and agreed upon this joint report, which is based upon the minutes of the discussion meetings and partial drafts prepared by individual members.

The objective the committee has had in view is the improvement of working relations between practitioners of the medical and legal professions. Since those relations have their focus in the court room, most of the problems considered by the committee arise out of or are connected with the presentation of medical evidence to triers of fact. They commonly arise, however, long before trial —almost as soon as the attorney-client relationship is superimposed upon the physician-patient relationship; and they may last longer than the litigation. The difficulties may be of various kinds: questions of financial responsibility and of the loss or unproductive consumption of valuable professional time are more frequent sources of irritation or friction than actual or suspected misconduct.

The committee was both surprised and encouraged to discover that most of these interprofessional troubles are traceable in large measure to ignorance on the part of one profession of matters taken for granted by the other. The mutual education which resulted from this discovery was a valuable experience for all the committee members, and the committee is therefore hopeful that a wider audience may also find something helpful in this report of its deliberations.

Before summarizing the conclusions reached on the particular subjects discussed, it seems wise to restate certain fundamental principles which are known to and accepted by both professions. They form a common point of departure for the thinking of medical and legal practitioners with respect to many of the practical problems, and illuminate—or should illuminate—the solutions to be reached. We identified three such principles, as follows:

1. A trial of issues of fact, whether before a jury, judge or referee, is an essential step in the administration of justice in a civilized society.
2. The quality of the justice thus administered is directly dependent upon the degree of accuracy achieved, in the determination of fact issues.
3. In practice, medicine and law are both arts and reflect intuitive processes and other subjective elements. To the extent that this is unavoidable, the conclusions reached on medical questions are not and cannot be altogether objective—that is, susceptible of scientific proof.

It follows that the trial of issues of medical fact should have as its goal the attainment of as great a degree of objective certainty as possible, not only on the part of the medical witnesses, but also on the part of examining and cross-examining counsel. Mutual recognition and acceptance of the fact that absolute certainty is rarely attainable in

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Report of Joint Committee on Medico-Legal Relations of the Ramsey County Medical Society and the Ramsey County Bar Association.

respect to cases which are brought to trial should encourage pre-trial education of lawyers by doctors. This in turn presupposes agreement as to the respective roles and obligations of attorney, client-patient and physician as to all matters connected with the impending trial. Is the physician expected to charge the time spent on such matters to the attorney, to the client-patient or not at all? Does it make any difference whether he is or has been the client's attending physician, or has been consulted for the purposes of the litigation only? Uncertainty or confusion as to how such questions should be answered often mark the beginning of misunderstanding, and so the committee took them up first, reaching the conclusions stated below.

Medical Fees

Attending or Consulting Physicians.—(For the purpose of this subject matter, these terms mean physicians or surgeons engaged for the purpose of care and treatment of the patient, as distinguished from a physician or surgeon engaged for the purpose of litigation.)

1. Expense of care and treatment is the obligation of the patient, irrespective of the litigation, or the outcome thereof.

2. Expense, if any, for preparation of medical reports for use by patient's counsel is the obligation of the patient.

3. Expense arising from court appearance is the obligation of the patient, irrespective of the outcome of the litigation. Counsel for the patient is under no legal obligation to pay such fees.

4. In the event of recovery, counsel have a moral obligation to use all reasonable measures to assure payment by the client of the statement for services of the attending or consulting physician, not only for the court appearance but also for services rendered. Whenever possible, unpaid statements should be satisfied as part of the process of distributing funds recovered in the litigation. Counsel unable to make arrangements for satisfaction of unpaid medical bills should promptly notify the doctor of such fact.

5. It is improper for the attending or consulting physician to insist, as a condition precedent to his testimony, that counsel guarantee payment of an unpaid bill for medical services, or bill for services to be incurred for the court appearance.

6. If, during the course of treatment, counsel believes that his client should receive further care or treatment from another physician, counsel should confer with the attending physician and in the event of disagreement make his recommendation to his client. Only with the approval of the attending physician or of the client should counsel engage the services of the second physician or consultant and then should make plain that the services, if undertaken, are to be at the expense of the client-patient.

Examining Physicians.—(For the purpose of this subject matter, this term means a physician or surgeon engaged for the purpose of litigation rather than for care and treatment.)

1. Expense of examination and report, as well as x-ray expense and similar costs necessary for a complete examination, are obligations in the first instance of counsel requesting the same. This fact does not relieve the client of his ultimate liability to pay the examining physician or reimburse his attorney.

2. Expense of court appearance is an obligation in the first instance of counsel requesting the same, but subject to the same comment as above.

3. It is proper and desirable practice that whenever an attorney refers a client to a physician for examination, he should do so in writing, stating the terms of the reference. If the attorney disclaims liability for the fee or if the physician doubts the attorney's credit, the physician may be justified in withholding his report until the fee is paid or in declining to testify until mutually satisfactory financial arrangements have been made.

4. It is improper for the physician and the attorney to enter into any arrangement making the payment of medical fees, or the amount thereof, contingent upon the outcome of the litigation. Fixed fees should be established prior to the examination or court appearance.

5. Payment of the examining physician's fees should be made promptly upon completion of his services. Delay in the final disposition, such as appellate procedure, is not sufficient reason for counsel to delay payment.

Medical Fees for Court Appearances.—

1. The fee to be charged by a medical witness for court appearance should be charged after

giving consideration to the following facts: (a) place of trial, (b) travel time involved in appearance, (c) actual time away from office, (d) cancelled appointments due to court appearance, (e) preparation required for testimony, (f) interference with daily schedule due to unforeseen and unavoidable delay, (g) duration of testimony, (h) nature of testimony, (i) ability to pay of party being charged for services.

Medico-Legal Aspects Before Trial

Physicians who undertake to treat an injured party must recognize an obligation to that patient to testify to the facts relating to the injuries; the care and treatment, and the duration of the pain or disabiliy. Attending physicians are not justified in declining to testify because of personal aversion to court rooms, dislike of patient or attorney, unpaid fees, or other personal reasons. The attending physician should realize that his appearance in court is not entirely a matter of discretion on his part. His appearance can be required by service of a subpoena and payment of ordinary witness fees, even though opinion evidence may be sought. Where a doctor has been so required to give an opinion, the Court in its discretion may allow the prevailing party in the law suit (if the doctor has testified in his behalf) to tax against the losing party expert witness fees for that doctor. In the State Courts the law allows the prevailing party to tax $25.00 a day or, in extraordinary cases, up to $50.00 a day in the discretion of the court. There is no such provision in the Federal Courts.

Conference before trial between the physician and attorney is essential to the proper presentation of medical testimony.

1. Physicians should willingly consent to such conference.

2. Professional courtesy should be afforded counsel in arranging the time and place of such meeting.

3. Sufficient time should be allotted to permit full discussion of the problem.

4. Counsel should acquaint the physician with any peculiarities of the law which may be encountered during the medical testimony.

5. The physician is entitled to compensation for time employed in such conferences. An attending or consulting physician may charge the patient; an examining physician, the attorney.

6. Similarly, an attending or consulting physician, on the one hand, or an examining physician, on the other, is entitled to charge the patient or the attorney, as the case may be, for the time necessarily spent in preparing a written report.

7. Counsel should acquaint their clients with the fact that they will be billed either directly by an attending or consulting physician or for reimbursement by the attorney for medical fees paid to physicians for services prior to the trial and for testimony upon the trial.

8. Counsel should extend every consideration to the doctor regarding the time at which he may be expected to appear in court. During the trial, lay testimony may be often harmlessly interrupted to allow a waiting medical witness to testify. Conversely, the doctor should extend every consideration to counsel in arranging for pre-trial conferences.

Histories and Examinations Before Trial

Histories.—

1. It is the privilege of an examining physician, even though the purpose of the examination is limited to qualifying the physician to present expert testimony, to inquire into the patient's history as fully as he deems necessary or desirable in order to arrive at an opinion concerning the patient's medical condition, even though some parts of the history may bear upon liability issues.

2. It is improper for an examining physician to record under the guise of history self-serving statements with respect to liability issues only, volunteered by the patient, or other items of history which the physician knows without further consideration to be irrelevant.

3. It is improper for an attorney to advise his client who is about to submit to an examination to refuse to disclose information within the scope of paragraph 1 or to volunteer statements within the scope of paragraph 2.

4. A patient or his attorney, with the patient's written authorization, may inspect all hospital records pertaining to the patient without the consent of the attending physician or hospital administrator. Such records may be removed, however, only by subpoena.

Examinations.—

1. It is inadvisable for an examining physician to solicit the opinion of an attending physician or even to consult with him except for the purpose of gaining essential information which cannot be obtained from other sources.

2. It is unnecessary and undesirable for an examining physician acting for the opposing side to express any opinion to the party being examined.

3. Findings of the examining physician should be imparted only to the person or party for whom the examination was made.

4. Joint examinations with other physicians expected to be called as witnesses by either side of a litigation tend to impair independent thinking and judgment and are therefore to be avoided except under unusual circumstances.

5. An attorney should not be present at an examination of his client.

6. An attending or examining physician should not express to the patient-client any opinion as to the settlement value of his law suit, nor should he express any opinion as to the effect of the litigation upon the patient-client's health unless he has first talked to the attorney and has ascertained that the attorney has failed or neglected to communicate the opinion to the client-patient.

The Medical Witness in Court

Production of Records.—

1. Medical witnesses are entitled to have the courage of their honest convictions and steadfastly to hold to a well-founded opinion based on sound medical principles. But dogmatic positions and attitudes are not ordinarily well founded, especially in a case in which reputable physicians are prepared to express contrary opinions, and are thus out of harmony with the basic purposes of the trial. Under no circumstance is a medical witness to regard himself as an advocate, or be encouraged to assume such a role by the attorney who has arranged for his testimony.

2. It is not only the privilege but the duty of counsel for the opposite side to explore the foundations of a medical opinion and to expose any weaknesses which may appear either in the basis or content of such an opinion. Provided that the technique employed by the cross-examining attorney is not unjust or ungentlemanly, the witness should not assume an antagonistic attitude. He should recall that in all probability the cross-examination reflects a contrary medical interpretation of the objective facts. An obstinate refusal to recognize the justifiable possibility of a contrary view does not advance the cause of justice. If the witness is "resolving a doubt" in favor of the party calling him, he should candidly admit that he is doing so.

3. Simplicity of expression is the finest attribute of a medical witness as well as of other witnesses. If technical language must be used, its meaning should be explained in lay terms sufficiently so that not only the judge and the attorneys but also the jurors may reasonably be expected to understand it.

The Redress of Grievances

The County and State organizations of each profession maintain standing committees for the hearing of complaints of professional misconduct, but the details are not widely known outside the membership of the respective professions. It seems desirable, therefore, to include a description of these committees as a part of this report.

Ramsey County Society and Minnesota State Medical Association

1. *Grievance Committee.*—This is a committee of five appointed by the President of the Ramsey County Medical Society. It does not hold regular sessions but meets on call whenever the need arises. The duties of the committee are to hear and investigate complaints concerning the professional conduct and ethical deportment of individual physicians and to attempt amicable adjustment. The committee hears complaints from any source: other physicians, organizations or members of the general public. All complaints must be in writing.

2. *Medical Defense Committee.*—This also is a committee of the County Society but is relatively inactive, its duties having been taken over by the State Committee on Malpractice Insurance.

3. *Committee on Malpractice Insurance.*—This is the organ of the State Medical Association which examines complaints in malpractice cases brought against members of the State Medical Association. The committee furnishes forms to

be filled out by the defendant physician in such cases and makes recommendations after the information called for has been compiled and evaluated. The state association also stands ready to provide expert testimony in a malpractice case against one of its members.

4. *Committee on Medical Testimony.*—This also is a committee of the State Association. It handles complaints against doctors appearing as expert witnesses received from other doctors, judges, lawyers and other interested parties. The committee concerns itself with the evaluation of the sworn testimony given by the doctor complained against. It calls in for assistance experts from relevant specialties who examine the testimony and make findings as to whether or not it was justifiable or at least honest. If the findings are that the witness was not acting in good faith or that his testimony was dishonest or if there was any other form of unethical conduct on his part, the committee then recommends to the State Board of Medical Examiners disciplinary action.

Ramsey County and Minnesota State Bar Associations

1. *Ethics Committee.*—This is a committee of the County Association whose functions parallel those of the Grievance Committee of the County Medical Society described in A-1 above. This committee has no disciplinary powers other than the power to recommend expulsion of an erring lawyer from membership in the County Bar Association, and therefore its most frequent recourse is to refer complaints found by it to be meritorious to the State Board of Law Examiners.

2. *State Board of Law Examiners.*—As its name implies, this is not an organ of the Bar Association but an official body. It is mentioned in this context because of the direct reference to it of complaints heard by the Ethics Committee of the County Bar Association, as stated above. The State Board conducts an independent investigation and if it finds probable cause to believe the complaint well founded, it files charges against the respondent attorney with the Supreme Court of Minnesota. The Supreme Court refers the matter to a District Judge, who hears sworn testimony and receives evidence as a referee and makes written findings of fact but no recommendations. The case is then briefed and argued before the Supreme Court on the record made before the referee, including his findings, and the Supreme Court, by written opinion, then determines what, if any, punishment shall be meted out.

3. *Practice of Law Committee.*—This committee of nine members of the State Bar Association is charged with the duty to investigate cases of unethical conduct, including the disbarment and reinstatement proceedings affecting members of the Bar. The by-laws require it "to take such action with respect to the matters investigated as may be directed by the Board of Governors or Supreme Court of Minnesota." The 1954 report of this committee indicates that it has been largely, if not exclusively, engaged in conducting correspondence with reference to grievances against attorneys by their clients. Grievances originating in St. Paul or Ramsey County presumably would be referred by the State Committee to the Ethics Committee of the County Association, rather than processed directly.

Members of the Ramsey County Medical Society Committee were: Drs. E. M. Hammes, Jr., D. L. McCain, B. I. Derauf, H. R. Tregilgas, and Gordon R. Kamman, Chairman.

Members of the Ramsey County Bar Association were: Russell Carlson, Linus Hammond, Lawrence Hazen, Charles Murnane, and David W. Raudenbush, Chairman.

DISEASES OF THE CHEST

Cancer of the lung, industrial lung disease, chronic bronchitis and emphysema going on to pulmonary heart disease, bronchiectasis and the like are, admittedly, less of a social scourge than pulmonary tuberculosis but they also bring in their train social and economic as well as medical problems. Moreover, the advances of thoracic surgery . . . make the inclusion of all chest diseases an economic proposition for the re-orientated chest service of the future. Tuberculosis will continue to lay first claim on our attention for many years ahead.—PHILIP ELLMAN, M.D., *Journal of Royal Institute of Public Health and Hygiene,* August, 1954.

Cholangiography

A Simplified Technique

G. M. A. FORTIER, M.D.
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THE PROBLEM of cholangiography, apart from its controversial aspect, has lent itself to many modifications. Each of these proposes either refinement or increased rapidity of technique, besides excellence of plate exposure; having as an end in view better diagnostic results.

The present article deals with only another practice of preparation for injecting the dye into the common duct. The procedure is offered because it is felt to be an improved, simplified method of operating room cholangiography. To be worthwhile it should properly offer increased rapidity over previously suggested methods, besides adding to the lessening of delay in the operation as a whole while the plate is being developed and interpreted. For the sake of comparison a review of the following methods is made:

Behrend clamps the cystic duct near the gall bladder, then injects the dye directly into that duct through a needle.[1]

Best places dye directly into the common duct through a needle.[2]

Bettman threads a ureteral catheter through the cystic duct, then ligates *to prevent backflow.*[3]

Carter and Gillette inject into the cystic duct, after ligating near the gall bladder. They also attempt to fill the gall bladder with dye under pressure, thereby forcing the media into the common duct.[4]

Caylor, after transecting the cystic duct, threads a ureteral catheter into the common duct, then doubly ligates the cystic duct to prevent backflow.[5]

Hagen and Townsend also pass a ureteral catheter through the cystic duct followed by ligation of that tube.[6]

Hicken inserts a large needle into the gall bladder. After aspirating that organ he distends it with dye through the same needle, forcing the media into the common duct.[7]

Loundsbury, et al, pass a ureteral catheter through the opened end of the cystic duct followed by ligation.[8]

Mehn places a catheter in the opened cystic duct, then ligates.[9]

Mirrizi employs a small cannula fitted with an attached bulb *to prevent backflow.* This he inserts into the open end of the cystic duct.[10]

Robins ligates the cystic duct before injecting the dye into it through a needle.[11]

In the preceding methods certain factors emerge: all of them are practiced in the erroneous belief that dye injected into the common duct under pressure will flow freely back into the gall bladder. Those methods dealing with cystic duct injection are practiced for the most part after the gall bladder has been removed, resulting in a period of inaction while the plate is being developed. Techniques which distend the gall bladder with dye must face a certain percentage of failures due to the imposition of the gall-bladder shadow on that of the common duct. *None is likely to be practiced in the presence of acute cholecystitis.*

The proposed approach to the problem apparently obviates the above mentioned objections. In all the cases studied there has not occurred any but minimal backflow of dye into the gall bladder, even though large cystic ducts have not been ligated. The maneuver is practiced before cholecystectomy is begun. That operation is performed during the waiting period necessary for development and interpretation of the x-ray plate. *The procedure may be carried out in the presence of acute cholecystitis if the ampullary region of the gall bladder is at all approachable.* If there is the least doubt regarding the absence of calculi in the common duct, exploratory choledochotomy is performed. It is in those cases only in which the presenting findings and history definitely assure the absence of calculi that the study is made, except as noted at the close of the article. Small common ducts are not studied.

Technique

A cannula of the type illustrated is employed. It is a Wright sinus cannula. It measures about 5.5 inches in length. The diameter of the bore is about one millimeter, and the tube diameter two millimeters. The end is fitted and ground to receive a standard syringe hub.

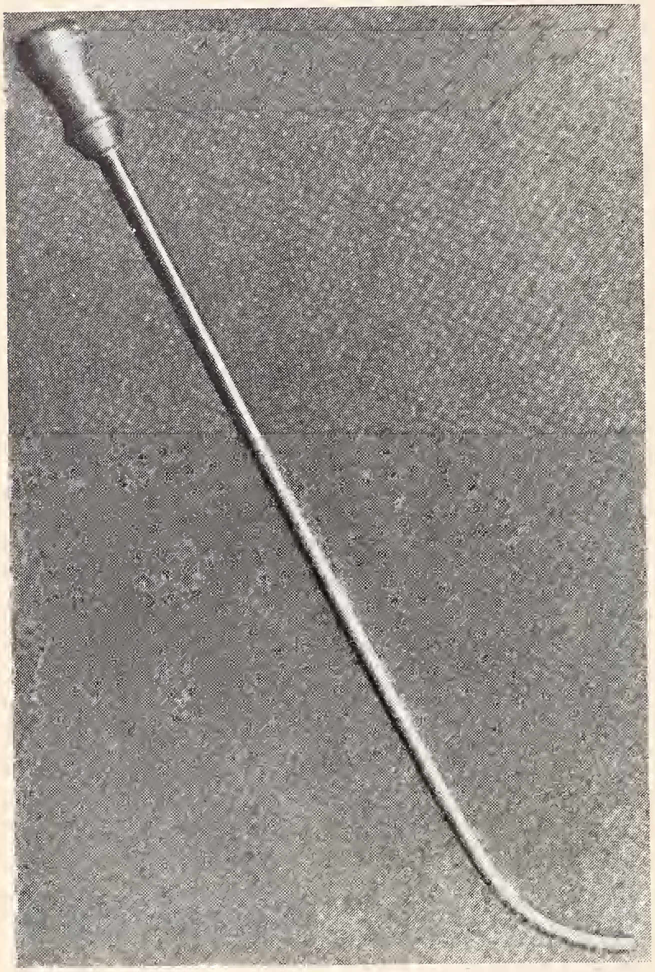

Fig. 1.

Following examination of the field and aspiration of the gall bladder, in necessary cases, one exposes the area of the ampulla. At a point where one usually applies a clamp in this area a small nick or scissor wound is made through the wall. The cannula is inserted into this opening and guided into the common duct by way of the cystic duct. This procedure should be carried out gently, no force being used. Resistance offered by the valves of Heister is not always present, but if so one gently jiggles and maneuvers the instrument, and very little difficulty is encountered, even in small cystic ducts. Proof of the cannula's entry into the common duct is easily verified. In most cases the tip can be observed, as it is rotated, slightly tenting the wall of the duct. In the presence of doubt, aspiration of bile proves the point. The one misfortune which might occur is a mal-direction resulting in the admission of the cannula into the submucosal layer of the cystic duct. Such an occurrence is disproven by the aspiration of bile. Gauze is gently packed about the tube and gall bladder, and, the x-ray unit being in readiness, the dye is injected. *No blocking of the cystic duct is required.* On no occasion has any backflow of dye been observed on the x-ray plate, except in minimal amounts.

Following exposure the cannula is removed, the opening clamped, and the cholecystectomy begun. This procedure is well progressed or finished by the time the x-ray plates are developed and returned to surgery for study. The time lost, therefore, is minimal as the procedure becomes an active part of the whole operation rather than being a disconnected interposition necessitating an inactive waiting period, as would be true of injection of the common duct with dye followed by x-raying after the gall bladder had been removed.

This simple method is employed also, on occasion, on two other types of cases. When several calculi are known to be present in the common duct at least the minimal number is learned, allowing the enjoyment of that little extra security such knowledge offers. In the case of acute cholecystitis when edema is of such extent as to invite disaster if the common duct is sought for, the assurance that the stones are present in the common duct might, in a particular case, influence one's decisions at the operating table as well as during the postoperative period.

The above outlined procedure has been employed in seventy-five cases. In no instance has any immediate or delayed harm been apparent. In no case was there any suggestion that the procedure affected in any degree the postoperative morbidity. There were no cases of pancreatitis. The criteria for considering the common duct to be free of calculi were narrowly limited. The results, therefore, of the seventy-five case studies were negative. The postoperative follow-up studies over a period of four years produced no instance of presence of overlooked calculi.

(Continued on Page 266)

Choking Accidents

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THE ACTUAL incidence of choking accidents is difficult to estimate, but these experiences in some form are very common. Few persons pass through childhood without experiencing at least a mildly disturbing episode of having had a particle of food "go the wrong way." In an occasional instance such an episode has serious possibilities. Newspaper references to asphyxia occurring while eating are not rare. When death occurs from the immediate effect of a foreign body lodging in the air passages, the site of this obstruction is usually in the larynx. More often than not a foreign body entering the air way is coughed up. Those that cannot be coughed up are usually found in a bronchus where a varying degree of morbidity occurs, depending on the size and type of the substance and the duration of its stay in the lung.

Etiology

In order of importance and frequency the following causes for choking accidents can be listed:

1. *Hurried eating.*—That often means inadequate mastication or chewing of food. In the edentulous who wear false teeth, chewing tends to be less thorough, or if attempts are made to chew without any teeth, the process of mastication is negligible except for very soft foods. Likewise, the infant can only grind up food to the extent of his teeth, and when he puts such a substance as a peanut in his mouth he has no molars to grind up this type of food.

2. *Lack of concentration on the business of eating, particularly among small children.*—Among adults, conversation is part of the pleasure of a dinner party and leads to leisurely eating, which should insure proper mastication. With the small child, distraction of his attention from the business of eating may be another matter. The child who so often may gluttonously shove food into his mouth is liable to the hazard of getting the food the wrong way if his attention is distracted by other children, or he attempts to play as he eats. Boisterous play among children at a dinner table is an excellent setting for a choking accident.

3. *Allowing a small infant to play or crawl where there are small objects within reach that he could put in his mouth.*—He will follow his natural instincts, which means that anything he picks up will be put in his mouth, if there is room. Pins of various types, nutmeats, popcorn kernels, beads, buttons, eggshells, pieces of wood, parts of toy instruments et cetera, have all been removed from the trachea or bronchi of small infants.

4. *Holding objects in the mouth.*—Professionally there is the seamstress who holds pins in her mouth, or the carpet layer with a mouth full of tacks or the carpenter with his nails. Since these are adults and their minds are usually on their work, choking accidents are uncommon. Also, there is a greater tendency for the foreign body to be swallowed than for it to go into the airway because the co-ordination of the swallowing mechanism and the laryngeal function is better developed in the adult. However, the size and shape of the type of foreign body mentioned precludes much of a possibility of actual asphyxia with an accident from this cause.

5. *Injury to the larynx from external trauma.*—A blow across the external surface of the larynx, particularly if localized at the prominence of the "Adams Apple" can cause enough progressive soft tissue swelling within the larynx to seriously embarrass the airway. Whenever there is loss of voice following external injury to the laryngeal area, or any complaint of difficulty in breathing, careful and continuous observation for the development of serious respiratory distress is in order.

Since this discussion is concerned only with accidents, other causes of choking have not been considered. Inflammatory diseases of the larynx plus the occasional case of edema from allergic reaction and tumors outrank in incidence foreign bodies as a cause of asphyxia, even though serious inflammatory lesions are less common than they were prior to the advent of the sulfonamides in

1937 and the antibiotics in 1942. In addition, the management of these cases from the standpoint of skill in early diagnosis and supportive care has improved.

Management

The immediate management of a choking accident is usually in the hands of a layman. A time-honored practice which the average adult follows is to turn the child "bottom side up" and slap him on the back of his chest. Presumably this maneuver is followed to utilize the force of gravity while the slap or pounding on the back of the chest may serve both to dislodge the foreign substance and aggravate the cough reflex. It seems to work in a fair percentage of cases and it seems logical. The only possible hazard in this would be in the instance where a foreign body had already passed through the glottis and is in the trachea or a bronchus, and that it might become lodged in the glottis as it came back when the patient was tipped "bottom side up."

Another maneuver that has some merit is to incite gagging so as to cause vomiting by "sticking a finger down the throat." This would be most effective in a situation where a foreign body is lodged at the introitus to the esophagus and in this position presses on the larynx to partially embarrass respiration.

When these measures do not bring relief, the hazard is often extremely serious and calls for action that the layman is not equipped to provide. The obstruction is usually at the glottis with impending asphyxia. Occasionally, the site of obstruction may be lower within the trachea from a foreign body lodged at the carina and partially obstructing both bronchi. In the latter event asphyxia is not as rapid.

Only tracheotomy will relieve complete glottic obstruction, and it has been only in extremely rare instances that a layman has had the knowledge of what to do. If a physician can be reached in time, he may have to act without adequate tools. Tracheotomies have been done with a jackknife. This discussion was not intended to cover the details of surgical technique of tracheotomy but the following general maneuvers can appropriately be outlined.

In the case of a small child the operator may find it helpful to be seated, if that is possible, with the child on his back across the operator's knees and with the head somewhat extended. The right-hand operator then palpates the site of incision with the index finger of the left hand, starting first with the prominence of the thyroid ("Adam's Apple") and then feeling the flat surface of the membrane between the firm surface of the thyroid cartilage and the rounded hardness of the cricoid ring below. The trachea begins below this ring. This same left index finger and thumb then act to spread tense the soft tissue over the trachea as a knife in the right hand makes a midline incision and with succeeding strokes bares some trachea. The isthmus of the thyroid is the most vascular structure encountered. If it can be displaced that is desirable, but because of the urgency of the situation a direct incision through it may be necessary. The trachea is next opened by a vertical slit through at least two rings or a horizontal slit through membrane below the first ring, and the margins of the slit are spread by any available means with an instrument or object small enough to keep the edges pried apart.

A more accessible site in certain instances for opening into the trachea below the level of the glottis is through the membrane between the thyroid cartilage and the cricoid ring (crico-thyroid membrane). A transverse slit through this membrane can be made usually without the necessity of transversing any very vascular tissue. Then after an airway has been established and the patient resuscitated, an orderly tracheotomy can be performed and the first incision repaired.

Prevention

The means of preventing choking accidents have already been suggested. These may be summarized as follows:

1. Avoid hurried eating and incomplete mastication of food.

In children this is most important. The business of eating should be their sole activity at the time. When mixed with play or boisterous conduct, a mouthful of food may easily go the wrong way.

Do not feed nuts in any form, or cookies containing nuts, hard candy, popcorn or firm pieces of fruit to infants. Be sure that any seeds are removed from oranges, et cetera.

2. Do not allow infants to play on a surface where there may be any small toys or objects within reach or a floor that is not clean of pins, toothpicks, hard food substances, seeds, et cetera.

3. The mouth should not be a temporary receptacle for pins, tacks, nails, et cetera. Special care should be observed on a farm so that small children do not put shelled corn or other seed materials in the mouth.

4. When the neck has been injured from a blow received in the area of the larynx, close observation is important if the voice is at all impaired.

Seminar

MULTIPLE SCLEROSIS

Diagnosis, Diagnostic Errors and Treatment

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ALTHOUGH multiple sclerosis was described more than a century ago, it remains one of modern medicine's baffling maladies. The etiology remains unknown, the prognosis uncertain, and the treatment unsatisfactory. Though at first considered to be a medical curiosity, it is now estimated to afflict approximately 250,000 individuals in the United States. The neurologic disorder the illness produces is exceedingly variable. Only a small percentage of cases are of the classical clear-cut variety, consisting of ataxia, muscle weakness, nystagmus, diplopia, impaired visual acuity, intention tremor, scanning speech, and euphoria. These textbook cases can be readily diagnosed by the medical student; however, there are a certain number of cases that cannot be properly diagnosed by the astute neurologic consultant, especially during the patient's first attack. The cautious clinician will in such instances make the diagnosis of multiple sclerosis on an exclusion basis. To date, no fresh information can be supplied to help in the establishment of the diagnosis of this demyelinating disease. Nevertheless it is conceivable that with the newer biochemical techniques being utilized in the research laboratories, the questions regarding the etiology, diagnosis, and treatment of multiple sclerosis will be firmly answered in the near future. Until such answers are supplied, renewed emphasis on the establishment of the clinical diagnosis is warranted.

The "pathologic unit" in multiple sclerosis consists of a circumscribed area of nervous tissue in which primarily the myelin sheaths of nerve fibers are damaged with resultant transitory or permanent axone damage. If complete destruction takes place, there results sclerotic, gliotic plaque formation.

Typically, the pathologic foci are multiple and spread in scattered fashion throughout the white matter of the nervous system. The neurologic symptoms, transient or permanent, depend on the regions of the nervous system involved, the intensity of the demyelinization, and subsequent axone impairment. Usually, a new plaque gives rise to maximal dysfunction within forty-eight hours, followed by a moderately semi-stable period of several weeks and then a gradual improvement. The degree of improvement parallels the myelin reformation and intactness of the nerve fibers.

What produces this neurologic disorder? The early pathologists believed that glial overgrowth was the prime factor, hence the term sclerosis. The disease has been attributed in the past to various infectious agents including spirochaetes and viruses; but no positive evidence has ever been produced. More recent theories ascribe the demyelinization to circulatory abnormalities including venular thrombosis, altered coagulability and sludging of blood; to myelin-splitting ferments; to hyperergic reactions; to altered fat metabolism; or to some unknown toxin. It is most probable that many of the newer theories are linked in some way producing a chain of biochemical events; and it remains to be determined which are the prime factors. We need to know more about the nature of myelin. Isotopic tracer substances, enzyme, and lipid studies are being investigated to give added information about myelin metabolism.

Diagnosis

The major criteria for diagnosis of multiple sclerosis are wholly clinical. To this date no reliable positive laboratory test is available. The diagnostic clues are present in the manner in which the neurologic disorder is produced. The vast majority of cases occur in the first half of adulthood. Rarely does the initial attack occur in an individual before the age of twenty or over forty. Although there is no consistent pattern, the history of remissions and exacerbations with

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From the Neurology Section, Veterans Administration Hospital and University Hospitals, Minneapolis, Minnesota.

gradual increasing neurologic disability is an important criterion. Another major criterion concerns evidence of multiplicity of neuropathologic lesions, based on neurologic signs, that unequivocally require scattered or disseminated foci. Thus it must be demonstrated that multiple sclerosis typically is a neurologic entity scattered both as to time and place. Nevertheless clinicians have seen and will continue to see cases that lack these criteria; for example, the steady progressive variety, the acute fulminating variety, and the single focal variety. The disease may begin with a single symptom of transient duration, as visual blurring, diplopia, weakness or awkwardness of limb, or numbness, to be followed by an interval of well-being for weeks, months or years before the onset of a second attack. Oftentimes the middle-aged adult during an acute attack will have forgotten an original bout of neurologic dysfunction unless specific inquiry is made. The spinal form of multiple sclerosis may reveal only a steadily progressive spastic paraparesis. Rarely an explosive fulminating variety with wide dissemination of lesions may initiate the onset and result in early death. As the diagnosis of multiple sclerosis usually carries with it a grave prognosis, each case warrents critical evaluation and especially so the atypical cases that do not fit in the "time and place formula." A diagnosis of multiple sclerosis made on the basis of a single focal sign, for example diplopia, must remain presumptive pending the subsequent development of neurologic signs of dysfunction.

The demyelinating process of multiple sclerosis has a predilection for certain areas of the nervous system; chiefly the posterior and lateral columns of the spinal cord, the cerebellar pathways, the medial longitudinal fasciculus, and the optic nerves. When all or most of these areas of the nervous system have been involved as a result of recurrent attacks, the diagnosis of multiple sclerosis is not difficult.

When considering the differential diagnosis, it is helpful to subdivide multiple sclerosis into various clinical types, depending on the major site of neurologic dysfunction. Thus we may have either a predominant cerebral, cerebellar, brain stem, cranial nerve (chiefly ocular), or spinal clinical subtype, or combinations thereof. Brain tumors, encephalitis, and subdural hematomas are to be differentiated from the cerebral variety; myasthenia gravis, carotid artery aneurysms, pineal, sellar and parasellar tumors from the ocular nerve form; acoustic neuromas, cerebellar tumors, pontine gliomas from the brain stem and cerebellar form; subacute combined degeneration, Friedreich's ataxia, amyotrophic lateral sclerosis, platybasia, cord tumors, and midline cervical discs from the spinal subtype of multiple sclerosis.

Various laboratory tests and procedures are utilized to give additional aid in differentiating a suspected case of multiple sclerosis from a host of neurologic disorders. The most commonly performed laboratory test is the lumbar puncture for examination of the spinal fluid. Even though in a small percentage of cases the cell count and spinal fluid protein are elevated, marked increases of either one or both strongly indicates that the disease is not multiple sclerosis. In no more than one-fourth of the cases does the characteristic first zone colloidal gold curve appear, and then usually occurring in the typical easily diagnosed cases. All cases with pure spinal signs require the Queckenstedt jugular compression test to rule out a spinal fluid block. It can be stated that a completely normal spinal fluid examination tends to enhance the diagnosis of multiple sclerosis rather than the reverse. Although various biochemical tests have been performed in the research laboratories on the spinal fluid and blood of patients with known multiple sclerosis, there still does not exist a practical test that will establish the diagnosis with the certainty present for diagnosing central nervous system syphilis.

Further specialized tests, including skull and spine x-rays, electroencephalography, myelography, angiography, and air encephalography are performed when indicated to rule out space-occupying lesions, which by the nature of their location might suggest multiple sclerosis. Myelography is a strongly recommended procedure in virtually all multiple sclerosis "suspect" cases that do not have findings or symptoms above the cervical cord level.

The greatest danger that can befall a patient with neurologic dysfunction is to be diagnosed and treated as having multiple sclerosis, when in actuality myasthenia gravis, chronic subdural hemotoma, benign spinal cord or brain tumor, or other specifically treatable neurologic entities exist. In the workup of the patient the clinician should pursue to uncover a specifically treatable disease if reasonably present in the differential diagnosis. As an illustration the patient with a combined degeneration syndrome (posterior and lateral tracts of spinal cord involved) may therefore require examination of the blood and urine, including studies for heavy metals (lead, arsenic, and mercury), gastric analysis, bone marrow; and in the event all these tests are normal, spinal fluid analysis, x-rays of the spine, and myelography which includes the entire cervical region are in order before labeling this patient as having the spinal form of multiple sclerosis.

Out of the many cases of multiple sclerosis seen, diagnosed, and treated at the neurologic service of the Minneapolis Veterans Administration Hospital from 1945-1955, the following cases are presented to demonstrate the need for constant surveillance and re-evaluation of all patients labelled with the diagnosis of multiple sclerosis. Furthermore these cases, all proven not to be multiple sclerotics, should caution all clinicians who treat neurologic patients, to be wary of all apparent multiple sclerotic cases that are not of the classical Charcot variety.

Case 1.—J. L. B., aged forty-three, was first admitted to Minneapolis VA Hospital, June, 1945, with the complaint of double vision followed shortly thereafter by weakness and impaired control of his lower extremities. During this period of hospitalization his diplopia improved only to return following an upper respiratory infection. His gait also deteriorated and it was noted that there was mild speech impairment with perioral weakness.

Positive neurologic findings included ptosis of the right upper eyelid, paresis of the right superior and lateral rectus, unsteady gait, absent abdominal and cremasteric reflexes, and symmetrically hypoactive deep tendon reflexes. Spinal fluid examination was entirely normal. A diagnosis of multiple sclerosis was made and patient was discharged much improved September, 1945.

The patient was readmitted to the Minneapolis VA Hospital on October 18, 1948, as a multiple sclerotic in an acute exacerbation. Two weeks prior to admission diplopia appeared on right lateral gaze with progression of diplopia in all fields of vision. He also noted progressive paresis of his arms, hands, and legs with a tendency for his ankles to buckle.

The significant positive neurologic signs on this admission included paresis of the right superior and internal rectus and of the left internal and inferior rectus, a right ptosis, and a mild to moderate generalized muscle weakness of the extremities which became more manifest on exercise. There were no positive pyramidal, posterior or spinothalamic column involvement. The previous diagnosis of multiple sclerosis was questioned in view of the age of onset and the chief neurologic signs of muscular paresis. A diagnosis of myasthenia gravis was considered and established following a presumptive prostigmine test. Twenty minutes after receiving 1 mg. of prostigmine with 1/150 gr. of atropine sulfate there was profound improvement of muscle strength with disappearance of the right ptosis.

Summary.—A forty-three-year-old veteran was first diagnosed as multiple sclerosis and treated for this disease until myasthenia gravis was firmly established three years later.

Case 2.—T. O. G., aged fifty-three, was first admitted to Minneapolis VA Hospital April 20, 1950, with a long history of gait impairment, incoordination of extremities, visual blurring, slurred speech, and intermittent headaches. More recently, he had had increasing difficulty with his right extremities, and the headaches which were previously considered to be of mild intensity became severe. A month prior to admission he had had two unconscious episodes, one lasting approximately five hours. A diagnosis of multiple sclerosis was first made on this individual in 1941 at a neurologic center and he carried that diagnosis ever since although seen and examined by many physicians at various clinics over a nine-year period.

On neurologic examination, it was noted that the patient was disoriented and confused. There was marked cranial nerve involvement including a bilateral three to four diopter papilledema, a right lateral rectus paresis, severe nystagmus in all directions being most severe on left lateral gaze, hypalgesia of the right side of the face with a decreased right corneal reflex, a right peripheral facial paresis, and a right nerve deafness. Other positive neurologic findings included ataxia of all extremities with intention tremors of the arms, worse on the right, hypalgesia of the left side of the body, bilateral positive Babinski, and bilateral ankle clonus and a mild nuchal rigidity.

Information received from a hospital where he was previously examined revealed a spinal fluid protein in excess of 200 mg. per cent. Significant laboratory data performed at this admission revealed pressure erosion of the sella turcica on skull x-rays and a diffusely abnormal electroencephalogram. A diagnosis of a right cerebello-pontine angle tumor was tentatively made, and a right suboccipital craniotomy was performed several days after admission revealing a large acoustic neuroma. There was a stormy postoperative course with resultant neurologic disability.

Summary.—A fifty-three-year-old veteran who had carried a diagnosis of multiple sclerosis for nine years to explain what was considered to be scattered neurologic signs and symptoms and without the characteristic history of exacerbations and remissions. There was progressive neurologic disability with the later development of signs of increased intracranial pressure which being present on his first admission to the Minneapolis VA Hospital indicated a brain tumor diagnosis and at surgery a right acoustic neuroma was removed.

Case 3.—R. B. H., aged twenty-four, was first admitted to Minneapolis VA Hospital December 9, 1947, on transfer from the VA Hospital, Fargo, North Dakota, with a differential diagnosis of chronic encephalitis and brain tumor. History included an episode of diplopia of two weeks' duration in November, 1943. In December, 1944, while in the Southwest Pacific, he noted blurring of vision and shortly thereafter developed diplopia and bilateral ptosis. The ptosis improved but the diplopia persisted to the present time. In November, 1945, he developed a coarse tremor of the right hand and arm and a month later noted numbness and awkwardness in this arm and hand which later spread to the right leg. Six months prior to admission, his speech was somewhat slurred and dysarthric, and he was unable to walk for two months preceding admission.

Neurologic examination revealed questionable temporal pallor of the optic discs bilaterally. There was a moderate bilateral ptosis with severe impairment of conjugate gaze and paralysis of upward and downward gaze and bilateral lateral rectus paresis. The eyes at times would move independently of one another. The pupils were small and fixed and did not react to light and accommodation. There was hypalgesia of the right side of the face with a reduced right corneal reflex. The motor system revealed a moderate hemiparesis of the right side with right hyperreflexia. Co-ordination tests were poor in all four extremities. Sensory examination disclosed impairment of all modalities on the entire right side. Mentally the patient appeared somewhat dull and there was a mild memory deficit for recent events.

From a neurologic standpoint a diagnosis of multiple sclerosis was made in view of scattered multiple lesions with suspected involvement of the mid brain, medulla, spinal cord and cerebral hemispheres. Included in the differential was a brain stem tumor. Laboratory data included a normal spinal fluid examination in all respects and a normal skull x-ray. A pneumoencephalogram was recommended to rule out a brain tumor diagnosis, and it disclosed a mild generalized dilatation of the ventricular system with normal or slightly enlarged subarachnoid spaces and basal cisterns, which indicated a degenerative process. Special serologic studies were negative for antibodies against eastern and western equine encephalitis. Patient was discharged on January 7, 1948, with a diagnosis of multiple sclerosis.

The patient expired at another hospital early in 1949, and an autopsy revealed an infiltrating pinealoma.

Summary.—A twenty-four-year-old veteran with a history of remissions and exacerbations with resultant progressive neurologic disability over a five-year period was diagnosed as having multiple sclerosis in view of multiple neurologic findings and a negative spinal fluid and pneumoencephalographic study. Post-mortem examination revealed an infiltrating pinealoma with severe involvement of his brain stem.

Case 4.—B. F. S., aged twenty-six, was first admitted to Minneapolis VA Hospital December 18, 1947. This patient carried a service-connected diagnosis of multiple sclerosis, previously diagnosed as post traumatic psychoneurosis. The patient gave a history of loss of control of his left leg with mild exacerbations and remissions over a four and a half year period. Patient first noted this disability shortly after bomb explosion during combat duty in 1943. He was examined at various service hospitals for this complaint. In spite of temporary remissions there was progressive impairment of his left leg, and in the fall of 1946 he was unable to work because of difficulty in ambulation.

Neurologic examination disclosed a spastic paresis of his left lower extremity, hyperreflexia of the lower extremities including bilateral ankle and knee clonus and bilateral positive toe signs and absent superficial abdominal reflexes. The pyramidal tract signs were more marked on the left. There was impaired superficial sensation in an area from T-2 to T-12. Positive laboratory data included a spinal fluid protein of 244 mg. per cent and a positive jugular compression test. Spinogram disclosed a block suggestive of an extramedullary lesion at T-1, and on laminectomy a neurofibroma was removed from the left C-8 nerve root.

Summary.—A twenty-six-year-old veteran who was previously diagnosed as having post traumatic psychoneurosis and later changed to multiple sclerosis was proven to have a neurofibroma of the cervical spinal cord four and a half years after his original symptoms.

Case 5.—A. F. P., aged twenty-three, was first admitted to Minneapolis VA Hospital December 30, 1946, with the complaint of awkwardness and impaired feeling in his right hand and arm. Objects would drop from his right hand. On examination there was mild weakness of his right hand and arm with an intention tremor. There was impairment of position, vibratory and discrimination sense in the right hand with no objective superficial sensory impairment. The deep tendon reflexes were definitely increased in the right upper extremity and suggestively increased in the right lower extremity with an equivocal Babinski sign. Spinal fluid examination was negative including the jugular compression test except for a spinal fluid protein of 60 mg. per cent. X-rays of the chest, cervical spine, and skull were negative. A focal mass lesion of the left sensory cortex was considered, and a pneumoencephalogram was performed which was entirely normal. The patient was discharged with the tentative diagnosis of multiple sclerosis and recommended that he return in six months for follow-up examination.

He was next admitted July 9, 1947. On this admission there was increasing difficulty with the function of his right arm and hand, and there was progressive gait disturbance, and three months prior to admission he had noted urinary urgency. Examination on this admission revealed moderate weakness of the right arm and leg, right hyperreflexia, a suggestively positive head and jaw reflex, positive Babinski on the right and a suggestive Babinski on the left. Intention tremor and past pointing was noted in the right arm, and there was impaired deep and superficial sensation in the right hand, including astereognosis. On spinal fluid examination the spinal fluid protein was now 36 mg. per cent and the colloidal gold curve was 0012100000. Jugular compression test was again negative. A myelogram was performed which was not entirely satisfactory; however, no significant defects were noted in the cervical region. The diagnosis of multiple sclerosis was again made, and after a course of hospital treatment he was discharged.

On the patient's third admission in December, 1947, there was increasing difficulty of his right upper extremity and increasing gait disturbance. The neurologic examination now disclosed marked pyramidal and posterior column damage with involvement of all four extremities, greater on the right. The patient's gait was ataxic and somewhat spastic. The coordination test disclosed adiadokokinesis and bilateral intention tremor. In view of the past work-up including a normal pneumoencephalogram and a negative spinogram, the diagnosis of multiple sclerosis of a progressive variety was again made and the patient was referred to the Corrective Rehabilitation Department for therapy.

When seen on his fourth admission January 11, 1949, the patient had marked difficulty in ambulation and because of the severe impairment in gait and the loss of sensation and control in his arms he was considered totally disabled. Neurologic examination revealed findings similar to those on his last admission; however, the findings were much more intense with severe involvement of the posterior and lateral columns. On this admission, the patient complained of pain in the right posterior cervical region with radiation into the right shoulder.

In reviewing this patient's case, it was noted that there were no findings at any time to suggest any significant cranial nerve involvement and that this patient's course was one of a progressive variety. The findings of incoordination and tremor noted in the extremities could be attributed to severe posterior column damage, and it was felt that a spinal cord tumor could account for all his findings. Therefore a repeat cervical myelogram was performed which revealed a constant block indicating an extramedullary lesion at C-4. A cervical laminectomy was performed, and an extra- and intradural dumbell neurofibroma was removed. Postoperative course was uneventful, and this patient showed marked neurologic improvement in the subsequent months and when seen two years later the patient was able to work a full schedule on his farm with the only complaint of ease of fatigue and mild paresis and numbness of his right arm and hand.

Summary.—This veteran had a three-and-a half-year history of progressive neurologic disability beginning in his right arm and then spreading to involve the other limbs. A diagnosis of multiple sclerosis was made on three separate admissions to the Minneapolis VA Hospital. Original laboratory procedures including pneumoencephalogram, spinal fluid examination and myelogram were not remarkable. The spinal fluid protein was never elevated beyond 60 mg. per cent and even though there was progressive neurologic impairment the spinal fluid protein actually decreased. The jugular compression test was never abnormal. A repeat myelogram performed on his fourth admission revealed a block at C-4, and at surgery a dumbell neurofibroma was removed.

These five cases, two with cervical cord tumors, two with brain tumors that involved the brain stem and cranial nerves, and one with myasthenia gravis, illustrate that even with the history of "so-called" remissions and exacerbations and suggestion of multiplicity of lesions, the diagnosis of multiple sclerosis requires constant scrutiny. Fortunately, four of these patients received a change of diagnosis during life to permit and receive specific therapy and in three of these (myasthenia gravis and two cord tumor cases) the results were most gratifying.

Treatment

As can be expected in all illnesses wherein the etiology is unknown a great number of drugs, agents, and techniques have been utilized in the treatment of multiple sclerosis. It is doubly difficult to evaluate the success of any therapeutic regime in this condition because of the inherent problems in establishing the diagnosis, the uncertainty of the particular course, and the natural tendency for the illness to remit. Even though there is no specific therapy and in spite of the fact that we have all seen cases of multiple sclerosis fail to respond or regress under treatment, a nihilistic approach is unwarranted. Individuals with this chronic neurologic disturbance look to the practitioner for hope, and help, and if informed that no treatment exists they may deteriorate emotionally as well as physically. Furthermore, many will seek out the unqualified, unethical practitioner. The challenge these patients and their families present should be met by an honest endeavor on the part of the respon-

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sible physican to "treat," fully realizing the limitations of any therapeutic program. A sincere medical interest on the part of the doctor, utilizing a positive dynamic approach, will result in many satisfying improvements. The increased awareness of corrective rehabilitation during the past decade along with newer non-specific pharmacologic agents make possible a broad therapeutic program.

For many young adults afflicted with multiple sclerosis, such a positive approach will bear dividends, if only to teach the patient how to adjust to his disability and utilize to full advantage the remaining motor function.

The full therapeutic regime includes the more commonly used drugs listed in the outline below: physical and corrective therapy, avoidance of physical and emotional stress, and employment of psychotherapeutic techniques.

Drug Therapy—Multiple Sclerosis

I. Vitamins and Tonics
    (1) Thiamine chloride (B_1) 100 mg. b.i.d. (O)
    (2) Vitamin B Complex—b.i.d. (O)
    (3) Vitamin E—Mixed tocopherols—wheat germ, 50 mg. t.i.d.
    (4) Vitamin B_{12} 100-1000u 3X week I.M.
    (5) Multiple vitamins
    (6) Crude liver extract (3-6 units weekly)
II. Vasodilators and Vascular Agents
    (1) Histamine—2.75 mg. (1 mg. of base) in 250 cc.—500 cc. N.S. intravenously X 20-30 days
    (2 Nicotinic Acid—50-100 mg. plus to produce flush reaction q.i.d.
    (3) Etamon—200-400 mg. I.M. b.i.d.
    (4) Amyl nitrate—inhalation
    (5) Papaverine—75 mg. q.i.d.
    (6) Dicoumerol—100 mg. plus daily to alter prothrombin time to 30-50 per cent of normal
III. Steroid Therapy
    (1) Cortisone—50-100 mg. daily X 30 days and/or
    (2) ACTH
IV. Anti-allergic Drugs
    (1) Histamine—as above
    (2) Pyribenzamine—50 mg. q.i.d.
    (3) Benadryl—50 mg q.i.d.
V. Spasticity Alleviating Drugs
    (1) Mephenesin—250-500 mg. plus q.i.d. (O)
    (2) Quinine—5-15 gr. t.i.d. (O)
    (3) Prostigmine—15-45 mg. of the hydrobromide q.i.d.
    (4) Atropine and atropine-like drugs
VI. Miscellaneous Agents
    (1) Piromen
    (2) Sodium succinate
    (3) Adenosine mono and triphosphate
    (4) Nucleotides
    (5) Isonicotinic acid hydrazide
    (6) Typhoid fever vaccine
    (7) Low fat diet

During an acute attack a combination of oral multiple vitamins, including large extra doses of thiamine, intravenous slow drip histamine infusion, Vitamin B_{12}, 1000 micrograms I.M., and physical therapy as required is an excellent beginning program to be followed for two to four weeks. If unsuccessful, the other pharmacological agents are to be given a trial.

The utilization of comprehensive corrective rehabilitation techniques concomitantly with the drug therapies will (1) aid in the prevention of secondary joint fixation, (2) prevent tissue breakdown, (3) keep the patient in positive protein balance, (4) teach the patient how best to handle himself in the presence of his various disabilities, (5) to utilize his residual capacities to fullest extent, and (6) synergistically the psychologic aspects of such a positive program will act as a catalytic factor, mobilizing an all out effort on the part of the patient. Even a bedridden multiple sclerotic patient, who can be rehabilitated to the use of a wheel chair with the potential of increased self-care activities, is a significant albeit minimal goal. Constant vigilance is required to prevent the development of immobilization and its attendant evils. Inactivity hastens the development of complications such as decubiti, contractures, protein and calcium imbalance, genitourinary infections and calculi formation, and draws heavily on the emotional health of the patient and also those that treat him.

Physical therapy can and does lessen these destructive complications. The patient undergoing dynamic corrective rehabilitation must be cautioned not to exercise to a state of physical exhaustion.

The physician with the above therapeutic armamentarium plus the benefits derived from physical therapy, and cognizant of the patient's and family's psychotherapeutic needs can and will be of utmost service to his multiple sclerotic patients.

Summary

The criteria for the establishment of the diagnosis of multiple sclerosis are reviewed. With the absence of the "time and place" formula the diagnosis should be made only on an exclusion basis, subject to change.

Five cases are reported that were originally diagnosed in error as multiple sclerosis.

The therapeutic program for each patient must be custom-tailored along a positive dynamic course, utilizing the various drugs available and exploiting corrective physical therapy to its fullest extent.

Laboratory Aids

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George G. Stilwell, Editor

FUNCTIONING OVARIAN TUMORS

MALCOLM B. DOCKERTY, M.D.
Rochester, Minnesota

Following the discovery by Virchow of the production of bile in a pulmonary metastatic tumor originating from a hepatoma, it became apparent that utter lack of function was no longer implicit in the definition of a neoplasm. With acquisition of knowledge concerning tumors of ductless glands came the realization that such tumors produced, often in large amounts, the hormones that were native to their tissue of origin. Such anatomic and physiologic evidence of function as relates to certain ovarian neoplasms is the subject of this report.

Granulosa Cell Tumors.—Comprising perhaps 2 per cent of all ovarian neoplasms, this growth characteristically feminizes its host. Children so afflicted are sexually and somatically precocious, with an early onset of "menses." During the period of sexual maturity, patients who have such tumors exhibit amenorrhea or anovulatory bleeding, and infertility is almost invariably present. Older patients often experience a postmenopausal return of "menses," often accompanied by soreness and enlargement of the breasts.

With such a history, the causative tumor often may be recognized grossly as a granulosa cell neoplasm, for it is typically unilateral, solid, lobulated and encapsulated, with a tan cut surface that is reminiscent of liver sausage. The uterus is unduly enlarged, containing fibroids in 50 per cent of cases. When the patients are more than fifty years of age, cancerous changes are present in the uterus in 15 per cent of instances. The occurrence of unilateral or bilateral carcinoma of the breast is another interesting side effect, possibly produced by the estrogen elaborated by the ovarian tumor cells. Suppression "atrophy" of the contralateral ovary is observed among patients of all ages. The "plush" condition of the vaginal mucosa in postmenopausal women suffering from the effects of granulosa cell tumors is still another evidence of function of such tumors.

Microscopically, the tumor shows a basic composition of granulosa cells in a variety of patterns of interest to both the gynecologist and the pathologist. An admixture of theca cells frequently can be found on careful search. The cellularity of the tumor is such that failure of recognition usually results in an "overdiagnosis" of ovarian sarcoma.

Aggressive tendencies relating to recurrence and metastasis are observed in about 25 per cent of patients in the older age groups, wherein policies of radical surgical intervention are also dictated by the high associated incidence of uterine carcinoma. Irradiation is extremely useful when total removal has not been possible. Among younger persons with granulosa cell tumors, simple removal of the tumors has been satisfactory.

Theca Cell Tumors.—Occurring either in pure form or hybridized with granulosa cell tumor, this second member of the feminizing group is observed principally among older women.

Theca cell tumors are unilateral, solid, lobulated, encapsulated yellowish-brown growths of moderate size. Pathologic changes in the uterus, including endometrial carcinoma, frequently are observed and the uninvolved ovary is often atrophic.

Microscopically, masses of lipide-laden, plump, spindle-shaped theca cells alternate with zones of hyalinization in a picture that may be confusingly similar to that of a fatty fibroma. Escape from this diagnostic dilemma is often afforded by the finding of clusters of granulosa cells in the truly functioning growths. Although practically all theca cell tumors are benign, their treatment in older patients has been of necessity somewhat

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From the Section of Surgical Pathology, Mayo Clinic, Rochester, Minnesota. This is the twenty-sixth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

radical because of the coexistent uterine pathologic changes.

Sertoli Cell Tumors.—Sertoli's cells are reputed to elaborate estrogen, and clinically feminizing tumors made up of these elements have been humans. Some investigators maintain that a human ovarian counterpart exists in the form of described in the testes of dogs and, rarely, also of certain rarely reported instances of tubular granulosa cell tumors.

Arrhenoblastomas.—Arising either from testicular remnants in the ovarian hilus or through an overgrowth of comparable elements in a teratoma, this masculinizing neoplasm induces amenorrhea associated with such conditions as infertility, hirsutism, changes in voice and atrophy of the breasts. Of the approximately 100 cases reported in the literature, the few in which necessary studies have been performed generally have exhibited decreased values for urinary estrogen and increased values for ketosteroids. Arrhenoblastomas, which are of a small average size, are characteristically solitary, solid, lobulated and encapsulated. Their local removal in young women has been followed by resumption of menses, pregnancy and parturition, and regression of hairiness and changes in the voice. Prolonged follow-up studies, however, reveal a 50 per cent incidence of recurrence and metastasis, indicating that such conservative treatment has not served the best interests of the patients.

Microscopically, many variations depart from the "average" picture of tubules resembling those of the testis, with intervening clusters of Leydig's cells. Sertoli's cells lining the tubules may secrete estrogen that neutralizes the male sex hormone of the interstitial (Leydig) cells. Such a valid (?) explanation has been offered to explain the absence of clinical masculinization sometimes noted in patients who have tumors that appear mature and differentiated. Interstitial cells are more abundant in the less differentiated growths, which usually produce the most pronounced degree of masculinization. Indeed, a few cases have been reported in which ovarian hilar proliferations of Leydiglike cells have produced masculinization even in the absence of any lesion deserving the designation of tumor.

Masculinovoblastomas.—Twenty case reports comprise the literature on this interesting growth, in which the pathologic features of an ectopic adrenal cortical carcinoma are linked with the clinical picture of Cushing's syndrome. The tumors presumably arise from adrenal cortical tissue sequestered within the ovarian hilus.

These growths are large, solid, friable carcinomas that fortunately are usually well encapsulated. Microscopically, they feature clusters of lipide-laden cells with sharp epitheliumlike outlines disposed in alveolar and trabecular patterns. Evidences of cellular anaplasia and pronounced pleomorphism usually have not been reflected in metastasizing aggressiveness, and many patients afflicted with these tumors have achieved permanent cure after simple oöphorectomy.

Dysgerminomas.—Comprising about 1 per cent of ovarian neoplasms, the dysgerminoma has been captioned a "neutralizing" tumor principally because of its frequent occurrence during prepubertal years and its occasional association with a state of pseudohermaphrodism. However, its removal does not alter the habitus of such patients. Since a number of these growths have been discovered during pregnancy, one wonders about the propriety of continuing to classify them as "neutralizing." Bilateral in a third of cases, dysgerminomas as a rule are large, soft, hemorrhagic and necrotic. Microscopically, the picture is exactly similar to that of seminoma testis, which likewise arises from primitive sex cells.

A discouraging 30 per cent five-year surgical survival rate indicates all too clearly that the body curves of the patients rather than the mortality curves in the past have fostered a somewhat conservative assault on this serious form of ovarian carcinoma.

Struma Ovarii.—As a producer of ovarian hormones, struma ovarii certainly stands on the side lines. However, this teratomatous tumor of thyroid tissue sometimes elaborates enough thyroxin to justify its inclusion in any group of functioning ovarian tumors. Basically, the functioning 5 per cent of such lesions are toxic adenomas rather than exophthalmic goiters and the patient's thyroid gland does not share the metabolic turmoil of its ovarian auxiliary. As a consequence, operations on the neck have not made these patients better risks for oöphorectomy. Peculiarly prone to undergo torsion of their pedicles, these tumors frequently induce ascites. Malignant changes eventually ensue in 5 per cent.

President's Letter

OUR 102nd MEETING

It may not be too generally known, but it is an accepted fact that the medical profession spends more of its time at postgraduate education than any other profession or guild. This is as it should be. The never-ending search for methods of cure and prevention is inherent in our philosophy of public service. Advances in scientific knowledge and improvement of techniques reveal themselves annually in improved mortality rates, decreased morbidity, and increased longevity. This dissemination of knowledge and the interchange of ideas takes several forms, best exemplified by hospital staff meetings, pathological conferences, county and state medical meetings, and specialty organizations, and the voluminous medical literature.

Our State Association assumes its rightful part in this postgraduate medical education program by attempting annually to present a program at its annual meeting which will be of interest and value to the profession of the state.

The value of our annual meeting is enhanced by the contributions made by our commercial exhibitors. They demonstrate improved technical instruments, inform us of improved therapeutic agents, make available for demonstration and inspection medical texts of all varieties. The value of their contribution cannot be overestimated.

This year your Committee on Scientific Assembly is introducing an innovation to our State Association meeting. Two and a half hours a day will be given to closed circuit television. This, of necessity, will decrease the number of formal papers that can be given at the meeting, and your officers and committee membrs are awaiting with a great deal of interest your reactions to such a program.

This year also we have a distinguished roster of guest speakers, and we welcome their contribution to our program. In addition, we have shifted the round tables to all three days of the meeting, and the topics will be discussed under competent moderators. I urge you all to make reservations for the round-table discussions as early as possible so that you will not be disappointed in attending the discussion of your choice. Programs and reservation cards will be sent to members around May 1.

Also, this year is the centennial of the Hennepin County Medical Society and we are very happy to join with the society in celebrating the occasion. Wouldn't it be interesting if by some magic means the founders of the Hennepin County Medical Society could attend our annual meeting? Their amazement can only be imagined, but we must remember that it is through the efforts of these original founders that our success as a county and a state organization was made possible. To them we owe our undying gratitude.

Your Committee on Local Arrangements, your Committee on Scientific Assembly, and all your state officers are eager to greet you and serve you at our annual meeting. We are also anxiously awaiting your reactions to the innovations in the program and hope for your constructive criticism.

Arnold O. Swenson

President, Minnesota State Medical Association

Editorial

JOHN F. BRIGGS, M.D.
HENRY G. MOEHRING, M.D.
ARTHUR H. WELLS, M.D.

THE STATUS OF NEUROLOGIC RESEARCH

In the 19th and early 20th centuries, neurological research was principally devoted to the elucidation of anatomical pathways, the study of pathologic alterations aided by special staining techniques, and the careful clinical description and categorization of neurological disease. Advancements in research techniques and the revision of clinicians' orientation towards neurological disease have resulted in drastic changes in neurological research in recent years.

The development of electroencephalographic and electrocortiographic techniques as well as the application of the cathode ray oscilloscope and of strichnine neuronography in human and animal experiments has provided physiological information unobtainable by older techniques. The adaptation of the nitrous oxide technique by Schmidt, Kety, and other investigators has clarified many speculations concerning cerebral circulation. The physiological functioning of the nervous system has now been demonstrated to be intimately concerned with a variety of enzymatic and other chemical reactions. The clinical applications of neurochemistry was strikingly demonstrated by studies such as those indicating Vitamin B_1 (thiamine-diphosphate) to be essential to the aerobic metabolism of nervous and other tissues. Failure in aerobic metabolism brought about by Vitamin B_1 deficiency results in the clinical symptomatology of polyneuritis as well as in central nervous system manifestations. Other recently developed techniques include tissue culture and phase microscopy which have contributed toward the improved diagnosis of poliomyelitis and other virus infections and have accelerated the development of anti-viral vaccines.

Clinical research methods have also undergone marked changes in the past few years. A dynamic approach to nervous system disease has replaced the purely descriptive and categorizing type of neurology. The advances in anatomical, physiological and chemical knowledge are being employed to improve the diagnosis and therapy of neurological disease. Newer techniques such as arteriography and other neuroradiological procedures have produced marked improvement in the neurological diagnosis. Among the most dramatic contributions of clinical neurologic research is the development of rehabilitation in the neurologically disabled patient.

The emphasis on newer methods in basic neurologic research and the accentuation of dynamic clinical neurology has contributed significantly to a greater understanding of neural mechanisms and a sounder approach to neurologic diagnosis and therapy.

MAYNARD M. COHEN, M.D.

MINNESOTA POLL

The *Minneapolis Sunday Tribune's* Minnesota Poll, published January 23, 1955, concerned itself with attitudes toward physicians and showed that in Minnesota: two-thirds of the people considered doctors' fees reasonable; more than four out of five people are satisfied that doctors "take a personal interest in their patients and their troubles"; 87 per cent believe doctors are better trained nowadays than they've ever been, and 68 per cent believe people in this country would be worse off if we had federal control of doctors.

This is a public relations testimonial of which physicians are proud. But, recall the repeated sage comment of the late Dr. J. C. Litzenberg, "*Any* physician can make the right diagnosis and undertake the correct treatment in 80 per cent of the patients who come to him. But what we want to train here at the University of Minnesota are physicians who can also be right in that other 20 per cent." Then recall also the anonymous research worker who contended that unless the proof of a hypothesis was so forthright that it fairly hit you in the face, it wasn't convincing enough to let you accept the hypothesis as correct. From this Minnesota Poll we have, in a general sort of way, that dissenting 20 per cent; e.g., the poll reports that 32 per cent of the people aged sixty and older, 25 per cent of the labor union members, 24 per cent of the men, 22 per cent of the Democratic-Farmer-Labor voters, and 20 per cent of the independent voters think that the country would be better off if medical care were controlled by the federal government and doctors were under federal regulation.

Apparently, Minnesota physicians, however proud they have a right to be about having pleased from two-thirds to seven-eighths of their patients, must offer still more to please the unconvinced 20 per cent. We could just assume that they are the incorrigibles, no matter how you look at them, but somehow, we're sure, the Litzenbergian principle will prevail and we'll continue to try to render satisfying service to 100 per cent of our patients.

H.G.M.

HOW A LOCAL BANKER MAY SERVE THE PHYSICIAN

We believe that a local bank serves the physician by making available to him a safe storage place for money; by providing personal and business checking accounts; by advancing long-term financing for his building and equipment; and by granting short-term financing for temporary needs. These are the major items of service which are in turn supplemented by other less routine services furnished by the banker who is his financial consultant.

The first item mentioned is, of course, one of the basic functions of banking, and within its structure, a convenient safe method of storing funds is provided. These funds are available immediately for the use of the physician, and in the meantime can be used by the bank in the form of loans, to develop and strengthen the trade territory in which both the physician and the bank operate.

Many physicians are noted for their lack of interest in the maintenance of good records. The bank provides a checking account service which is invaluable in this respect to provide legal receipts for bills paid, and to provide good evidence for income tax records. This service is important both from the standpoint of the professional or office account, as well as for the personal account, and in many cases is the only record maintained in the latter area.

In many communities, the physician is expected to maintain complete and extensive office facilities to supplement the hospitals in his town. These facilities may take the form of a modern clinic for several doctors, or doctors and dentists combined, and represents a considerable investment of money. Long-term financing is required to enable these facilities to be constructed and paid for on an orderly basis, and the banker helps to provide these funds as well as aids in making decisions regarding the size of the project and the proper location for it. Even where the office is maintained in rented quarters, the physician's investment in equipment and supplies can be quite large and will require relatively long-term financing. Short-term financing for other temporary needs is readily available at commercial banks for the purpose of financing accounts receivable and unusual temporary expense items and supplies.

After the physician is well established in his community and has acquired and paid for his "plant and equipment," the banker is often called upon to serve in less routine ways. The physician can properly look to him for assistance in investing his funds. The banker can discuss the field of investment generally, and suggest to the doctor the type of investment he feels might be best suited to his needs, pointing out the safety features, the rate of return, and call attention to the speculative nature, if any, of the investment under consideration. Ordinarily, too, the banker is an excellent source of information regarding community projects of various kinds that professional people are called upon to support, and by merely placing a telephone call to the banker, reliable information of this kind can be obtained.

In addition, we feel that the banker and the doctor share common interests in two very important community matters. Both are interested in the over-all welfare of the people in their trade area, and the bank, by careful and judicious use of credit, builds its community in terms of improvement, of employment potential, by attracting new businesses and aiding established firms in their growth, and therefore develops a larger clientele for the work of the doctor. In recent years, more and more families are coming to the bank for family credit and the banker helps these people stabilize their personal affairs, which in turn eases the collection problem for the doctor. It is also our feeling that both the medical profession and the banking profession are among the strongest proponents of the capitalistic system, both believing that socialization of their professions would in the long run not operate to the advantage of most of the people. Both of these professions have a responsibility to make these views known within their community, and it would therefore seem that the banker and doctor can function as a team, not only strengthening each other in their ability to serve, but in turn strengthening the community they both serve, and from which they draw their livelihood.

Walter V. Dorle
President, Northwestern State Bank
Saint Paul, Minnesota

HISTOPLASMOSIS IN MINNESOTA

Waring, in Minnesota Medicine, August, 1954, presented a fine article on non-tuberculous cavities of the lung, which summarizes the picture of the fungus infections, coccidioidomycosis, histoplasmosis, and actinomycosis; these three represent the vast majority of fungus infections of the lung. Waring illustrates the similarities and dissimilarities between these diseases and tuberculosis, and emphasizes many practical points in differential diagnosis. This becomes increasingly important to physicians in Minnesota because of new evidence of the existence of histoplasmosis in the state on a scale hitherto unsuspected.

The fungus, Histoplasma capsulatum, is widely distributed in nature, mainly but not exclusively in the Mississippi Valley. Following human infection, the skin test with histoplasmin converts from negative to positive within one to two weeks after the onset of illness, and remains positive for years. This skin test may be used to measure the extent of human infection with the fungus in any area. There is no evidence of person-to-person communication of infection. Age, as well as residence, will affect expected histoplasmin sensitivity, since children may have a much lower frequency of reactions than the adult population.

Using such survey techniques, Furcolow et al have shown, in the Cincinnati, Ohio, area, that 15 per cent of children five years old reacted positively to histoplasmin, the rate rising steadily to reach a high of 80 per cent positive at age eighteen years. This rate curve is similar to that of children in Kansas City, but a little lower. The same children reacted positively to tuberculin in less than 1 per cent at age five, rising to about 5 per cent at eighteen.

In May, 1954, tuberculin and histoplasmin tests were given to 5,487 school children in the grade and high schools in Wright County, in central Minnesota, and to 199 adults connected with these schools. Of the children, sixty-five (1.2 per cent) reacted positively to tuberculin and 404 (7.3 per cent) reacted positively to histoplasmin. Of the adults, forty-five (22.6 per cent) were positive to tuberculin, and thirty-two (15 per cent) were positive to histoplasmin. Further analysis of the survey will be available soon.

Histoplasmosis has been recognized as occurring in Minnesota in epidemic form on at least two occasions: in 1948 in the Detroit Lakes area, and in 1949 and 1953 in Wright County. Waring points out that the lungs are involved in about 50 per cent of cases reported in the literature, and that about 20 per cent of reported cases are in infants.

It seems apparent that histoplasmosis infection may not be as infrequent in Minnesota as hitherto believed, and that the disease occasionally occurs in localized epidemics. Because of frequent pulmonary involvement, the disease must be considered in the diagnosis of respiratory illness, and it may be the cause of unexplained illness in children.

D. S. Fleming, M.D.

ACCIDENTS, A KILLER OF OLD AND YOUNG

Readers of Minnesota Medicine have had several topics of accidents brought to their attention during the past several months in a series of articles focused on accidents as the prime killer of children. In this issue still another is presented: *"Choking Accidents."*

Are you teaching young parents the dangers to which their children are exposed? Are you using the new safety vaccine?

T.C.C.

Evidence on all sides shows that the enduring health programs are those the people have understood and those they have had a hand in developing.—Hugh R. Leavell, M.D., Dr. P. H., *American Journal of Public Health,* November, 1954.

The Dean's Page

THE PROCUREMENT OF ANIMALS FOR RESEARCH

The 1949 Legislature passed an act "to promote scientific research and instruction in animal and public health by making available to educational and scientific institutions, unclaimed and unredeemed animals impounded by public authority in animal pounds" and "to provide licenses therefor and penalties for violations thereof." The title of this act clearly indicates the intent of the 1949 Legislature to aid educational and scientific institutions in the procurement of animals for teaching and research purposes for the benefit of both human beings and animals.

Primarily, through the Antivivisection Society and similar groups, resistance to the provisions of this law was encountered. It became obvious that several points in the law needed strengthening. First, the definition of "animal pound" was not sufficiently explicit to prevent legal arguments. This controversy centered around the question of whether or not a privately owned kennel impounding stray animals through a contract with a municipality was subject to this law. The second difficulty with the original act was the lack of a specific procedure to be followed in the event of an alleged violation of the law.

The 1955 Legislature is to be commended for correcting the weak points in the original act of 1949. Amendments were passed which clearly stipulate:

> "Any public or private agency, person, society or corporation having custody of animals which are seized under authority of the State or any political subdivision of the State must make available to licensed institutions all animals unclaimed and unredeemed by their owners . . ."

One of the most effective amendments was as follows:

> "Any establishment which fails to comply with these provisions shall become immediately ineligible for any further public funds from any county or municipality."

Rapid enforcement procedures and an arbitration agency were added in the following amendment:

> "Upon receipt of a sworn statement by an authorized officer or employe of any institution licensed hereunder of non-compliance by an establishment with these provisions it shall be unlawful for the treasurer of any municipality or other political subdivision of the State to pay any public funds to such establishment until the complainant withdraws its statement of non-compliance or until the State Livestock Sanitary Board shall either determine that the complaint of non-compliance was without foundation or that the establishment has given adequate assurance of future compliance, and the treasurer of such municipality or other political subdivision has been notified of such determination in writing. If it appears upon the complaint of any person that any officer, agent, or employe of such establishment is violating or failing to carry out the provisions of this section, the Attorney General or County Attorney of the county in which the establishment is located, in addition to any other remedies, may bring an action in the name of the State of Minnesota against any such establishment, officer, agent, or employe thereof to enjoin compliance with this section."

Chapter 195 of the session of the laws of the 1949 Legislature, as amended by the 1955 Legislature, stands as a milestone in the course of progress in experimental medicine. It clearly indicates strong public support for scientific progress. The members of the Legislature who made this law possible are to be congratulated for a job well done!

Harold S. Diehl
Dean of Medical Sciences
University of Minnesota

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

HOOVER COMMISSION REPORTS ON MEDICAL SERVICES

The medical services report of the Hoover Commission (on Organization of the Executive Branch of the Government) was sent to Congress recently.

The more important recommendations are: (1) Establish a Federal Health Council; (2) Provide contributory health insurance for military dependents and for personnel of the Coast Guard, the Public Health Service and the Coast and Geodetic Survey, and for dependents of the latter three groups; (3) Regionalize military medical services, with one service responsible for each region; (4) Tighten up ability-to-pay requirements in VA, and codify VA laws and regulations; (5) Virtually wipe out the present Public Health Service hospital and medical care program.

The Medical Task Force of the Hoover Commission made studies which report that United States spending for medical programs and compensation totals $4.1 billion a year and that the federal government is responsible in whole or in part for the medical care of 30 million people. These facts are reported in a recent issue of the American Medical Association Washington Letter.

The Letter states:

> "It is expected that legislation shortly will be introduced to carry out the recommendations. In a legislative sense, the Commission and Task Force suggestions carry only what weight the Congress wants to give them. The major medical recommendation of the first Hoover Commission (1949) was for a united medical administration to take over most of the medical responsibilities of the federal government. Hearings were held in Congress on this recommendation, but it was not put into effect."

AMA COMMENTS ON HOOVER REPORT

Recommendations of the Hoover Commission on non-service-connection disability regulations were surprising to the American Medical Association, according to a recent Secretary's Letter. The Letter states:

> "It was surprising to many of us . . ., and certainly to the nation's taxpayers, that the Hoover Commission, in announcing its report on federal medical services on Monday, February 28, junked the major recommendation of its own 16-member Medical Task Force. At the last minute of deliberations, it substituted instead proposals which will expand instead of curtail government medical care for veterans with non-service-connected disabilities."

The Letter goes on to say that in its study report to the commission, the Medical Task Force recommended that veterans with non-service-connected disabilities receive hospital care if the need for such disability is established within three years following discharge from active duty. Members estimated that the number of potential patients with non-service-connected disabilities would be reduced from 17.5 million to about 3 million, with an estimated annual savings of $150,000,000.

"The commission, however," states the Letter, "chose to reject the cut off provision and recommended that this service be reduced through a closer financial screening of applicants. This recommendation, as we all know, already has been tried and proved to be ineffective."

Inconsistent Views

It is noted that the commission has made recommendations which were not in keeping with recommendations of its Medical Task Force. The AMA comments:

> "While the Medical Task Force did recommend non-service-connected out-patient care, it was to be within the framework of the three-year cut off date. The commission, in eliminating the three-year eligibility clause, chose to retain the provision of out-patient service, which in the past has been available only to veterans with service-connected disabilities. This out-patient provision would actually skyrocket the cost of veterans' medical care."

In conclusion, the AMA states that the Commission has reverted from its original objective:

> "To remove the three-year time limit and, at the same time, furnish the non-service-disabled veteran out-

patient care in veterans' hospitals is certainly not in keeping with the commission's objective of trying to eliminate wasteful government spending and the unnecessary intrusion of the federal government in private affairs."

SURPLUS PROPERTY SOUGHT FOR HEALTH FACILITIES

According to a House Government Operations Subcommittee, a new Defense Department regulation may be preventing distribution of surplus property to health and educational institutions. The subcommittee, under direction of John W. McCormack (D., Mass.) is investigating the charges. The regulation requires that certain salable surplus items in the military services be placed in a "capital account," to be sold at prices said to average less than 5 per cent of their acquisition cost. A recent AMA Washington Letter reports that a system for free distribution to health and educational institutions has been in existence for some years, under supervision of the Department of Health, Education and Welfare.

The subcommittee had before it a bill which would amend the present law to:

> "1. Require all departments to release surpluses if the states and other non-profit agencies want them, and if HEW approves.
>
> "2. Remove after one year any restrictions on resale or disposal of surplus property previously acquired, and permit immediate passage of title in future acquisitions.
>
> "3. Allow for unrestricted transfer of surplus property ownership among federal and state agencies."

RURAL HEALTH LEADERS GATHER

Community efforts to bring better health to rural areas was the point of emphasis at the recent 10th National Rural Health Conference in Milwaukee. About 500 physicians, farm organization officials, agricultural extension workers, farmers and others met to discuss mutual rural health problems and projects.

Subjects of discussion included farm and home safety, family responsibility for health, using available health and medical care resources, and how the job is done. Dr. F. S. Crockett, Lafayette, Indiana, chairman of the AMA's Council on Rural Health, said:

> "Our first ten years in this important endeavor of creating more healthful living for people in rural areas has produced a remarkable unity of purpose between the medical profession and farm people. Medicine is no longer a job for the doctor alone—it is a community problem, too."

One of the striking aspects of the conference was its deep personal basis—the feeling that the participants were not talking about "problems" or "goals" or "programs" but about people, the rural people whose work is the mainstay of the entire nation's well-being. According to an AMA news release on the meeting, "The accent was first of all on individuals, then families, then communities. The individual man's responsibilities and tasks were foremost, while the family unit was stressed as the starting-point for cooperative effort on other levels."

Looking Both Ways

Theme of the meeting was "Looking Both Ways"—to past achievements and future goals. Dr. Leonard W. Larson, Bismarck, N. D., AMA Trustee, summing up the meeting, noted that doctors have been in the minority at the ten national conferences and that "their viewpoint, although freely expressed, has not dominated the discussions."

"This is as it should be," he continued, "for the people know best what is available and what is needed to improve the health condition of the farmer. The doctor can tell them how to utilize existing facilities and how, from the medical viewpoint, he can be of assistance in the attainment of their objective."

"JUST KEEPING UP" TAKES TIME

The average American physician, who puts in a sixty-hour work week, spends about ten hours a week just keeping up his education, a recent American Medical Association survey shows.

And, even with this exacting schedule of hours, most physicians surveyed said they wished it were possible to spend much more time keeping up to date with medical progress.

Dr. Douglas D. Vollan, assistant secretary of the Council on Medical Education and Hospitals, reported on the survey of the physician's career-long education in a recent issue of the AMA Journal. He stated:

> "Every year, every month, and every day brings new changes in the panorama of knowledge of human biology,

human disease, and related scientific fields. With this knowledge comes new skill in the prevention, diagnosis, and treatment of the ills that plague mankind. . . . What may have been acceptable treatment for one condition 20 years ago might now be considered less than worthless."

Therefore, it is "absolutely essential" for the physician to continue his education as long as he practices medicine. The Council survey found out from about 5,000 physicians and numerous postgradate education facilities just how it's done.

According to the survey, the surveyed physicians said they spend a yearly average of 667 hours (about 83 eight-hour days) in the five general fields of learning after college—reading, professional contacts with other physicians, attending hospital staff meetings, going to medical society meetings, and taking formal postgraduate courses.

Reading Important

Most physicians said reading is the most important way to learn, and wished they had more time for it. A third of these 667 hours are spent in reading. Another third of the time is in professional contacts with other physicians, since "bedside consultation" is probably one of the best ways to learn. Physicians spend from one day to as much as three months a year taking courses, which they ranked second in educational importance. Those surveyed said they would like to spend 200 per cent more time in postgraduate courses.

Dr. Vollan, in summing up the report, quoted a comparison of the American physician "to a shiny new automobile, the product of a very large initial investment but one that requires constant care, repair, and refueling in order to do the job for which it is intended."

HOBBY SAYS AMA WILL ACCEPT REINSURANCE

Secretary to the Department of Health, Education and Welfare, Mrs. Oveta Culp Hobby, believes the American Medical Association will show a turnabout and accept the government's reinsurance plan. Testifying in support of the President's national health program recently, Mrs. Hobby deprecated suspicions that reinsurance is a leadup to socialized medicine.

In response to questioning, Mrs. Hobby definitely indicated that she expects the AMA will not oppose reinsurance. One of the strongest deterrents to passage last year was AMA's claim that it was an opening wedge to socialized medicine. "I think when you come to question AMA," Mrs. Hobby stated, "they will not say it is an opening wedge." Mrs. Hobby continued:

> "I thought that old socialized medicine ghost was dead. Reinsurance is just an attempt to help men and their families help themselves. I don't believe people want socialized medicine, but they do want a way to pay medical bills. We must find a way by which they can pay those bills. If we don't do it by a voluntary way, then it must be done by subsidy or by compulsory health insurance."

This proposal is still being studied by the AMA and no commitment has yet been made on approval or opposition.

PRACTICAL COURSE FOR DOCTORS-TO-BE

What advantages do general practitioners have over specialists? How does a young doctor start his practice? What are some of the early problems he faces, and how can he solve them to his own and the community's advantage?

These are some of the questions which are discussed in a course on "The Practice of Medicine," offered at the University of Colorado School of Medicine. This course, which is intended to give medical students practical advice on the situations they will face at the start of their careers, is described by Dr. Gertrud Weiss, associate professor and acting head of the department of preventive medicine at the school, in the March issue of *The Journal of Medical Education.*

The study, which is offered in the junior year, is based on case history presentations, which are selected in advance. The students have time to study the cases, which cover such problems as the choice of locality and office space, relations with other doctors, fee setting, religion and patient attitude, and the business end of medical practice.

An appropriate resource person from the community is invited to appear at the scheduled seminars to discuss the particular case with small groups of students. Participation by the students is encouraged, and the lecturer often asks the students how they would solve the problem under discussion *before* he presents his lecture.

At present, a question which perturbs many medical students is the decision to enter general practice or to specialize. This problem is handled by a panel discussion between a general practitioner, an obstetrician, a pediatrician and an internist. Also, two general practitioners from small communities describe their practice and the situations they face. Dr. Weiss points out that this was the first time the students had met and been taught by general practitioners, and yet a poll taken at the end of the year showed that almost half of the junior class intended to enter general practice.

Public Health

STATE DENTAL HEALTH PROGRAM HAS MANY PHASES

Dental health has been a major concern of the Minnesota Department of Health for the past twenty years. It is recognized today as a legitimate public health concern because of the universal scope of the problem and the increasing effectiveness of preventive measures. In line with the philosophy that practicing dentists should have a basic knowledge of dental public health problems and programs, a series of lectures in this field was started in the School of Public Health, University of Minnesota, during the winter quarter of 1955, and will continue during winter and spring quarters each year.

The health department's dental health staff is much too limited to meet the numerous requests for help that come from all parts of the state. Hence, the plan usually followed is to give aid in setting up local programs with the co-operation of the dentists, public health nurses, school authorities, and citizen groups in communities requesting help, and then to turn over operation of the program as far as possible to the local people.

Outstanding dental health programs now in operation under the sponsorship of the Minnesota Department of Health include the following:

Topical Fluoride.—Three entire counties, comprising a total of 12 communities, plus nine other individual communities, have been aided in carrying out programs involving the topical application of fluoride to the teeth of children in grades 2, 5 and 8. Two new programs in this area are already planned for the fall of 1955. These topical fluoride programs provide field experience for senior dental hygiene students from the School of Public Health, University of Minnesota. The students assist professional staff members of the Section of Dental Health and the local dentists who give the fluoride treatments. Topical application of fluoride to children's teeth has been shown to reduce dental caries incidence on an average of 40 per cent.

Fluoridation of Community Water Supplies.—Projects in this area have been aided and promoted in 50 Minnesota communities. This program has been approved by the American Medical Association, American Public Health Association, and many other responsible professional groups. It has been demonstrated that continuous use of properly fluoridated drinking water by children during the period of tooth development reduces dental caries on an average of 65 per cent.

Research.—The Section of Dental Health is currently working on two research projects. The Askov demonstration was planned to demonstrate how much improvement can be effected in children's dental health when all known and available protective methods are used. This demonstration is now in its seventh year. Annual reports are published and are available on request. The other research project is being conducted in Mound, to determine the comparative value of sodium silicofluoride and two-per-cent sodium fluoride in controlling dental caries. A third study—on the effects of brushing the teeth with stannous fluoride —is planned to start in Bloomington schools next fall.

School Programs.—The Section of Dental Health sponsors and assists in a statewide dental health education program in all schools that wish to participate. During 1954, this program included 404,925 pupils in 3,101 public and parochial schools. (In the large cities, school boards operate their own school dental health programs.) Eighty-one of Minnesota's 87 counties were represented in the statewide program sponsored by the Department of Health.

Community Programs.—Each year the Section of Dental Health works closely with the Minnesota State Dental Association in planning and promoting educational programs for National Children's Dental Health day. These programs include radio and television broadcasts, exhibits, film showings, talks by dentists, and the distribution of Dental health literature.

In addition to these activities, staff members of the Section of Dental Health give many talks to P.T.A. groups, civic organizations, and schools, and participate in a number of conferences, workshops. short courses, and university classes. Last December, the Section of Dental Health participated in the first joint conference held in Minnesota for school superintendents, physicians. and dentists. Dental health conferences have been held for youth leaders in six Minnesota counties, in collaboration with the State Dental Association. Recently a county-wide educational program was carried out in Pope county, when every school in the county was visited by a team of dental health educators. Section of Dental Health personnel assisted at a cavity detection x-ray exhibit at the Minnesota State Fair last year.

William A. Jordan, D.D.S., M.P.H.
Chief, Section of Dental Health
Minnesota Department of Health

Minnesota State Medical Association

102nd Annual Meeting

Minneapolis, Minnesota—May 23, 24 and 25, 1955

Preliminary Program

BUSINESS SESSIONS

Hotel Radisson

SATURDAY, MAY 21

2:00 P.M.—Council ....Admiral Room
6:00 P.M.—Council ....Admiral Room

SUNDAY, MAY 22

8:30 A.M.—Council ....Admiral Room
10:00 A.M.—Reference Committees ...Mezzanine Floor
2:00 P.M.—House of Delegates ....Gold Room
6:00 P.M.—Council ....Admiral Room
8:00 P.M.—House of Delegates ....Gold Room

MONDAY, MAY 23

8:30 A.M.—Council ....Admiral Room
12:15 P.M.—House of Delegates ....Gold Room

TUESDAY, MAY 24

8:30 A.M.—Council ....Admiral Room
8:30 A.M.—Committee Breakfasts
Rooms to be assigned

WEDNESDAY, MAY 25

8:30 A.M.—Council ....Admiral Room
8:30 A.M.—Committee Breakfasts
Rooms to be assigned

SOCIAL EVENTS

SUNDAY, MAY 22

6:30 P.M.—SPORTS EVENTS DINNER, Minneapolis Golf Club. All participants in golf, archery and skeet and trap shooting are invited to attend with their wives, a joint sports events dinner after the Sunday afternoon sports events.

MONDAY, MAY 23

Luncheons

12:15 P.M.—American Medical Women's Association, Inc., Minnesota Chapter. All women physicians are invited to attend.
12:30 P.M.—Minnesota Medical Foundation, Junior Ballroom, Hotel Radisson.
12:30 P.M.—Minnesota Society of Clinical Pathologists. Curtis Hotel.
12:30 P.M.—Minnesota Chapter, American College of Chest Physicians (place to be announced).

Dinners

6:00 P.M.—Minnesota Academy of General Practice. Ballroom, Hotel Radisson.
6:00 P.M.—Reunion, Class of 1905. Hotel Radisson.
6:30 P.M.—Minnesota Society of Clinical Pathologists. Curtis Hotel.

Open House

9:00 P.M.—Minnesota State Medical Association. Ballroom, Hotel Radisson.

TUESDAY, MAY 24

Dinner

7:00 P.M.—Minnesota State Medical Association, Annual Banquet, Ballroom, Hotel Radisson.

ROUND TABLES

Hotel Radisson—12:15 P.M.

MONDAY, MAY 23

Newer Insulins ....Moses Barron, Minneapolis
Office Treatment of Foot Complaints
W. J. McDonald, Crookston
Diverticulitis of the Colon..David P. Anderson, Austin
The Injured Nose ....Jerome A. Hilger, St. Paul
Oxygen Therapy in the Premature Infant
R. P. Buckley, Duluth
Robert W. Hollenhorst, Rochester
Disorders Simulating Coronary Disease
Asher A. White, Minneapolis
The Changing Status of Cesarean Section
C. O. McCormick, Indianapolis, Ind.

TUESDAY, MAY 24

Office Gynecology ......Edward A. Banner, Rochester
Emergency Treatment of Head Injuries
Wallace P. Ritchie, St. Paul
Blood Transfusions in Therapy
T. H. Seldon, Rochester
Eczema in Childhood
Albert V. Stoesser, Minneapolis
Vaginal Procidentia and Stress and Continence
J. H. Pratt, Rochester
The University In-Patient Psychiatric Service for Children..Reynold A. Jensen, University of Minnesota
Defibrination Syndrome
Harold A. Oh, Royal Oak, Michigan

WEDNESDAY, MAY 25

Spinal Puncture Headache......L. R. Gowan, Duluth
Anti-hypertensive Drugs
Arthur C. Kerkhof, Minneapolis
Problems of Sudden Death
Arthur C. Aufderheide, Duluth
Treatment of Birthmarks ....John F. Schmid, Duluth
Urethral Strictures in Young Females
Edward J. Richardson, St. Paul
Edward L. Strem, St. Paul
Convulsive States ......Joseph A. Resch, Minneapolis
Pregnancy Associated with Heart Disease
John E. Faber, Rochester
Fulminating Eclampsia
Jack A. Pritchard, Cleveland, Ohio

GENERAL SESSION

Minneapolis Auditorium

MONDAY, MAY 23

Morning Session

A.M.

8:30 Visit Scientific and Technical Exhibits Auditorium

9:00 Closed Circuit Color Television ....Auditorium
Surgical Procedures—General Surgery

Intermission

10:15 Visit Scientific and Technical Exhibits Auditorium

11:00 RUSSELL D. CARMAN MEMORIAL LECTURE
"The Position of Radiology in Cardiovascular Diagnosis"
Fred J. Hodges, Ann Arbor, Professor and Chairman Department of Radiology, University of Michigan

11:30 MINNESOTA HEART ASSOCIATION LECTURE
"Diagnostic Cardiac Silhouettes and Electrocardiograms"
Arthur Master, New York, Associate Clinical Professor of Medicine, Columbia University

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheons

Afternoon Session

1:30 Visit Scientific and Technical Exhibits Auditorium

2:00 Closed Circuit Color Television.....Auditorium
Clinical Procedures—Orthopedics

Intermission

3:15 Visit Scientific and Technical Exhibits Auditorium

4:00 ARTHUR H. SANFORD MEMORIAL LECTURE
"Enigmas in Lymphoma: The Reticulum Cell and Mycosis Fungoides"
Edward A. Gall, Cincinnati, Ohio Professor of Pathology, College of Medicine, University of Cincinnati

4:30 MINNESOTA DIVISION, AMERICAN CANCER SOCIETY LECTURE
"Causal and Preventive Aspects of Environmental Cancer"
W. C. Hueper, Chief, Environmental Cancer Section, National Cancer Institute, Bethesda, Maryland

5:15 Visit Scientific and Technical Exhibits Auditorium

6:00 Special Dinners

9:00 Open House ....Hotel Radisson

GENERAL SESSION

Minneapolis Auditorium

TUESDAY, MAY 24

Morning Session

A.M.

8:30 Visit Scientific and Technical Exhibits Auditorium

9:00 Closed Circuit Color Television ....Auditorium
Surgical Procedures—Obstetrics and Gynecology

Intermission

10:15 Visit Scientific and Technical Exhibits Auditorium

11:00 NORTHWESTERN PEDIATRIC SOCIETY LECTURE
"The Diabetic Child"
John A. Anderson, Professor and Head, Department of Pediatrics, University of Minnesota

11:30 MINNESOTA MEDICAL FOUNDATION LECTURE
"Present-Day Concepts of Diabetes Mellitus"
Arnold Lazarow, Professor and Head, Department of Anatomy, University of Minnesota

P.M.

12:15 Round-Table Luncheons

Afternoon Session

1:30 Visit Scientific and Technical Exhibits Auditorium

2:00 Closed Circuit Color Television.....Auditorium
Clinical Procedures—Medicine

Intermission

3:15 Visit Scientific and Technical Exhibits Auditorium

4:00 OBSTETRICAL SYMPOSIUM....Auditorium
Moderator—James J. Swendson, St. Paul
"Current Report on the Minnesota Maternal Mortality Study"
Alex Barno, Minneapolis
"Obstetric Anesthesiology"
John Osborn, Rochester
"Foibles and Pitfalls in Obstetric Hemorrhage"
Harold A. Ott, Royal Oak, Michigan

5:15 Visit Scientific and Technical Exhibits Auditorium

Evening Entertainment

7:00 ANNUAL BANQUET
Grand Ballroom, Hotel Radisson

GENERAL SESSION

Minneapolis Auditorium

WEDNESDAY, MAY 25

Morning Session

A.M.

8:30 Visit Scientific and Technical Exhibits
Auditorium

9:00 Closed Circuit Color Television ....Auditorium
Surgical Procedures—Vascular Disease

Intermission

10:15 Visit Scientific and Technical Exhibits
Auditorium

11:00 "Management of Severe Hand Injuries"
Auditorium

Lester W. Carlander, Minneapolis

11:30 "Bleeding from the Genito-urinary Tract"
Auditorium

O. E. Sarff, Duluth

P.M.

12:15 Round-Table Luncheons

Afternoon Session

P.M.

1:30 Visit Scientific and Technical Exhibits
Auditorium

2:00 Closed Circuit Color Television......Auditorium
Surgical Procedures—Proctology

3:15 WHAT'S NEW IN MEDICINE?
Auditorium

Moderator—H. F. R. Plass, Minneapolis

"Cardiac Trauma"
Richard B. Tregilgas, St. Paul

"Management of Diabetes with Insulin"
Dale H. Correa, Minneapolis

"Modern Ulcer Management"
R. S. Ylvisaker, Minneapolis

"Anticoagulant Therapy"
John J. Boehrer, Minneapolis

"Thorazine and Rauwolfia"
C. O. Erickson, Minneapolis

4:30 Adjournment

ARCHERY TOURNAMENT AT ANNUAL MEETING

The First Archery Tournament for physicians will be held at 1:00 p.m., Sunday, May 22, just prior to the annual meeting of the Minnesota State Medical Association. Nokomis Archery Range, located in the park adjoining Lake Nokomis on the corner of 50th street and 54th avenue south, Minneapolis, will be the scene of this contest.

This is the first time that an archery contest has been offered as one of the sports events sponsored by the Minnesota State Medical Association. As far as is known, this is the first archery tournament ever offered in the United States exclusively for members of the medical profession. This is an experiment, and it is hoped that a sufficient number of physicians will participate in this contest to justify repeating it in future years.

The contest will be simple and not too demanding. A Junior American Round will be shot consisting of 30 arrows at 50 yards, 30 arrows at 40 yards and 30 arrows at 30 yards. Two classes of archers may compete—an instinctive group which shoots without the aid of a bow sight and a free style group which uses a bow sight. Suitable trophies will be presented to the first three winners in each class. Entrance fee will be $1.00.

Robert Rhode, Minnesota State Archery champion and United States National Target champion, will set up and conduct this archery contest. In June, Robert Rhode will be the American representative to the International World Championship Archery contest to be held in Helsinki, Finland.

Participants in sports events are invited to attend with their wives a sports events dinner at 6:30 p.m., Sunday, May 22, at the Minneapolis Golf Club, South Cedar Lake Road and Flagg Avenue, following the various events of the day. Tickets are $4.50, gratuity included. Entertainment for doctors' wives will be provided during the afternoon. This is an informal dinner, and sports participants are urged to attend the dinner as they are.

The success of this contest requires the participation of all physicians who are archery enthusiasts. Contact either Carl O. Rice, M.D., 1635 Medical Arts Building, Minneapolis 2, or George M. Tangen, M.D., 1427 Medical Arts Building, Minneapolis 2, for further details on this event.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota
F. H. Magney, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 12, 1954

January, 1954, Examination

Name	*School*		*Address*
AXLER, Gueri	U. of Beirut, Lebanon	MD 1947	1018 Lowry Medical Arts Bldg., St. Paul, Minn.
BAUER, Paul Gregory	U. of Minnesota	MD 1951	8-A NW 4th St., Faribault, Minn.
BENTHACK, Elaine Marguerite	U. of Nebraska	MD 1949	Student Health Service, U. of Minn., Minneapolis, Minn.
BOWERS, Dorrance	U. of Toronto	MD 1949	200 1st St., S.W., Rochester, Minn.
BRANNICK, Thomas Leonard	U. of Illinois	MD 1952	200 1st St., S.W., Rochester, Minn.
CAREY, Lewis Stafford de Sausmarez	Queen's U.	MD 1950	200 1st St., S.W., Rochester, Minn.
CELLA, JR., Joseph Anthony	Marquette U.	MD 1952	St. Mary's Hospital, Minneapolis, Minn.
CONNOLLY, Joseph Patrick	St. Louis U.	MD 1952	1074 S. Robert St., W. St. Paul, Minn.
DIERKER, Heinrich	Friedrich Wilhelm U., Berlin, Germany	MD 1938	23 4th St., S.E., Minneapolis, Minn.
FISCHER, Robert Francis	U. of Minnesota	MD 1953	1266 Lowry Medical Arts Bldg., St. Paul, Minn.
FRICK, Paul	U. of Zurich, Switzerland	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
GARDNER, Jack Kelly	State U. of Iowa	MD 1953	Northwestern Hosp., Minneapolis, Minn.
HARSHBARGER, Harry Glen	U. of Illinois	MD 1950	200 1st St., S.W., Rochester, Minn.
HESKEL, Milton M.	U. of Pennsylvania	MD 1948	200 1st St., S.W., Rochester, Minn.
KIPPEN, Neil Ralph	U. of Manitoba	MD 1948	Breckenridge Clinic, Breckenridge, Minn.
KODRES, Nina	Leopold Franciscus U., Innsbruck, Austria	MD 1950	462 S. Robert St., St. Paul, Minn.
LEINONEN, Wendla E.	U. of Minnesota	MD 1953	201 Jefferson St., S., Wadena, Minn.
LEVIN, Manly Bernard	U. of Manitoba	MD 1952	200 1st St., S.W., Rochester, Minn.
MacDONALD, Hugh Neil Archibald	Dalhousie U.	MD 1953	200 1st St., S.W., Rochester, Minn.
MacDONALD, Mary Joyce	Dalhousie U.	MD 1953	1421 4th Ave., S.W., Rochester, Minn.
MACHELEDT, Neil Lester	U. of Minnesota	MD 1953	201 Jefferson St., S., Wadena, Minn.
MacLEAN, Lloyd Douglas	U. of Alberta	MD 1949	U. of Minnesota Hospitals, Minneapolis, Minn.
MADALIN, Herbert Edward	U. of Michigan	MD 1952	200 1st St., S.W., Rochester, Minn.
MAHONEY, Jack Lawrence	Jefferson Med. Col.	MD 1952	200 1st St., S.W., Rochester, Minn.
NELSON, Erland Randall	Columbia U	MD 1951	4221 Standish Ave. S., Minneapolis, Minn.
NEWTON, William Evans	McGill U.	MD 1951	620 View St., Victoria, B.C., Canada
OPFELL, Harriet Monson	State U. of Iowa	MD 1949	200 1st St., S.W., Rochester, Minn.
POLLARD, William Stanley	U. of Manitoba	MD 1949	200 1st St., S.W., Rochester, Minn.
RUDY, Warren B.	Northwestern U.	MD 1951	U. of Minnesota Hospitals, Minneapolis, Minn.
SCHMID, Rudolf	U. of Zurich, Switzerland	MD 1950	Columbia U., 630 W. 168 St., New York, N. Y.
SCHUMACHER, Otto Peter	Johns Hopkins U.	MD 1952	200 1st St., S.W., Rochester, Minn.
TURNER, John Jacob	Harvard U.	MD 1952	200 1st St., S.W., Rochester, Minn.

Reciprocity Candidates

Name	*School*		*Address*
ANDREW, William Fredrick	Washington U., Mo.	MD 1945	205 W. 2nd St., Duluth, Minn.
ANTONOW, Arthur Marshall	Indiana U.	MD 1945	6th Ave. and 9th St., N., Virginia, Minn.
BULLUCK, Matt Heyer	U. of Maryland	MD 1948	200 1st St., S.W., Rochester, Minn.
EPSTEIN, John Howard	U. of California	MD 1952	200 1st St., S.W., Rochester, Minn.
FITE, James Mitchell	U. of Virginia	MD 1952	U. of Minn. Hosp., Minneapolis, Minn.
FUQUAY, Maurice Coyle	U. of Oklahoma	MD 1952	200 1st St., S.W., Rochester, Minn.
GOULD, JR., Allan Bryant	Jefferson Med. Col.	MD 1952	200 1st St., S.W., Rochester, Minn.
HERBERT, Edna Eva	Creighton U.	MD 1952	200 1st St., S.W., Rochester, Minn.
HOOKER, Theodore Challence	U. of Chicago	MD 1949	200 1st St., S.W., Rochester, Minn.
KEMPER, James Willard	Indiana U.	MD 1952	200 1st St., S.W., Rochester, Minn.
KRUEGER, JR., Julius Thomas	Tulane U.	MD 1952	200 1st St., S.W., Rochester, Minn.
LUNDSTEN, Lloyd Richmond	Stanford U.	MD 1952	200 1st St., S.W., Rochester, Minn.

MARTINOV, William Edgar	Indiana U.	MD 1948	200 1st St., S.W., Rochester, Minn.
PIPER, JR., Charles E.	U. of Pittsburgh	MD 1952	200 1st St., S.W., Rochester, Minn.
SCHAEFER, Kenneth Franklyn	U. of Pennsylvania	MD 1941	Prudential Insurance Co., Minneapolis, Minn.
SCHLESINGER, Martin R.	U. of Pittsburgh	MD 1944	9040 Sunland Blvd., Sun Valley, Calif.
SCHNEIDER, John Phillip	Tulane U.	MD 1952	200 1st St., S.W., Rochester, Minn.
SCHULTZ, Herman Joseph	U. of Texas	MD 1952	200 1st St., S.W., Rochester, Minn.
SEAY, III, James Elbert	U. of Pennsylvania	MD 1952	200 1st St., S.W., Rochester, Minn.
SOMMERVILLE, Robert L.	Med. Col. of Virginia	MD 1946	200 1st St., S.W., Rochester, Minn.
WHITE, Malcolm Eugene	Louisiana State U.	MD 1945	200 1st St., S.W., Rochester, Minn.
WINDESHEIM, John Henry	U. of California	MD 1952	200 1st St., S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
CARLON, Donald L.	State U. New York	MD 1952	200 1st St., S.W., Rochester, Minn.
CIRIACY, Edward Walter	Temple U.	MD 1952	208 E. Chapman St., Ely, Minn.
COLOSEY, Frederick John	Temple U.	MD 1952	208 E. Chapman St., Ely, Minn.
CORNELIUS, Eugene Albert	U. of Toronto	MD 1952	200 1st St., S.W., Rochester, Minn.
FLOR, Frank Stephen	N. Y. Med. Col.	MD 1952	200 1st St., S.W., Rochester, Minn.
GARONO, Barbara Jane	U. of Buffalo	MD 1952	200 1st St., S.W., Rochester, Minn.
HAMBIDGE, Jr., Gove	Yale U.	MD 1945	U. of Minn. Hosp., Minneapolis, Minn.
HARTWIG, John Arnold	U. of Minnesota	MD 1953	200 1st St., S.W., Rochester, Minn.
HONAKER, Walker	U. of Colorado	MD 1945	200 1st St., S.W., Rochester, Minn.
HOWELL, Milton M.	U. of Rochester	MD 1952	624 Franklin Ave., Glencoe, Minn.
KIBLER, Charles Eugene	Johns Hopkins U.	MD 1948	200 1st St., S.W., Rochester, Minn.
KYLE, Robert A.	Northwestern U.	MD 1952	200 1st St., S.W., Rochester, Minn.
OLSON, Rolland Alfred	Col. Med. Evang.	MD 1945	Box 548, Wayzata, Minn.
PAWLIAS, Kenneth Theodore	Loyola U.	MD 1952	200 1st St., S.W., Rochester, Minn.
PINSKY, Harry Joseph	U. of Rochester	MD 1951	Strong Memorial Hospital, Rochester, N. Y.
REED, William Byron	Columbia U.	MD 1952	200 1st St., S.W., Rochester, Minn.
SCHERER, William Franklin	U. of Rochester	MD 1947	U. of Minn. Faculty Dept., Minneapolis, Minn.
SCHOLL, Virginia May	U. of Oregon	MD 1951	2447 N.W. Westover Rd., Portland, Ore.
WATKINS, Jr., Lee Clifford	Harvard U.	MD 1952	200 1st St., S.W., Rochester, Minn.
WILSON, William Sleight	U. of Pennsylvania	MD 1950	U. Michigan Medical School, Ann Arbor, Mich.

March, 1954, Examination

Name	*School*		*Address*
CAMERON, Dale Corbin	U. of Nebraska	MD 1936	117 University Ave., St. Paul, Minn.

PHYSICIANS LICENSED May 14, 1954

April, 1954 Examination

Name	*School*		*Address*
ANDERSON, Donald Albert	U. of Chicago	MD 1951	99 Williamsburg Road, Evanston, Ill.
BACHEM, Maria	U. of Wurzburg, Germany	MD 1939	St. Luke's Hospital, St. Paul, Minn.
BIRD, Grant Sutton	U. of Toronto	MD 1951	200 1st St., S.W., Rochester, Minn.
BRANDT, Henry Erwin	U. of Minnesota	MD 1953	3441 3rd Ave. S., Minneapolis, Minn.
BRUWER, Andre Johannes	U. of Cape Town, So. Africa	Bach. Med. Bach. Surg. 1942	200 1st St., S.W., Rochester, Minn.
BULL, Frances Eleanor	U. of Michigan	MD 1952	200 1st St., S.W., Rochester, Minn.
CAPLINGER, Robert Allan	U. of Michigan	MD 1952	200 1st St., S.W., Rochester, Minn.
COTÉ, Robert	Laval U.	MD 1951	200 1st St., S.W., Rochester, Minn.
DEHNEL, Luther Lenker	U. of Minnesota	MD 1953	Veterans' Adm. Hospital, Minneapolis, Minn.
EWENS, George Brown	U. of Minnesota	MD 1953	Ancker Hospital, St. Paul, Minn.
FURR, Leo O'Neil	St. Louis U.	MD 1953	Bird Island, Minn.
HODGINS, Thomas Emerson	U. of Western Ontario	MD 1951	200 1st St., S.W., Rochester, Minn.
JACKSON, Sidney Charles	Harvard U.	MD 1945	200 1st St., S.W., Rochester, Minn.
KELLNER, Hildegard	U. of Halle, E. Germany	MD 1947	State Hospital, Hastings, Minn.
LARSON, Keith Delmar	Northwestern U.	MD 1941	Moose Lake State Hospital, Moose Lake, Minn.
LINDBERG, Arthur Nathaniel	U. of Minnesota	MD 1953	538 LaSalle Bldg., Minneapolis, Minn.

MATEO, Guillermo	U. of Chicago	MD 1948	1163 Lowry Medical Arts Bldg., St. Paul, Minn.
MATTHEWS, Jr., Louis Brown	U. of Tennessee	MD 1949	200 1st St., S.W., Rochester, Minn.
MAUS, Philip Walter	U. of Minnesota	MD 1953	USAF Dispensary, APO 825, New Orleans, La.
McCASLIN, Jr., Frank Erwin	U. of Oregon	MD 1953	02430 S.W. Military Rd., Portland, Ore.
McINTOSH, James Eugene	U. of Chicago	MD 1952	U. of Minn. Hosp., Minneapolis, Minn.
METZ, Donald Delwin	Marquette U.	MD 1953	Buffalo Lake, Minn.
MULLER, Rudolf	U. of Marburg, Germany	MD 1945	Richmond, Minn.
MURPHY, Vincent John	U. of Alberta	MD 1951	200 1st St., S.W., Rochester, Minn.
NEHER, Frederick James	Harvard U.	MD 1952	U. of Minn. Hosp., Minneapolis, Minn.
OLSON, Robert Charles	Marquette U.	MD 1953	205 E. Walnut St., Green Bay, Wis.
ORCHARD, Jr., Welland John	U. of Colorado	MD 1953	727 Broadway, Fargo, No. Dak.
PHILLIPS, Donald Francis	Tufts U.	MD 1949	200 1st St., S.W., Rochester, Minn.
PRIBEK, Robert Andrew	Marquette U.	MD 1953	Veterans' Adm. Hospital, Wood, Wis.
ROTTENBERG, Everett Newton	U. of Michigan	MD 1952	200 1st St., S.W., Rochester, Minn.
ROVELSTAD, Roger Andrew	Northwestern U.	MD 1953	221 E. First St., Fairmont, Minn.
SCAPPATURA, Antonio Rudolfo	U. of Ottawa	MD 1952	200 1st St., S.W., Rochester, Minn.
SCHOSSOW, George William	U. of Nebraska	MD 1953	Erskine, Minn.
SEGAL, Edward L.	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
SEMLER, Herbert Joseph	U. of Oregon	MD 1953	200 1st St., S.W., Rochester, Minn.
SWERDLOW, Ralph Hyman	U. of Illinois	MD 1949	3415 Emerson Ave. S., Minneapolis, Minn.
TAUXE, Welby Newlon	U. of Tennessee	MD 1950	200 1st St., S.W., Rochester, Minn.
THOMPSON, Ardell Henry	U. of Minnesota	MD 1953	35 E. 4th, Litchfield, Minn.
THORGERSEN, Thor Magnus	Marquette U.	MD 1953	R.F.D. No. 3, Rochester, Minn.
TUURA, James Lynn	U. of Wisconsin	MD 1953	U. of Wisconsin Hospital, Madison, Wis.
VREELAND, Karl Edward	U. of Oregon	MD 1953	916 N. Tacoma Ave., Tacoma, Wash.
WATSON, Robert Neil	U. of Illinois	MD 1953	Wood Veterans' Adm. Hospital, Wood, Wisconsin
WEIL, Max Harry	U. of New York	MD 1952	U. of Minn. Hosp., Minneapolis, Minn.
WILCOX, William A.	Northwestern U.	MD 1950	Veterans' Adm. Hospitals, Minneapolis, Minn.
YUE, Wen Yao	St. John's U., Shanghai, China	MD 1945	Glen Lake San., Oak Terrace, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BARTZEN, Peter James	Marquette U.	MD 1948	205 W. 2nd St., Duluth, Minn.
CAMPBELL, Jr., Gilbert Sadler	U. of Virginia	MD 1946	U. of Minn. Hosp., Minneapolis, Minn.
CORR, Donald John	Stanford U.	MD 1953	200 1st St., S.W., Rochester, Minn.
CROWLEY, Jr., William Patrick	U. of Wisconsin	MD 1952	200 1st St., S.W., Rochester, Minn.
DICKINSON, Lane Sawyer	U. of Rochester	MD 1949	200 1st St., S.W., Rochester, Minn.
DOWNING, William Conrad	Western Reserve U.	MD 1945	220 S. Broadway, Crookston, Minn.
GURNETT, Thomas Joseph	Creighton U.	MD 1946	200 1st St., S.W., Rochester, Minn.
KNIGHT, James Harry	U. of California	MD 1951	200 1st St., S.W., Rochester, Minn.
LUBY, Thomas Harry	U. of Nebraska	MD 1952	200 1st St., S.W., Rochester, Minn.
PATTEE, James J.	Creighton U.	MD 1953	5615 Hampshire Ave. N., Minneapolis, Minn.
PEARCE, Jr., Francis Marion	Tulane U.	MD 1951	200 1st St., S.W., Rochester, Minn.
RODENBAUGH, Fredrich Hase	Stanford U.	MD 1953	200 1st St., S.W., Rochester, Minn.
SARNO, John Anthony	Hahnemann Med. Col.	MD 1946	318 N. Labree Ave., Thief River Falls, Minn.
SELZER, Donald Wright	U. of Kansas	MD 1947	200 1st St., S.W., Rochester, Minn.
SMID, Arthur Charles	Creighton U.	MD 1948	200 1st St., S.W., Rochester, Minn.
STAHL, George William	U. of Wisconsin	MD 1950	404½ N. Main, Austin, Minn.
WATSON, Virgil Austin	St. Louis U.	MD 1949	Bronson Methodist Hospital, Kalamazoo, Mich.

National Board Candidates

Name	*School*		*Address*
CONKLIN, Frances Phillips	U. of Vermont	MD 1951	U. of Minn. Hosp., Minneapolis, Minn.
COOK, Jr., Roderick Walker	Temple U.	MD 1951	200 1st St., S.W., Rochester, Minn.
JIM, Vernon Kang Sung	U. of Chicago	MD 1944	200 1st St., S.W., Rochester, Minn.
McNEAR, Jr., George Roger	Cornell U.	MD 1947	Mankato Clinic, Mankato, Minn.
MOERSCH, Richard Norval	Harvard U.	MD 1952	200 1st St., S.W., Rochester, Minn.
ROBBINS, Arnold Robert	N. Y. Med. Col.	MD 1947	Heron Lake, Minn.
VASSALOTTI, Margaret J.	Woman's Med. Col., Pa.	MD 1952	200 1st St., S.W., Rochester, Minn.
WOLD, Keith Clinton	U. of Vermont	MD 1951	852 Lowry Medical Arts Bldg., St. Paul, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

PHYSICIANS LICENSED JULY 16, 1954

June, 1954, Examination

Name	*School*		*Address*
ABRAMS, Jr., Alexander	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
AHLSTROM, Robert Clarence	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
ANDERSON, Eunice Rose	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
ANDERSON, William Evan	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
ANONSEN, Richard Eldon	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
APPLEN, John Ellsworth	U. of Minnesota	MD 1954	U.S.P.H.S. Hospital, Baltimore, Md.
ARONSOHN, Richard Burton	U. of Minnesota	MD 1953	1515 Charles Ave., St. Paul, Minn.
BAH, Merle Charles	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
BAKEN, Jr., Melvin Palmer	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
BELL, Donald Craig	U. of Minnesota	MD 1953	200 1st St., S.W., Rochester, Minn.
BERGLUND, Jr., Alvin E.	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
BESSER, Vera	U. of Minnesota	MD 1953	Anoka State Hospital, Anoka, Minn.
BRAUTI, Erling Frederick	U. of Oregon	MD 1953	Mpls. Gen. Hospital, Minneapolis, Minn.
BRIGGS, Brian Earl	U. of Minnesota	MD 1954	St. Mary's Hospital, Duluth, Minn.
BROWN, Jr., James Edward	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
BURGER, Thomas Charles	St. Louis U.	MD 1949	200 1st St., S.W., Rochester, Minn.
BURKE, Gerald F.	U. of Minnesota	MD 1954	Hartford Hospital, Hartford, Conn.
BURNSIDE, Jr., Alfred Flournoy	Med. Col. of So. Carolina	MD 1952	200 1st St., S.W., Rochester, Minn.
BUTZER, John Francis	Loyola U.	MD 1953	203 Nat. City Bank Bldg., Mankato, Minn.
CALLAN, Joseph Albert	U. of Minnesota	MD 1954	Hurley Hospital, Flint, Mich.
CAMPBELL, Wayne Leigh	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
CARLSON, Robert Gerald	U. of Minnesota	MD 1954	U. of Oregon Hospital, Portland, Ore.
CASEY, James Hugh	Marquette U.	MD 1951	U. of Minn. Hosp., Minneapolis, Minn.
CHRISTENSEN, Warren Albert	U. of Minnesota	MD 1954	Kaiser Foundation Hospital, Oakland, Calif.
CLARK, William Bruce	U. of Minnesota	MD 1954	401 E. 2nd St., Duluth, Minn.
DECKER, James Windsor	U. of Minnesota	MD 1954	Los Angeles County Hospital, Los Angeles, Calif.
DECKER, Kathleen Hogan	U. of Minnesota	MD 1954	Los Angeles County Hospital, Los Angeles, Calif.
DENNIS, Frank De Atley	U. of Minnesota	MD 1954	Denver General Hospital, Denver, Colo.
DITMANSON, Marcy Leroy	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
DONALDSON, James Adrian	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
DORSEY, Jr., George Charles	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
DUNPHY, Stephen R.	U. of Minnesota	MD 1954	St. Luke's Hospital, Denver, Colo.
DYRDAL, Paul Jerome	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
ELLINGSON, William Lyle	U. of Minnesota	MD 1954	Los Angeles County Hospital, Los Angeles, Calif.
ETTINGER, Milton G.	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
EVERS, William	Fed. Institute of Med., Samarkand, Russia	MD 1945	214 Wesley Temple Bldg., Minneapolis, Minn.
FEDOR, Robert D.	U. of Minnesota	MD 1954	Chas. T. Miller Hospital, St. Paul, Minn.
FELLER, William Frank	U. of Minnesota	MD 1954	U. of Minn. Hosp., Minneapolis, Minn.
FINGERHUT, Aaron Gerald	U. of Minnesota	MD 1954	Wm. Beaumont Army Hospital, El Paso, Texas
FINLEY, Richard Clayton	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
GAARDER, Kenneth Ray	U. of Minnesota	MD 1954	Detroit Receiving Hosp., Detroit, Mich.
GAEBE, Milton B.	U. of Louisville	MD 1953	Tholen Bldg., Clara City, Minn.
GAMM, Edgar R.	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
GEIST, Robert Williams	U. of Minnesota	MD 1954	Philadelphia General Hospital, Philadelphia, Pa.
GEORGE, Jr., Vane P.	U. of Rochester	MD 1952	737 E. 22nd St., Minneapolis, Minn.
GISLASON, Paul Harold	U. of Maryland	MD 1952	7233 Pillsbury Ave. S., Minneapolis, Minn.
GOEHRS, Homer Richard	Baylor U.	MD 1950	200 1st St., S.W., Rochester, Minn.
GREENE, Leonard H.	U. of Minnesota	MD 1954	Broadlawns Polk Co. Hospital, Des Moines, Iowa
GRIMES, Paul Thomas	U. of Minnesota	MD 1954	Harrisburg Hospital, Harrisburg, Pa.
GULLEN, Warren Hartley	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
GUNSOLUS, John Martin	U. of Minnesota	MD 1952	940 E. 4th St., Stuart, Fla.
HAGLUND, Roger V.	U. of Minnesota	MD 1954	U.S.P.H.S. Hospital, Seattle, Wash.
HANDLER, Seymour	U. of Minnesota	MD 1953	101 7th Ave. S., St. Cloud, Minn.
HANSON, Kent Oscar	U. of Minnesota	MD 1954	Denver General Hospital, Denver, Colo.
HAUGAN, Norman Rye	U. of Minnesota	MD 1953	119 N. Broadway, Crookston, Minn.
HAUKENESS, Stanley J.	U. of Minnesota	MD 1954	King County Hospital, Seattle, Wash.
HEDRICK, William Louis	U. of Minnesota	MD 1954	Detroit Receiving Hosp., Detroit, Mich.
HEETDERKS, Jr., Bernard John	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
HILL, John Paul	Northwestern U.	MD 1952	401 1st Ave. So., Virginia, Minn.
HODGES, Kenneth Verplank	U. of Minnesota	MD 1954	Gorgas Hospital, Ancon, Canal Zone

HOTTINGER, George Creighton	U. of Minnesota	MD 1954	34 Primrose Place, New Dorp, S.I. 6, New York
HULTENG, Donald Bruce	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
JENNE, John William	U. of Minnesota	MD 1954	Williamsport Hospital, Williamsport, Pa.
JERNBERG, Kenneth Irving	U. of Minnesota	MD 1954	King County Hospital, Seattle, Wash.
JOHNSON, Emery Allen	U. of Minnesota	MD 1954	Asbury Meth. Hosp., Minneapolis, Minn.
JOHNSON, Ronald Glen	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
JOHNSON, Jr., Thomas Harrington	U. of Minnesota	MD 1954	Mount Sinai Hospital, Minneapolis, Minn.
JORGENSEN, Edward Olaf	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
KATKOV, Morris Ralph	U. of Minnesota	MD 1954	Highland-Alameda Co. Hospital, Oakland, Calif.
KAUTH, John Edward	U. of Minnesota	MD 1954	Mercy Hospital, Toledo, Ohio
KOSIAK, Michael	U. of Minnesota	MD 1954	Minn. Gen. Hospital, Minneapolis, Minn.
KOTTKE, Bruce Allen	U. of Minnesota	MD 1954	Indianapolis Gen. Hospital, Indianapolis, Ind.
KREBSBACH, Richard James	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
KRIVIT, William	Tulane U.	MD 1948	U. of Minn. Hosp., Minneapolis, Minn.
KUHLMANN, Vernon George	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
LABERGE, Martin	Laval U.	MD 1951	200 1st St., S.W., Rochester, Minn.
LARSEN, Martha Carlisle	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
LARSON, Arthur King	U. of Pittsburgh	MD 1953	Children's Hospital, Pittsburgh, Pa.
LARSON, Arthur Norman	U. of Minnesota	MD 1954	U. of Minn. Hosp., Minneapolis, Minn.
LARSON, Dorette Wheat	U. of Minnesota	MD 1954	Mt. Sinai Hosp., Minneapolis, Minn.
LEON, Paul Roy	U. of Minnesota	MD 1954	St. Mary's Hospital, Grand Rapids, Mich.
LILLINGTON, Glen Alan	U. of Manitoba	MD 1951	200 1st St., S.W., Rochester, Minn.
LINDAHL, James Bushnell	U. of Minnesota	MD 1954	Wm. Beaumont Army Hospital, El Paso, Texas
LITKEWITSCH, Helene	Med. Inst. Charkow, Ukraine	1935 "Physician"	St. Luke's Hospital, St. Paul, Minn.
LITTLE, James McNair	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
LUKK, Olaf	Eberhard-Karls U. Tubingen, Germany	1949 "Physician"	Gosewisch Bldg., Montgomery, Minn.
LYNN, Carol Lavon	U. of Minnesota	MD 1954	Highland Alameda Co. Hospital, Oakland, Calif.
LYNNER, Robert Ecklund	U. of Minnesota	MD 1954	City Hospital, Akron, Ohio
MAST, Fredric L.	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
MATTSON, Donald Sheldon	U. of Minnesota	MD 1954	Asbury Meth. Hosp., Minneapolis, Minn.
McALLISTER, Robert Lynn	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
McCARTHY, Charles Joseph	U. of Minnesota	MD 1954	9344 Genessee Ave., Detroit, Mich.
MEGER, Gene Edward	U. of Minnesota	MD 1954	Philadelphia Gen. Hospital, Philadelphia, Pa.
MERKEL, Giles Gene	U. of Minnesota	MD 1954	4540 W. 10th Ave., Phoenix, Ariz.
MEYER, Richard Hollister	U. of Minnesota	MD 1954	Presbyterian Hospital, Chicago. Ill.
MIETTUNEN, John Bernard	U. of Minnesota	MD 1953	30 N.W. 1st St., Chisholm, Minn.
MILLER, William Peters	U. of Minnesota	MD 1954	Asbury Methodist Hospital, Minneapolis, Minn.
MORAN, Leo Joseph	U. of Minnesota	MD 1954	City & County Hospital, San Francisco, Calif.
NEISWANGER, Walter Erwin	Loyola U.	MD 1952	200 1st St., S.W., Rochester, Minn.
NELSON, Douglas Alan	U. of Minnesota	MD 1954	Philadelphia Gen. Hospital, Philadelphia, Pa.
NERENBERG, Sidney	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
NICHOLS, Francis Charles	Loyola U.	MD 1952	200 1st St., S.W., Rochester, Minn.
OTT, Eugene C.	U. of Minnesota	MD 1954	Detroit Receiving Hosp., Detroit, Mich.
PARDEE, Neely E.	U. of Minnesota	MD 1954	U.S.P.H.S. Hosp., San Francisco, Calif.
PARKER, Thomas Myles	U. of Minnesota	MD 1954	St. Mary's Hospital, Duluth, Minn.
PELZL, Charles R.	U. of Minnesota	MD 1954	St. Mary's Hospital, Duluth, Minn.
PETERSON, Edward Nohl	U. of Minnesota	MD 1954	Edward J. Meyer Memorial Hospital, Buffalo, N. Y.
PETTEE, Richard Addison	U. of Nebraska	MD 1951	Vets' Adm. Hospital, Minneapolis, Minn.
PHILLIPS, Leonard	Marquette U.	MD 1952	St. Joseph's Hospital, St. Paul, Minn.
PILGRIM, Robert Dwight	U. of Minnesota	MD 1954	Kaiser Foundation Hosp., Oakland, Calif.
RABCEVICH, Anatole	U. of Minnesota	MD 1953	Ancker Hospital, St. Paul, Minn.
RADKE, Robert Lyle	U. of Minnesota	MD 1954	Asbury Meth. Hosp., Minneapolis, Minn.
REARDON, Andrew Eugene	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
REIN, Burton Irwin	U. of Minnesota	MD 1954	Dist. of Columbia Gen. Hosp., Washington, D. C.
ROBBINS, Mason C.	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
ROCK, William Henry	U. of Minnesota	MD 1954	816 Greenwich Rd., Minneapolis, Minn.
ROOT, Harlan David	Cornell U.	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
SADOFF, Leonard	U. of Minnesota	MD 1954	Santa Clara Co. Hosp., San Jose, Calif.
SALOUM, Lucille Mary	U. of Minnesota	MD 1954	Oakland Naval Hosp., Oakland, Calif.
SAVARD, Marc	U. of Montreal	MD 1952	200 1st St., S.W., Rochester, Minn.
SAWARYNJUK, Iwan	Goethe U. at Frankfurt, Germany	MD 1951	Diessner Bldg., Waconia, Minn.

SCHABACKER, Charles Walker	U. of Minnesota	MD 1954	Fitzsimons Army Gen. Hosp., Denver, Colo.
SCHLICHTING, James Robert	U. of Minnesota	MD 1954	Ancker Hospital, St. Paul, Minn.
SCHMITZ, Robert Eugene	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
SCHOENING, Herbert Alan	U. of Minnesota	MD 1954	San Francisco City & County Hosp., San Francisco, Calif.
SCHOON, Doris Vivien	U. of Minnesota	MD 1954	402 N. Kniss, Luverne, Minn.
SCHULTZ, Russell Thomas	U. of Minnesota	MD 1953	5212 Glengarry Pkwy., Minneapolis, Minn.
SELBY, John Bayne	U. of Tennessee	MD 1946	200 1st St., S.W., Rochester, Minn.
SENDERS, Wilbur Lester	U. of Oregon	MD 1950	200 1st St., S.W., Rochester, Minn.
SERBER, Lawrence	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
SETHRE, Arthur E.	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
SINYKIN, Gerald Bernard	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
SMITH, Duane Samuel	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
STANLEY, Charles Otis	U. of Illinois	MD 1950	200 1st St., S.W., Rochester, Minn.
STEIN, William Arthur	U. of Iowa	MD 1953	So. 1st Av. E. & Harvey, Ely, Minn.
STENZEL, David Milton	U. of Minnesota	MD 1954	U. S. Naval Hospital, Bethesda, Md.
STUDENT, Richard Edward	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
SWEDLUND, Harry Arvid	U. of Minnesota	MD 1954	U.S.P.H.S. Hosp., San Francisco, Calif.
THURBER, Deloran Leigh	U. of Minnesota	MD 1954	Denver Gen. Hospital, Denver Colo.
TRAUTMANN, James Curtis	U. of Minnesota	MD 1954	St. Luke's Hospital, Duluth, Minn.
VIBERT, James Charles	Dalhousie U.	MD 1951	200 1st St., S.W., Rochester, Minn.
WARWICK, Warren James	U. of Minnesota	MD 1954	White Cross Hospital, Columbus, Ohio
WEEKS, Richard Elvin	Northwestern U.	MD 1949	200 1st St., S.W., Rochester, Minn.
WETHERBY, Jr., Macnider	U. of Minnesota	MD 1954	Vets' Adm. Hosp., Long Beach, Calif.
WETTELAND, Thomas Franklin	Northwestern U.	MD 1953	Midway Hospital, St. Paul, Minn.
WHITACRE, II, John Clifford	U. of Minnesota	MD 1954	926 Summit Ave., St. Paul, Minn.
WILSON, Robert Edward	U. of Minnesota	MD 1954	930 E. 2nd St., Duluth, Minn.
WOHLRABE, Robert G.	U. of Minnesota	MD 1954	6528 Logan Ave. S., Minneapolis, Minn.
WOODLEY, Donald William	U. of Minnesota	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
YUMIBE, Toshio	U. of Minnesota	MD 1954	Santa Clara Co. Hosp., San Jose, Calif.

Reciprocity Candidates

Name	*School*		*Address*
CAMP, Ray Junior	U. of Nebraska	MD 1944	Madison Clinic, Madison, Minn.
CLAPP, Hubert Dickason	Western Reserve U.	MD 1939	220 S. Broadway, Crookston, Minn.
COMPTON, Russell Frederick	U. of Illinois	MD 1951	200 1st St., S.W., Rochester, Minn.
GROSCHUPF, Richard Paul	Creighton U.	MD 1953	6th & Beltrami, Bemidji, Minn.
HENDERSON, Robert Earl	U. of Pennsylvania	MD 1952	200 1st St., S.W., Rochester, Minn.
HIMEBAUGH, Gilbert Joseph	Indiana U.	MD 1942	200 1st St., S.W., Rochester, Minn.
HULT, John Edward	Washington U.	MD 1949	903 Cloquet Ave., Cloquet, Minn.
JOHNSON, J. W.	Johns Hopkins U.	MD 1948	200 1st St., S.W., Rochester, Minn.
LANDIS, Charles Walter	Indiana U.	MD 1951	Central State Hosp., Indianapolis, Ind.
MANN, George Adam	Marquette U.	MD 1949	214 Wesley Temple Bldg., Minneapolis, Minn.
McCLELLAN, Robert James	U. of Pittsburgh	MD 1947	Ancker Hospital, St. Paul, Minn.
McKAY, John Warren	U. of Kansas	MD 1950	200 1st St., S.W., Rochester, Minn.
McKAY, Kenneth Hubert	State U. of Iowa	MD 1952	200 1st St., S.W., Rochester, Minn.
McNEIL, Elbert Daniel	U. of Colorado	MD 1948	200 1st St., S.W., Rochester, Minn.
NICHOLS, Raymond Dane	Ohio State U.	MD 1953	Main St., Crosby, Minn.
ONIFER, Theodore Michael	Temple U.	MD 1948	200 1st St., S.W., Rochester, Minn.
OSBORNE, John Edward	U. of Virginia	MD 1952	R.R. No. 1, Box 252-A, Miamisburg, Ohio
PAYNE, William Spencer	Washington U., Mo.	MD 1950	1515 Lafayette, St. Louis, Mo.
PETERSEN, Robert William	U. of Colorado	MD 1949	200 1st St., S.W., Rochester, Minn.
PHALEN, Patrick Thomas	Creighton U.	MD 1952	200 1st St., S.W., Rochester, Minn.
SCIARRA, Paschal A.	U. of Cincinnati	MD 1951	200 1st St., S.W., Rochester, Minn.
WILCOX, III, Charles Frederick	Temple U.	MD 1952	200 1st St., S.W., Rochester, Minn.
YARBRO, Harold Ray	U. of Virginia	MD 1949	200 1st St., S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
BEJIAN, Henry Leonard	Albany Med. Col.	MD 1940	600 McClellan St., Schenectady, N. Y.
BOYEA, Lyle Henry	Syracuse U.	MD 1949	Babbitt, Minn.
HINKLEY, William Corriden	U. of Minnesota	MD 1952	Veterans' Adm. Hospital, Minneapolis, Minn.
IRELAND, Gerald William	U. of Minnesota	MD 1953	Itasca Clinic, Nashwauk, Minn.
JOHNSON, William Joseph	Yale U.	MD 1952	200 1st St., S. W., Rochester, Minn.
LESTER, Richard Garrison	Columbia U.	MD 1948	Heart Hospital, U. of Minnesota, Minneapolis, Minn.

MASSARELLI, JR., John Joseph	Georgetown U.	MD 1950	200 1st St., S.W., Rochester, Minn.
MURPHY, Thomas Ogden	Johns Hopkins U.	MD 1949	U. of Minn. Hosp., Minneapolis, Minn.
PARIS-ROCA, Jaime	Syracuse Med. Col.	MD 1948	200 1st St., S.W., Rochester, Minn.
SIMPSON, Silvan Aaron	U. of Buffalo	MD 1952	Veterans' Adm. Hospital, Minneapolis, Minn.

PHYSICIANS LICENSED NOVEMBER 12, 1954

October, 1954, Examination

Name	*School*		*Address*
ABULLARADE, Jose Antonio	U. El Salvador San Salvador, C. A.	MD 1950	Cokato Hospital, Cokato, Minn.
ADAMS, Hilde Helene Annemarie Margarete	U. of Hamburg, Germany	MD 1943	1408 Mayo Memorial Bldg., U. of Minn., Minneapolis, Minn.
ADKINS, James Thomas	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
ARONSON, Jason Allen	U. of Minnesota	MD 1953	610 E. 15th St., Minneapolis, Minn.
BARNES, William Henry	Queen's U.	MD 1951	200 1st St., S.W., Rochester, Minn.
BEVIS, William David	Yale U.	MD 1949	U. of Minn. Hosp., Minneapolis, Minn.
BIRD, Bruce Lyn	U. of Toronto	MD 1952	200 1st St., S.W., Rochester, Minn.
BULKLEY, Andrew Derick	Cornell U.	MD 1951	645 J. St., Medford, Ore.
CARLSON, Curtis Harvey	U. of Minnesota	MD 1953	6508 Newton Ave. S., Minneapolis, Minn.
FALLS, John Laurance	U. of Illinois	MD 1947	628 W. 3rd St., Red Wing, Minn.
GREGG, Robert Harry	U. of Iowa	MD 1947	4334 N. 56th St., Omaha, Nebr.
HALIBURTON, Robert Alexander	U. of Toronto	MD 1951	200 1st St., S.W., Rochester, Minn.
HAMMERSTROM, Robert Norman	U. of Utah	MD 1948	U. of Minn. Hosp., Minneapolis, Minn.
HAYWA, Eugene William	U. of Minnesota	MD 1953	421 2nd Ave. S.E., Minneapolis, Minn.
HILDING, David Anderson	U. of Minnesota	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
HUNT, James Calvin	Bowman-Gray	MD 1953	200 1st St., S.W., Rochester, Minn.
MACKINNON, Margaret	McGill U.	MD 1953	200 1st St., S.W., Rochester, Minn.
McELLISTREM, Gerald Dominic	Creighton U.	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
MESSICK, Richard Taylor	Ohio State U.	MD 1953	200 1st St., S.W., Rochester, Minn.
MOERTEL, Charles George	U. of Illinois	MD 1953	200 1st St., S.W., Rochester, Minn.
MYDLAND, Wallace Elton	U. of Alberta	MD 1952	200 1st St., S.W., Rochester, Minn.
NAGOBADS, Visvaldis	U. of Tubingen, Germany	MD 1950	Swedish Hospital, Minneapolis, Minn.
NISSWANDT, Albert Louis	U. of Minnesota	MD 1953	Hastings State Hospital, Hastings, Minn.
OLSON, Philip Arthur	U. of Illinois	MD 1947	Medical Arts Bldg., Minneapolis, Minn.
QUIE, Paul Gerhardt	Yale U.	MD 1953	Minneapolis Gen. Hospitals, Minneapolis, Minn.
RICHTER, Paul Hartwell	U. of Nebraska	MD 1954	Minneapolis Gen. Hospitals, Minneapolis, Minn.
SNYDER, Carol Therese	Western Reserve U.	MD 1952	833 Goodrich Ave., St. Paul, Minn.
SPENCER, Robert J.	Loyola U.	MD 1953	200 1st St., S.W., Rochester, Minn.
STURTZ, SR., George Stephen	Georgetown U.	MD 1953	200 1st St., S.W., Rochester, Minn.
TOWNSEND, James Dayton	U. of Michigan	MD 1952	Veterans' Adm. Hospital, Minneapolis, Minn.
WOODINGTON, George Fraser	U. of Wisconsin	MD 1953	200 1st St., S.W., Rochester, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BARIDON, JR., David	Northwestern U.	MD 1953	200 1st St., S.W., Rochester, Minn.
BOGLE, Warren Cyrus	State U. of Iowa	MD 1944	U. of Minn. Hosp., Minneapolis, Minn.
BURNETT, Lonnie Sheldon	U. of Texas	MD 1953	200 1st St., S.W., Rochester, Minn.
DINES, David Eaton	U. of Colorado	MD 1953	200 1st St., S.W., Rochester, Minn.
ELLENZ, George B.	Loyola U.	MD 1951	Chas. T. Miller Hospital, St. Paul, Minn.
EWEN, Edgar Francis	Loyola U.	MD 1944	200 1st St., S.W., Rochester, Minn.
GIBSON, Smith Hyson	U. of Louisville	MD 1950	200 1st St., S.W., Rochester, Minn.
GUIDRY, Leslie David	Louisiana State U.	MD 1953	200 1st St., S.W., Rochester, Minn.
HARLAN, William Lee	Indiana U.	MD 1947	200 1st St., S.W., Rochester, Minn.
HAYLETT, Clarice Helm	Stanford U.	MD 1949	200 1st St., S.W., Rochester, Minn.
HICKS, Shelby Leon	U. of Arkansas	MD 1951	200 1st St., S.W., Rochester, Minn.
JEFFERIES, William Ladson	U. of Arkansas	MD 1945	3981 Zartlan Ave., St. Louis Park, Minneapolis, Minn.
McCLEARY, Jack Eugene	Stanford U.	MD 1951	200 1st St., S.W., Rochester, Minn.
PHIFER, Robert Louis	Harvard U.	MD 1944	200 1st St., S.W., Rochester, Minn.
SMOLLEN, William James	U. of Wisconsin	MD 1953	200 1st St., S.W., Rochester, Minn.
SPERL, JR., Michael Paul	U. of Minnesota	MD 1951	200 1st St., S.W., Rochester, Minn.
STEVENS, Robert Graham	Jefferson Med. Col.	MD 1946	200 1st St., S.W., Rochester, Minn.
TEETER, Richard Roop	U. of Pennsylvania	MD 1947	U. of Minn. Hosp., Minneapolis, Minn.
TUCKER, Robert L.	Indiana U.	MD 1944	200 1st St., S.W., Rochester, Minn.

TURNER, Charles Robert	U. of Arkansas	MD 1950	609 Medical Arts Bldg., Minneapolis, Minn.
WALL, Wendell Hardy	Col. Med. Evang.	MD 1953	116 6th St. N., Wahpeton, No. Dak.
WILSON, Paul Frederick	U. of Oregon	MD 1947	200 1st St., S.W., Rochester, Minn.
WINGE, Herbert Clayton	Northwestern U.	MD 1952	310 Dakota Ave., Wahpeton, No. Dak.

National Board Candidates

Name	*School*		*Address*
ALDEN, Charles W.	Geo. Washington U.	MD 1944	Box 70, Kimball, Minn.
BARTHOLOMEW, Lee Enderlin	Columbia U.	MD 1950	200 1st St., S.W., Rochester, Minn.
BERMAN, Herbert L.	U. of Buffalo	MD 1950	200 1st St., S.W., Rochester, Minn.
BURROUGHS, John Townsend	Harvard U.	MD 1950	200 1st St., S.W., Rochester, Minn.
CAVA, Edward Ennio	U. of Louisville	MD 1947	U. S. Army Hospital, Dugway, Utah
COLMAN, Edward Leonard	Harvard U.	MD 1950	Veterans' Adm. Hospital, Minneapolis, Minn.
FURLOW, William Loomis	Geo. Washington U.	MD 1953	108 Central, Faribault, Minn.
GALL, JR., John Christian	Geo. Washington U.	MD 1953	200 1st St., S.W., Rochester, Minn.
GLEW, William Bainbridge	Geo. Washington U.	MD 1953	200 1st St., S.W., Rochester, Minn.
HARRISON, JR., Edgar George	St. Louis U.	MD 1952	200 1st St., S.W., Rochester, Minn.
HILL, Richard Woolsey	Jefferson Med. Col.	MD 1953	200 1st St., S.W., Rochester, Minn.
HUSTEAD, Arthur Procter	Yale U.	MD 1952	200 1st St., S.W., Rochester, Minn.
MARGULIS, Alexander Rafailo	Harvard U.	MD 1950	U. of Minn. Hosp., Minneapolis, Minn.
McKENNA, John Joseph	Tufts Col. Med. Sch.	MD 1944	401 1st Ave. S., Virginia, Minn.
MILLER, Fletcher Allen	U. of Iowa	MD 1946	U. of Minn. Hosp., Minneapolis, Minn.
PASCUZZI, Chris Anthony	Creighton U.	MD 1950	200 1st St., S.W., Rochester, Minn.
PLUM, George Elwood	Harvard U.	MD 1951	200 1st St., S.W., Rochester, Minn.
RANDALL, David Addison	U. of Minnesota	MD 1953	200 1st St., S.W., Rochester, Minn.
SABIN, Frederick Chapman	Albany Med. Col.	MD 1953	200 1st St., S.W., Rochester, Minn.
SPRING, John Denison	McGill U.	MD 1930	135 Main St., Nashua, N. H.
STEIN, Harold Aaron	U. of Toronto	MD 1953	200 1st St., S.W., Rochester, Minn.
TORKELSON, Lorraine Jeanette	Woman's Med. Col., Pa.	MD 1951	Wesley Memorial Hospital, Chicago, Ill.
TROEN, Philip	Harvard U.	MD 1948	200 1st St., S.W., Rochester, Minn.
WAKAI, Coolidge Shiro	U. of Buffalo	MD 1953	200 1st St., S.W., Rochester, Minn.
WEINER, Alan David	Boston U.	MD 1952	200 1st St., S.W., Rochester, Minn.
WINTER, JR., Malcolm D.	Northwestern U.	MD 1953	200 1st St., S.W., Rochester, Minn.
WORMER, Duncan Conrad	U. of Pennsylvania	MD 1950	200 1st St., S.W., Rochester, Minn.

CHOLANGIOGRAPHY

(Continued from Page 234)

Conclusions

A method of cholangiography has been described which seems to fulfill the requirements for a rapid, simply performed procedure. It is placed at the stage of the operation which allows progress with minimal delay. In seventy-five cases studied, the x-ray pictures resulted in as good exposures as by any other technique heretofore employed for the introduction of dye into the common duct. It can be employed in acute cases in which any of the techniques previously described probably could not be used.

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2. Best, R. R.: Cholangiography. Internat. Abst. of Surg., 126-131, 1938.
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Minnesota Academy of Medicine

Meeting of April 14, 1954

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, April 14, 1954. Dinner was served at 7 o'clock and the meeting was called to order by the President, Dr. E. A. Regnier, at 8:30 p.m.

There were fifty-one members and two guests present.

Minutes of the March meeting were not read because of the length of this evening's program.

Upon ballot, the following men were elected as candidates for membership in the Academy:

Harold E. Miller	Minneapolis
J. P. Medelman	Saint Paul
Bernard G. Lannin	Saint Paul

The following were elected as officers for the coming year:

Charles N. Hensel	President
Frederick H. K. Schaaf	Vice President
Robert E. Priest	Secretary-Treasurer (re-elected)

Dr. Priest read the Secretary's annual report and also the Treasurer's annual report; both were accepted.

The scientific program followed:

Dr. Ben Sommers, Saint Paul, read his inaugural thesis entitled "Problems of the Clinical Diagnosis and Classification of Ventricular Hypertrophy." Lantern slides were shown, and cases were reported.

Discussion

Dr. Joseph Borg, Saint Paul: I think we are very fortunate in having this subject brought to us, as it should be occasionally. It is very humbling to have cases brought to our attention, as errors in diagnosis are so frequent. We can learn much from an exchange of experiences in these patients, and I should like to mention at this time two interesting cases.

One was a girl, aged sixteen, who had hypertrophy of the left ventricle and who was seen first in the early stage of myocardial failure. In spite of the fact that this was her first decompensation, she failed to respond to all measures available and gradually pursued a downhill course to death in about three months. At autopsy a marked hypoplasia with narrowing of the entire aorta, associated with cardiac hypertrophy and chronic passive congestion, was the only finding.

The other patient was a boy, aged fifteen, who had relatively minor complaints, especially nausea, vomiting, and abdominal distress, with a markedly enlarged heart and left bundle branch block. He died this morning [April 14, 1954]. He had a loud, rough systolic murmur, transmitted over the entire chest, heard best at the left sternal border and thought to be probably due to an interventricular septal defect. Cardiac catheterization, however, showed no abnormality. At autopsy, the heart showed general enlargement, weight 1000 grams. No valve defect or congenital abnormality was found. The myocardium showed large areas of fibrosis, the significance of which is as yet undetermined.

These cases illustrate difficulty presented in diagnosis. Many cases of ventricular hypertrophy are probably due to previously existing hypertension, but obviously there is no reason for arriving at such a conclusion simply because no other reason for enlargement is found.

Dr. T. A. Peppard, Minneapolis: I have been delighted to hear Dr. Sommers' splendid presentation. We all see occasional cases with cardiac enlargement, which go on to failure and death, in which the autopsy does not explain the cardiac hypertrophy; these are then termed idiopathic. I am interested in the familial type, of which Dr. Sommers speaks, because I was early conditioned to consideration of the constitution in relation to internal diseases. I should like to ask Dr. Sommers whether he believes that relative coronary insufficiency might act as a possible cause of the hypertrophy.

Dr. Moses Barron, Minneapolis: I want to compliment Dr. Sommers for his very interesting presentation of a series of cases on idiopathic cardiac enlargement. The mechanism of cardiac enlargement in hypertension is not entirely clear, unless there be some relation between the extra strain in hypertension to the muscular strength. We think we can tell whether the heart muscle is normal by examining it microscopically. However, it is conceivable that the heart muscle may appear normal histologically while at the same time it is weak physiologically; an inherently weak heart muscle may enlarge under normal blood pressure just as a normal heart muscle may enlarge with increased blood pressure. The fibrosis and pathological changes which we wrongly call myocarditis may result when the weak muscles have to work even against normal pressure.

Dr. Sommers, in closing: I am very grateful to the doctors for their discussion. In regard to Doctor Peppard's question concerning the possibility of coronary insufficiency to account for left ventricular hypertrophy, I can only say that the heart muscle in the two cases I presented was searched diligently for subendocardial fibrosis, which should be present if coronary insufficiency had developed. No fibrosis was present.

Dr. Barron brought up the interesting question of possible constitutional inability to withstand so-called normal blood pressures in etiology of left ventricular hypertrophy. This is a possibility which is difficult to rule out. Dr. Ben Clawson also feels that this factor may be responsible for some of the 5 per cent of Dr. Clawson's large series of left ventricular hypertrophies which had no clinical hypertension.

I should like to make one further remark concerning this paper. It would be quite unfair to Dr. Ikeda if I did not point out the fact that the pulmonary artery involvement in Case 3 was in reality quite inconspicuous and was not well brought out until special elastic tissue stains were made. Even then, only a relatively few areas were involved.

Dr. Ylvisaker, of Minneapolis, read his inaugural thesis, "Biopsy Studies of the Gastric Mucosa." Lantern slides were shown.

BIOPSY STUDIES OF THE GASTRIC MUCOSA

R. S. YLVISAKER, M.D.

Minneapolis, Minnesota

In 1945, we presented before the Medical Staff of the University of Minnesota Hospitals,[25] a review of the subject of chronic gastritis. At that time, we could find histologic data on only six cases of atrophic gastritis among the many hundreds of gastroscopic examinations which had been done previouly in the gastroscopy clinic. In other words, virtually all of the diagnoses of the various types of gastritis were based on gross decriptions of the gastric mucosa as seen through the gastroscope. We cited at that time the crying need for more histopathologic information to serve as an important foundation for these diagnoses.

Along with most gastroscopists, we have used Schindler's classification of the gastritide as a convenient outline of the gross appearance of the various forms of gastritis. This classification with descriptions was completely detailed in Schindler's original monograph "Gastroscopy."[20] This treatise, together with his later monumental work "Gastritis,"[21] has served as a tremendous impetus to the further study of gastritis as well as other conditions affecting the gastric mucosa.

Schindler, himself, has seen the need for more histologic data and has devised a method of obtaining full thickness biopsies of the gastric wall at laparotomy.[21] Unfortunately, this method does not lend itself to any large scale study of the gastric mucosa.

Hebbel[7,8,9] has continued his studies and has further clarified knowledge as to the detailed histologic appearance of gastritis as well as its incidence and its topographical distribution in otherwise normal stomachs, those with gastric and duodenal ulcer and those with gastric carcinoma. He questions the validity of the objections of Schindler, Necheles and Gold[22] to the use of surgically resected specimens for the study of gastritis.

On the clinical side, questions are being raised by many, notably W. L. Palmer and his group,[19] as to the significance of the gastritides in the production of symptoms. One senses this questioning in the, at times, rather lengthy philosophical dissertations on this subject in Schindler's monograph on gastritis.[21] In our opinion, this does not measurably detract from the wealth of factual data presented. Nevertheless, this healthy doubting has further pointed up the need for more correlation between the histologic and the gross appearance of the gastric mucosa.

Although the appearance of Benedict's operating gastroscope in 1948[1] was greeted with a good deal of caution by those interested in this field, it has gradually assumed an important position in the study of both diffuse lesions of the stomach such as gastritis and the diffusely infiltrating neoplasms, and the more localized lesions such as ulcer and circumscribed tumors. In 1950, Benedict[2] reported on the first sixty-three specimens which had been taken for biopsies without accident with his instrument. He called attention to the value of these biopsies in the differential diagnosis of gastritis and tumor and in the diagnosis and study of gastritis. This report was soon followed by that of Shallenberger, DeWan, Weed, and Reganis.[23] They report their experiences in sixty cases including cases of gastritis, gastric ulcer, malignancy, gastric polypi and marginal ulcer, detailing not only the advantages, but also the difficulties and limitations they had encountered. They concluded, among other things that the instrument is valuable and safe for general use and that indication for gastroscopy is indication for biopsy. In 1951, Benedict[3] reported further on his experience and included 203 biopsies thus far taken without accident. He did not report on his over-all experience with these cases, but detailed ten illustrative cases, in five of which positive diagnosis of carcinoma was established by biopsy and in five others diffuse gastric malignancy was excluded with reasonable certainty by gastroscopic examination and biopsy. He reported on the value of a positive biopsy finding but warned against the acceptance of a negative report especially in focal lesions. In 1953, Selesnick and Kinsella[24] reported their study of fifty-six gastroscopic examinations with mucosal biopsies in fifty patients in which only gastric mucosae that were interpreted as appearing normal or manifesting some form of gastritis were reviewed. They reported 91 per cent agreement with the pathologist in thirty-three cases showing gross evidence of gastritis, but only 60 per cent agreement where the gross diagnosis was that of a normal mucosa. In other words, the greatest disparity existed where the gastroscopist called the mucosa normal, overlooking pathology in 40 per cent of the cases. They also called attention to the fact that where a gross diagnosis of chronic hypertrophic gastritis was made, the pathologist usually found a normal mucosa.

In 1949, Doig[4] reported his use of a new flexible gastric biopsy tube devised by him. With this instrument a fragment of mucosa is drawn into a side opening in a flexible tube by suction and then cut off by a sharp blade. He found this method of value chiefly in the study of diffuse lesions such as gastritis, its main disadvantage being that it is a blind biopsy and usually reaches only one area—the greater curvature of the lower pars media. By 1952, Doig and Wood[5,6] had studied 112 cases of gastritis diagnosed by this method and compared them with groups of cases of duodenal and gastric ulcer. They found that the age of onset of both superficial and atrophic gastritis compared with that of gastric ulcer, but that the age of reporting was later for atrophic gastritis. They studied the symptomatology and course of gastritis extensively and concluded that this disease is a definite clinical and pathological entity, showing a recognizable, though variable and usually mild clinical picture. They also studied cases of subacute combined degeneration of the spinal cord and pernicious anemia and concluded that many cases without the characteristic blood picture could be diagnosed by finding true gastric atrophy on gastric biopsy.

In the United States, the method of Doig and Wood has been used extensively by Eddie Palmer.[10-18] In his clinic, the use of the suction biopsy is followed by gastroscopic examination so that he is able to see the area biopsied and study its gross appearance. In this way, he early obtained a remarkable correlation of gastroscopic and histologic findings, but some of his later reports admit to more frequent disagreement. He has studied the gastric mucosa in normal subjects, patients with duodenal ulcer, virus hepatitis, staphylococcus gastroenteritis, atrophic gastritis, postoperative gastritis, sarcoid disease, and chronic superficial gastritis. Apparently, one advantage of the suction biopsy over the biopsy taken with the Benedict operating gastroscope is that the fragment of tissue obtained is thicker and includes the muscularis mucosae and some submucosa.

The present report deals with the total gastroscopic biopsy experience in the gastroscopy clinic in the University of Minnesota Hospitals. The Benedict operating gastroscope has been used. This has been a co-operative undertaking. The gastroscopic examination and the taking of biopsies has been carried out by four different men, all well grounded in this procedure. I am indebted to Dr. J. B. Carey, Sr., Dr. James Myhre and Dr. J. B. Carey, Jr., for their part in this program. To some, this division of responsibility would appear to be a disadvantage. To us, it has been an advantage in that it affords a better over-all picture of the gross gastroscopic interpretation. Furthermore, in the few cases where more than one of the four persons has examined the same patient at different times, the agreement in interpretation has been remarkable. The histopathological studies were made by Dr. Robert Hebbel of the Department of Pathology of the University of Minnesota, who has had a large experience in the microscopic study of the gastric mucosa.[7,8,9] We feel that we have been fortunate in having Dr. Hebbel's co-operation and advice in this work.

A few words of explanation in regard to the instrument used might be pertinent. Its value in affording an opportunity to obtain biopsies of the gastric mucosa under direct vision is obvious. Certain disadvantages should be mentioned. In the first place, the instrument is definitely more cumbersome for the operator to handle and for the patient to tolerate than the Schindler instrument. This disadvantage can be overcome by being more deliberate with the examination. In the second place, it seems that the objective lens becomes more easily smudged and is less effectively "wiped off" on the posterior wall of the stomach, necessitating occasionally the removal of the instrument for cleaning and re-introduction, a procedure which in our opinion should be avoided. In the third place, it has been difficult at times to direct the biopsy forceps to the area of involvement. In a more or less diffuse lesion such as gastritis, this difficulty has not become too important. However, we have not been successful in obtaining biopsies from the antrum. On only one occasion did the biopsy specimen reveal antral mucosa, although the attempt to obtain specimens from the antrum has been made often. A few specimens showed the so-called transitional type of mucosa.

It is well to mention, too, that the specimens which we have obtained are often thin, including only parts of the mucosa without the muscularis mucosae. Then, too, it is not possible, even with several biopsies of the same stomach (we have made up to four) to study the whole gastric mucosa. We are studying merely samplings of the mucosa. The fact that the samplings are taken under visual control helps overcome this difficulty and affords an opportunity to study at least the area visualized.

Most of the examinations detailed in this report have been made during the past year, a few prior to that time. Although the series includes some focal lesions other than gastritis, it has been the distinct purpose of each investigation to study the gastric mucosa in its entirety. This study can, therefore, properly be called a study of the gastric mucosa as related to the appearance and incidence of gastritis. Except in a few instances, the operating gastroscope was used routinely during the past ten months. Since the series is small, we wish to designate this a preliminary report.

Eighty-four patients were examined. On one occasion the gastroscopic examination was unsatisfactory; in eight others, the biopsies were unsatisfactory; leaving seventy-five cases with satisfactory gastroscopic examinations and biopsies. In a few other cases, the gastroscopic examination was incomplete in some details, but enough visualization of the mucosa was obtained to make the correlation with the histologic findings satisfactory. There were eleven other individual biopsies which were unsatisfactory, but in these cases multiple biopsies had been taken, leaving other satisfactory biopsies in each case. Two patients were examined twice with biopsies, making a total of seventy-seven satisfactory examinations in seventy-five cases. There were thirty examinations with one biopsy each. Forty-seven with two to four biopsies each. Altogether 141 satisfactory biopsies were taken.

Two complications should be reported. These were complications of the gastroscopic examination, not of the biopsy taking as such. One patient developed a mediastinitis with subcutaneous emphysema of the lower cervical region. This subsided on antibiotic therapy after a short febrile course. The other patient developed severe esophageal pain and dysphagia, sufficient to require hospitalization. Protective anbtiotic therapy was given, and the symptoms subsided after a brief afebrile course. Both of these cases had required removal and re-introduction of the gastroscope.

Histologically, each sample was classified as showing normal mucosa, superficial gastritis, or atrophic gastritis. In order to be classified as normal, the sections were required to show intact glandular elements with a normal component of specific cells and little or no cellular infiltration in the stroma. Those with superficial gastritis showed excessive cellular infiltration in the

TABLE I. DISTRIBUTION OF GASTRITIS ACCORDING TO AGE AND GASTRIC CONTENTS IN WOMEN

Age	Total Cases	Cases with Free HCl				Cases with Achlorhydria				Cases with No Gastric Analysis			
		Normal	Gastritis			Normal	Gastritis			Normal	Gastritis		
			Superficial	Atrophic	Both*		Superficial	Atrophic	Both*		Superficial	Atrophic	Both*
20 to 29	1		1										
30 to 39	3	2	1										
40 to 49	4	2	1							1			
50 to 59	7	2	1					3		1			
60 to 69	5	1					1	1				1	
							1 ade	nocarcin	oma				
70 to 79	3		1					2					
80 to 89	1							1					
Total		7	5	0	0	0	1 1 ade	7 nocarcin	0 oma	2	0	1	0
Total	24	12				9				3			

*This signifies one type of gastritis in one sample and the other type in another sample from the same mucosa.

TABLE II. DISTRIBUTION OF GASTRITIS ACCORDING TO AGE AND GASTRIC CONTENTS IN MEN

Age	Total Cases	Cases with Free HCl				Cases with Achlorhydria				Cases with No Gastric Analysis			
		Normal	Gastritis			Normal	Gastritis			Normal	Gastritis		
			Superficial	Atrophic	Both*		Superficial	Atrophic	Both*		Superficial	Atrophic	Both*
20 to 29													
30 to 39	4	3			1								
40 to 49	9	4		1		1		1		2			
50 to 59	10	3	2	1				1		1		2	
60 to 69	21	9		4	1	1		5	1				
70 to 79	7	1		2		1		2	1				
80 to 89	2			1						1			
Total		20	2	9	2	3	0	9	2	4	0	2	0
Total	53	33				14				6			

*This signifies one type of gastritis in one sample and the other type in another sample from the same mucosa.

TABLE III. TOTALS FOR MEN AND WOMEN

Total Cases	Normal	Superficial Gastritis	Atrophic Gastritis	Both*	Adenocarcinoma
77 (100%)	36 (46.75%)	8 (10.39%)	28 (36.36%)	4 (5.20%)	1 (1.30%)

Total	Cases with Free HCl				Cases with Achlorhydria				Cases with No Gastric Analysis			
77 (100%)	45 (58.44%)				23 (29.87%)				9 (11.69%)			
	Normal	Gastritis			Normal	Gastritis			Normal	Gastritis		
		Superficial	Atrophic	Both*		Superficial	Atrophic	Both*		Superficial	Atrophic	Both*
	27 35.06%	7 9.09%	9 11.69%	2 2.60%	3 3.90%	1 1.30% 1 ade	16 20.77% nocarcinom	2 2.60% a	6 7.79%	0 0%	3 3.90%	0 0%

*This signifies one type of gastritis in one sample and the other type in another sample from the same mucosa.

stroma but no change in the glandular elements including no significant loss of specific cells. We do not feel that any one case of superficial gastritis can be stated as being acute or chronic from its histologic appearance. Repeated biopsies over a period of time would be needed to determine this. Those with atrophic gastritis included any that showed loss or change in the glandular elements, i.e., loss of specific cells and/or total glandular substance, and/or metaplasia of the glandular elements. No attempt was made to distinguish between atrophic gastritis and gastric atrophy nor do we believe any clear-cut distinction exists.

Tables I, II and III show our over-all experience based on histologic diagnoses. No attempt at statistical analysis has been made because the series is small. Only certain obvious trends can be shown. Percentage figures are given for the larger totals. There were fifty-three men and twenty-four women. Grouped according to ages, there were four men from thirty to thirty-nine, nine from forty to forty-nine, ten from fifty to fifty-

TABLE IV.

Histology	Gastroscopy	No. Cases
Normal 36 cases	Normal	21
	Superficial gastritis	0
	Atrophic mucosa	1
	Hypertrophic mucosa	13
	Probable carcinoma	1

nine, twenty-one from sixty to sixty-nine, seven from seventy to seventy-nine, and two over eighty. Among the women, there was one between twenty and twenty-nine, three between thirty and thirty-nine, four between forty and forty-nine, seven between fifty and fifty-nine, five between sixty and sixty-nine, three between seventy and seventy-nine, and one over eighty.

Of the total group, forty-five had free HCl in their gastric secretion, twenty-three had achlorhydria, and nine had no gastric analysis performed. All the achlorhydrias were histamine achlorhydrias, many having repeated determinations. Histologically, thirty-six showed normal mucosae, eight superficial gastritis, twenty-eight atrophic gastritis, four superficial gastritis on one or more samples and atrophic gastritis on other samples from the same stomach and one had adenocarcinoma on a single biopsy from a tumor. The tables clearly demonstrate the usual preponderance of atrophic gastritis in the group with achlorhydria. Whereas, those with free HCl in their stomach contents showed twenty-seven normal mucosae on biopsy and only eleven with atrophic gastritis (including two with both superficial and atrophic), those with achlorhydria showed only three normals but eighteen atrophic gastritides. Seven out of eight of those with superficial gastritis had free HCl in their stomach contents. Only two out of the eight cases of superficial gastritis were men, whereas the latter accounted for twenty-four out of thirty-two cases with atrophic gastritis. None of the women with free HCl showed atrophic gastritis and none of the women with achlorhydria had normal mucosae. There was one case of proven pernicious anemia and one of subacute combined degeneration of the spinal cord with a normal blood picture. Both showed atrophic gastritis on gastric biopsy.

Grouped according to ages, fifty-six (72.7 per cent) of the seventy-seven patients were over fifty years of age. Of the thirty-two patients showing evidence of atrophic gastritis (including the four which showed both superficial and atrophic gastritis) only one was under forty years of age and three under fifty; or 90.6 per cent were over fifty years of age. Conversely, of the fifty-six persons in the series over fifty, 51.8 per cent had evidence of atrophic gastritis and 60.7 per cent had evidence of one type of gastritis or the other or both, while only 14.3 per cent of those under fifty showed atrophic gastritis and 28.6 per cent one type of gastritis or the other or both. This high incidence of atrophic gastritis in the older age group agrees with the findings of Hebbel[8] and others.

Correlation of gross gastroscopic findings with the histologic findings brought out some very interesting results. Table IV shows the gastroscopic appearance in thirty-six cases with normal histology.

TABLE V.

Histology	Gastroscopy	No. Cases
Superficial gastritis 8 cases	Normal	3
	Superficial gastritis	0
	Atrophic mucosa	1
	Hypertrophic mucosa	0
	Small raised area	1
	Edematous, hyperemic mucosa	2
	Increased high-lighting	1

TABLE VI.

Histology	Gastroscopy	No. Cases
Atrophic gastritis 28 cases	Normal	8
	Superficial gastritis	0
	Atrophic mucosa	14
	Hypertrophic mucosa	1
	Edematous mucosa with multiple, small, irregular ulcers	1
	Rim of gastric ulcer	1
	Postoperative gastritis	1
	Polypi with red mucosa	1
	Probable carcinoma	1

The cases with hypertrophic mucosae will be commented upon later. As it will be seen, these cases should probably henceforth be classified as normal mucosae. The case of probable carcinoma turned out actually to be carcinoma and was missed by biopsy. This case points up the warning of Benedict previously mentioned, that a negative biopsy in a focal lesion should not be accepted as a final diagnosis. The one case diagnosed as atrophic mucosa, then, becomes the only error in gastroscopic diagnosis in this group. If allowance is made for the possibility that the atrophic area might have been missed on biopsy (even though three areas were biopsied in this case) the correlation between gross gastroscopic and histologic findings in this group becomes remarkable.

Table V shows the gastroscopic appearance in eight cases diagnosed as superficial gastritis histologically.

It will be noted that no diagnoses of chronic superficial gastritis were made on gross appearance in this series. There were probably two reasons for this. In the first place, as time went on there was an agreed upon tendency among the four coöperating gastroscopists to describe what was seen rather than to make diagnoses. In the second place, none of the cases in this series showed the mucosal exudate or sticky mucus thought to be characteristic of chronic superficial gastritis. In this group, then, there were four mistaken diagnoses and four cases in which a raised, white area, the hyperemic edematous mucosa, and the increased high-lighting were not considered sufficient evidence by the gastroscopist to make a definite diagnosis, but proved to be the only surface indications of an underlying, superficial gastritis.

Table VI shows the gastroscopic appearance in twenty-eight cases of atrophic gastritis.

It will be noted that there were fourteen cases in which the gastroscopist definitely agreed with the histologic diagnosis, eight cases in which he definitely

TABLE VII.

Histology	Gastroscopy	No. Cases
Superficial and atrophic gastritis on separate samples	Hypertrophy anterior wall and greater curvature, atrophy posterior wall	1
	Pseudopolypi antral floor	1
	Atrophy anterior wall	1
	Slight atrophy	1

TABLE VIII.

Gastroscopy	Histology	No. Cases
Normal 26 cases	Normal	14
	Superficial gastritis	3
	Atrophic gastritis	9
	*Both	0
Atrophic mucosa 19 cases	Normal	1
	Superficial gastritis	2
	Atrophic gastritis	14
	*Both	2
Hypertrophic mucosa 12 cases	Normal	11
	Superficial gastritis	0
	Atrophic gastritis	1
	*Both	0

*This signifies one type of gastritis in one sample and the other type in another sample from the same mucosa.

missed the diagnosis and called the mucosa normal, one case in which he called the mucosa hypertrophic (the only case of hypertrophic mucosa showing gastritis histologically), one case with multiple small, irregular ulcers—probably a true case of ulcerative gastritis, one case in which the biopsy was taken from the rim of a gastric ulcer, one case of postoperative gastritis, one case with benign polypi and a very red mucosa, and one case thought to be carcinoma by the gastroscopist. The rather large number of normals in this group tends to confirm the finding of Selesnick and Kinsella[24] and others that the greatest disparity between the gastroscopic appearance and histologic findings lies in the gastroscopist missing gastritis when it is definitely present in a fairly large number of cases.

The case thought to be carcinoma was one in which the patient had bone lesions interpreted as being metastases, but who died elsewhere without an autopsy being performed.

Table VII indicates the gastroscopic findings in four cases found to have both superficial and atrophic gastritis histologically on different samples by biopsy. It will be seen that various types of gastritis likewise were found gastroscopically.

If we reverse the situation and investigate the various histologic diagnoses when definite gastroscopic diagnoses had been made, equally interesting results are obtained. Table VIII tabulates these results. It will be noted that only those cases are tabulated in which a definite diagnosis was made gastroscopically.

Equivocal diagnoses or those with merely descriptive accounts without a definite gastroscopic diagnosis are not included. Here again, when a diagnosis of a normal mucosa was made gastroscopically, a considerable number of atrophic gastritides were overlooked; whereas, when a diagnosis of atrophic mucosa was made, a good correlation with atrophic gastritis histologically is seen. Again, the cases which were diagnosed as showing hypertrophic mucosa all proved to be normal histologically except one. It should be explained in this connection that the fragments of tissue obtained by this method of biopsy are often thin and the pathologist was unable to measure the full thickness of the mucosa in the great majority of cases. A true evaluation of the presence or degree of hypertrophy was, therefore, not possible. However, it can be stated that none of the sections in the cases designated as hypertrophic by the gastroscopist showed any of the other evidences of gastritis histologically, nor was there any visible piling up of the glandular elements. We shall have to agree with Selesnick and Kinsella,[24] therefore, that on the basis of our findings, cases with the usual evidences of hypertrophy gastroscopically should be called normal and that this finding should be considered a normal variation probably related chiefly to the functional state of the muscularis mucosae. It might appear that cases showing hypertrophy gastroscopically are more apt to be normal histologically than those that are normal gastroscopically.

TABLE IX. DISTRIBUTION OF GASTRITIS IN ULCER CASES

Type of Ulcer	Total	Type of Mucosa				
		Normal	Superficial Gastritis	Atrophic Gastritis	Normal & Gastritis	Mixed Gastritis
Duodenal	9	6	0	1	2	0
Gastric	8	2	0	4	2	0
Gastric and duodenal	2	2	0	0	0	0
Pyloric	5	3	0	1	1	0
Duodenal and Stomal	1	1	0	0	0	0
Stomal	1	1	0	0	0	0
Total	26	15	0	6	5	0

Certain other features of this series should be mentioned. There were twenty-six cases of peptic ulcer: nine duodenal, eight gastric, two gastric and duodenal, five pyloric, one duodenal and stomal, and one stomal. The five pyloric ulcers included those which might be either just pre-pyloric or post-pyloric, most cases being difficult to determine roentgenologically.

Table IX shows the type of mucosa found histologically in the body of the stomach in the various types of ulcer.

The group is too small to make any comments but, in general, the findings agree with those of Hebbel[7] that gastritis is common in the body mucosa in cases with gastric ulcer and unusual in cases with duodenal ulcer.

In regard to the distribution of the gastritis, in the case of atrophic gastritis we shall have to agree again with Hebbel[8] that the lesions show a wide range between focal and diffuse. The only exception, as he points out, lies in those cases in which the greater curvature is involved. When this is the case, according to him, the lesion is more apt to be diffuse. In our series, only seven cases, among those with multiple biopsies, were found which showed atrophic gastritis on the greater curvature. In all of these, atrophic gastritis was found in all other areas sampled. On the other hand, atrophic gastritis was found on the lesser curvature in nine cases, and in only five of these were all other areas similarly involved.

Table X indicates the types of symptoms complained of in the cases without ulcer, gallbladder disease, or cancer. Again, the series is too small to draw any conclusions. The only symptoms which seem to be more common in cases with gastritis than in the normals are pain after eating and flatulence. On the other hand, the asymptomatic cases fall almost entirely in the gastritis group. In other words, no definite symptom complex for gastritis is present.

Summary and Conclusions

This report has presented a review of the literature in regard to histologic findings by gastric biopsy in gastritis with special reference to its correlation with the gastroscopic appearance, and has detailed the total gastroscopic biopsy experience at the University of Minnesota. In the latter work, the operating gastroscope of Benedict was used. Some of its advantages and disadvantages as well as those of the type of biopsy obtained have been pointed out. Seventy-seven gastroscopic examinations with 141 biopsies were done.

Although the total number of cases is not sufficient to allow for any final conclusions, some very definite trends are brought out.

1. The well-known increased incidence of atrophic gastritis with age and in cases with achlorhydria is confirmed.

2. The correlation of gastroscopy with histology in cases which proved to be normal histologically is found to be excellent if cases showing hypertrophic mucosae gastroscopically are included with the gastroscopic normals. The finding of normal mucosae microscopically in those with hypertrophy gastroscopically agrees with the finding of Selesnick and Kinsella. We are inclined to agree with their suggestion that mucosal hypertrophy is due to a functional derangement of the muscularis mucosae and does not represent a true gastritis.

3. A definite disparity in the correlation of gastroscopy with histology in cases showing atrophic gastritis histologically is confirmed. In other words, no surface evidence of an underlying atrophic gastritis was found in a considerable number of cases. Conversely, when atrophic mucosa is seen gastroscopically, atrophic gastritis will be found histologically in a high percentage of cases.

TABLE X. SYMPTOMS IN CASES WITHOUT ULCER, GALLBLADDER DISEASE, OR CANCER

Symptoms	Normal	Gastritis
Pain after eating	3	7
Flatulence	1	4
Asymptomatic	1	8
Asymptomatic—(post-gastrectomy)	0	2
Vague epigastric distress	3	3
Abdominal pain other than epigastric	4	6
Nausea and/or vomiting	4	2
Typical ulcer distress	2	3
Weight loss	1	3
Nervousness	0	2
Spells of violent epigastric pain	1	0
Pyrosis	0	1
Weakness	2	1
Alternating constipation and diarrhea	0	1

4. Superficial gastritis shows a comparatively low incidence and a varied gastroscopic picture in this series. Surface exudate or sticky mucus does not appear to be an essential finding.

5. Hebbel's finding of the unusual occurrence of atrophic gastritis in the body mucosa in cases of duodenal ulcer as well as its common occurrence in cases with gastric ulcer is supported in this series.

6. Likewise, Hebbel's finding of a wide range indistribution of gastritis from focal to diffuse, and the definite tendency for it to be diffuse when the greater curvature is involved, finds support.

7. No characteristic symptom complex for gastritis is found.

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Discussion

Dr. James B. Carey, Minneapolis: I appreciate the opportunity of participation in this study with the Benedict biopsy gastroscope. When the flexible gastroscope was first introduced in this country by Benedict in Boston in 1934, then by Schindler in Chicago in 1935, and in this territory in 1936, the classification of gastritis suggested by Schindler was accepted, i.e., superficial gastritis, atrophic gastritis, hypertrophy gastritis, and gastritis of the postoperative stomach. This classification of Schindler's was not unequivocally substantiated by direct histologic evidence. It was based on objective gastroscopic appearance of the gastric mucosa. The only substantiation was by histopathologic studies of autopsy or surgical material from individuals different from those examined gastroscopically.

Two methods for obtaining biopsies from the mucosa of the stomach are now being used; one is with the flexible instrument in which a biopsy forceps is incorporated and by which the part of the stomach from which the specimen is obtained may be seen; the other method is that of Doig and Wood, of Australia, which is blind. A tube with an open end, a section device, and a cutting obturator is passed into the stomach. The specimen is usually of the greater curvature of the stomach opposite to the orifice of the esophagus. Later the part of the stomach from which the biopsy was taken may be identified through the diagnostic gastroscope. Obviously, two passages of instruments is required.

Before biopsies of the stomachs of living patients were possible, some of us were beginning to be a little skeptical about superficial and hypertrophic gastritis. The small number of patients presented by Dr. Ylvisaker stresses this doubt. The original concept, based on Faber's work, was that superficial gastritis might accompany several infectious diseases, and, if repeated, could be the basis for permanent atrophic gastritis. Faber's specimens were obtained after introducing formalin solution into the stomach as soon as possible after death in order to inhibit postmortem disintegration. These studies supported the gastroscopic diagnoses of superficial and atrophic gastritis. But interpretation of changes apparently indicative of hypertrophic gastritis has been uncertain. The latest work on this subject by Col. Eddy Palmer of Walter Reed Hospital does not support the concept of hypertrophic gastritis as described by gastroscopists as pavement mucosa, cobblestone, thick, and other terms. Palmer did say, however, that the gastroscopic appearances were truly seen, but that the clinical significance of such observations was dubious.

The other question is whether all changes called atrophic gastritis are actually gastritis, that is, inflammatory. Some of the changes may be nutritional or senile, related to degeneration of other parts of the body. Also, the gastric mucosa may differ from person to person, as do other body structures; for instance, the skin. In other words, what may appear to be atrophy or hypertrophy may actually be normal for the particular individual.

I believe that, from knowledge derived from biopsies of gastric mucosa, the orderly classification of Schindler must be modified. For instance, English and Australian workers have virtually abandoned the term hypertrophic gastritis.

Dr. L. G. Rigler, University of Minnesota: It is very difficult and possibly unwise to make a diagnosis of gastritis from x-ray examination of the stomach. I did see a case of true hypertrophic gastritis just this morning—a seventy-two-year-old doctor who came into the hospital with a very severe gastric hemorrhage. Dr. Varco did a subtotal resection of his stomach. He had very large folds; not only that, but the gastric glands penetrated well into the muscularis although there were no actual points of bleeding. Many of the cases with large folds are not gastritis, simply redundancies of the mucosa.

Dr. Ylvisaker, in closing: I wish to thank Doctor Carey and Doctor Rigler for their discussions. We plan to carry on this study over a period of years with the hope especially of obtaining more information in regard to the development of gastritis. Doctor Hebbel tells me that he is interested in this phase of the study. In regard to the matter of hypertrophic mucosa, while we have not found any true hypertrophic gastritis in our series, we feel quite certain that there are cases of so-called giant rugae which should be classified as true hypertrophic gastritis. These are not at all common.

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

Meeting of May 12, 1954

This meeting, at which the retiring President read his Address, was held at the Minneapolis Club on Wednesday evening, May 12, 1954. Dinner was served at 7 o'clock and the meeting was called to order by the President, Dr. Regnier, at 8:15 p.m.

There were fifty-two members and five guests present.

No business was transacted at this meeting, and minutes of the April meeting were not read.

RETIRING PRESIDENT'S REMARKS

Dr. E. A. Regnier: My fellow members of the Academy, you have done me a great honor in electing me your president in the past year. For this privilege, I assure you my sincere gratitude for your respect and confidence. It has been a pleasure to serve, and its reward, namely, your confidence, I shall always cherish. At this time I want to thank the members who have served with me on the various committees. The co-operation of the Executive Committee, the Membership Committee, the Program Committee was most gratifying. I want to commend particularly the Program Committee for furnishing the excellent programs which we have enjoyed throughout the past year. My sincere thanks to all of you, and particularly to our secretary, Dr. Robert Priest.

This Society is now completing its seventy-seventh year, and it has reached a stage of maturity, whereby it should occupy a position of great prestige in this community. Your present officers have served an added six months due to the change voted by the Academy last December. The presidential meetings are to be held alternately at the Minneapolis Club and the Minnesota Club.

To our new president, I extend greetings and best wishes for continued progress toward our objectives and give assurance of my full co-operation.

As you know, we have increased our membership to a capacity of 125 men when the roster is filled. This, I believe, is a far-reaching step and in my opinion should be further amended. It is my opinion there should be no limit to the number of memberships in this Society, if the candidates could meet the requirements of the organization.

The Academy derives its strength, not from a numerical role, but from intellectual and cultural qualities and moral attainments. We should be truly representative of the medical fraternity before society. In other words, the Minnesota Academy of Medicine should occupy and deserve a high position in public relations and medical ethics throughout the Northwest.

One of my recommendations to this Society is that next year's Executive Committee sponsor a committee on public relations and inter-medical relations. This committee should be very carefully selected, and it should concern itself with medical education, inter-relations of the public with medicine and promotion of more wholesome respect and co-operation between medicine, as practiced by specialists and general practitioners. This committee should also concern itself with socialistic trends in medicine and should be ready and free to voice an opinion in rebuke or approval of any group from the level of a county society to the American Medical Association. If every state had an organization of men with mature judgment and the fortitude to function fearlessly and justly, we would not have to apologize or alibi for endorsement of the Bricker Amendment in Congress, by representatives of organized medicine. And I think if such a committee was supported by the opinion of the Society, we could forestall the possibility of a pseudo-scientist going unchallenged in exposing the indecency of his mind under the guise of giving a scientific report.

This Society has been called a dining club of professional men. True, this caption covers one phase, and a very essential one, of our purpose but surely only a limited one. The social and convivial aspects of this Society are second to none and I am sure, cherished by every member. However, our fundamental purpose is to band together to promote higher standards of medical ethics and practice of medicine through education, mutual and fraternal assistance, to the end that our noblest instincts will be guided by the Light of Truth.

It is true, we have very limited funds, but ways and means should be implemented to increase these funds. I submit to you that no member of this Society would be hurt by an annual assessment of $10 to $25 toward raising a scholarship fund. This assessment should be one which would raise $1200 to $1500 annually. It could be augmented by voluntary donations and by funds given as memorials by friends of our departed members if our members were solicitous of such. I am leaving a memorandum with our secretary to the effect that these matters be placed before the Society through the Executive Committee early next year. The members of the scholarship and educational committee could make recommendations to the Academy and the entire membership could decide on what fellowship to sponsor and how often this fellowship could be supported. It would be known as the Minnesota Academy of Medicine Fellowship at the University of Minnesota. Such a small sacrifice by each member would accrue to the honor of our Society and give its individual members a sense of pride and justification in an act accomplished, in fostering not only an excellent dining club, but a legacy to posterity in a scholarship fund. By establishing and supporting a fellowship at the University of Minnesota the influence of the Academy would become a living memorial to its founders. Our compensation would be the realization of the furtherance of the art and practice of a noble profession. An assessment sufficient to raise $1,000 plus the interest on our bonds plus memorial contributions and voluntary donations should support a fellowship every three years until our membership grows to a point where a year or biennial endowment could be

(Continued on Page 289)

History of Medicine in Minnesota

THE PIONEER DOCTORS OF CHIPPEWA COUNTY
PRIOR TO THE YEAR 1900

LEON G. SMITH, M.D.

Montevideo, Minnesota

(Continued from January, 1955, issue)

From a civic standpoint, his most notable achievement was that of inducing the Milwaukee Railroad to move its division headquarters to Montevideo. It is generally acknowledged that it was largely due to his foresight, energy and persistence in working upon the railroad officials that this was finally accomplished in July, 1893. In consequence, Montevideo occupied one of the most important railroad positions of any town in western Minnesota. The round-house, car-repair shops, freight yards, stock yards and dispatcher's offices brought many families to the town, and supplied a payroll which was a tremendous factor in its growth and development.

From a combined civic and medical standpoint, almost as notable an accomplishment was the campaign to build the Montevideo Hospital. This would probably have been built eventually in any case, but Dr. Rogers is given the credit for being the instigator of the project, and the moving force in carrying it to its completion in 1910.

He did not live continuously in Montevideo. For a few years he practiced in Minneapolis, in 1890 becoming associated once more with his old friend and preceptor, Dr. Hoegh, who had moved there from La Crosse.

Dr. Rogers was always good newspaper copy. Quoting the *Montevideo Leader* of March 25, 1887: "A series of articles, published by the *Commercial* man, uncomplimentary to Dr. C. E. Rogers, resulted in the latter soundly slapping the former's face last Saturday night." He was undoubtedly a better than average surgeon for his day, and as a result was widely called in consultation. The newspaper files, during these years, alluded to these calls frequently and described several of the difficult operations that he performed. This is not noted in any derogatory sense, as news stories about doctors and their achievements seem to have been entirely permissible in those days.

Even when he was practicing in Minneapolis was this true. I quote the *St. Paul Dispatch* of May 18, 1894: "A dislocated neck, twisted back in to proper shape, and medical science has another remarkable event to recall.

"One of the most interesting incidents, in the experience of medical men of the Northwest, happened in Minneapolis yesterday. Indeed, it will be numbered among the remarkable events in the medical history of the world. A man whose neck had been dislocated by a fall, causing his head to be twisted almost one-fourth around, and with a forward tilt, walked into the office of Dr. C. E. Rogers, and asked to have the kink straightened out, not seeming to realize the serious character of his case. It is rare that such a dislocation occurs without paralysis, due to pressure on the spinal cord, and the physician saw he had a truly wonderful case before him. The patient, Thos. H. Golrick, of Keegan's Lake, was put under an anaesthetic and the neck gradually twisted back to its right position. As it sprang back into place, a distinct click was heard, and, suddenly, respiration ceased. The head fell to the other side, the neck became perfectly lax, and general collapse seemed to have taken place. Golrick's position, however, was quickly changed, his head raised and his tongue held so it could not obstruct the air passage. Quickly, respiration began again, and he slowly came from under the influence of the anaesthetic. When he was able to talk, he could tell nothing of the terrible experience he had been through, and was quite surprised when it was related to him. After a few minutes, he started for home, apparently as well as before the accident."

In 1897, Dr. Rogers bought part ownership in the Riverside Sanatorium for Tuberculosis and Lung Diseases, located in St. Paul, where the "pneumo-chemic system of treatment for consumption, bronchitis, catarrh, asthma and hay-fever" was used.

In July, 1898, he and his family moved back to Montevideo where he spent the rest of his life.

In 1904, he nearly lost his life. Again I quote from the *Montevideo Leader* of January 15: "Last Saturday night, Dr. C. E. Rogers went home, feeling very poorly. Next day, he was considerably worse, the indications pointing strongly toward appendicitis. Sunday night he suffered a more serious attack, which confirmed the diagnosis of Dr. Zimbeck and Dr. Herbert Rogers. Dr. Hoegh was asked to come up from Minneapolis on the next train, and Dr. Stratton of Granite Falls, Dr. Titus of Sacred Heart and Dr. La Rue of Appleton were also called. All came except Dr. La Rue, who did not receive word in time. Monday morning, Dr. Hoegh performed an operation, assisted by the other physicians present. It was found to be a very serious and complicated case, requiring an operation also for hernia. The operation was successfully performed, however, and the patient got along well until Wednesday morning, when he had a bad time, due to coughing and vomiting. That evening he was very low, indeed, but the symptoms grew more favorable through the night and yesterday, and his friends now have hopes for his complete recovery." And recover, he did.

Dr. and Mrs. Rogers had six children, one son and five daughters, of whom three, including twin girls, died in infancy or childhood. The son, Herbert W. (or Bert, as he was always called) attended the Minneapolis and Montevideo public schools and studied medicine at Northwestern University in Evanston, Illinois, joining his father in practice in 1903. Only three years later, he died as the result of a ruptured appendix.

As was to be expected, the loss of his only son, reared in the atmosphere of a doctor's home, trained in the study of medicine, and associated with him for three years of practice, was a terrific blow to Dr. Rogers. However, he carried on his work as usual and it was after Bert's death that he successfully pushed to completion the hospital project.

In 1911, Dr. Carl M. Johnson, a young doctor, just out of the University of Minnesota Medical School and a year's interneship in St. Barnabas Hospital in Minneapolis, became associated with him.

Upon December 3, 1912, his death occurred quite suddenly, from "angina pectoris." His wife survived him for several years, and his two daughters, Gertrude Titus and Jean Hubbard, are now living in San Diego, California.

One last quotation about this remarkable man taken from the *Montevideo*

Leader of December 6, 1912: "In the death of Dr. Rogers, our community loses one of its most worthy and public-spirited citizens, the medical profession one of its most competent and successful practitioners, and his family and friends a loving, generous and whole-souled husband, father and friend."

Shortly after Dr. Rogers came to Montevideo, the first Medical society was organized. September 28, 1883, a news item stated: "The physicians along the line of the H. and D. (Hastings and Dakota was the name of this division of the Milwaukee Railroad) met at Montevideo September 13 and organized a society called the Western Minnesota Medical Association, such society to meet again at Montevideo December 4, at 9 A.M." The records of this society must have been lost, including the roster of its members, and it is not until 1903 that there is any further mention of a medical organization. At that time Chippewa, Lac qui Parle, Yellow Medicine, and Renville Counties joined and formed the Camp Release District Medical Society as a component group of the Minnesota State Medical Association. The earlier records of this society have also been lost, so, at least locally, no roster of the charter members is available.

In 1883, **Dr. Galen D. Moyer** located in Montevideo, qualified and licensed both as a dentist and a physician. He was born in Sanborn, New York, and received his dental education in the College of Dentistry of the University of New York, graduating in 1879. He went to Pau, France, after graduation, being associated for a year there in dental practice with a Doctor Edwards. He returned to the United States in 1880, and studied medicine at the New York College of Medicine, graduating in 1883. His older brothers, Lycurgus R. and Lloyd G. Moyer, had come to Montevideo several years earlier and had taken a prominent part in starting and building up the new town. Since two sisters had also moved here, it was the natural thing for Dr. Moyer to follow suit immediately after graduation.

While he was carried in the records of Polk's Medical Register for many years as a Doctor of Medicine, all his professional cards and the news items about him during this period indicate that he actively practiced dentistry, but did no medical work except in the role of anaesthetist. He married a teacher in the Montevideo schools, Harriet Page, April 29, 1891, to whom two children, Margret and Galen Dana, were born.

He moved to Minneapolis in 1911, though a year or two later, his name is noted as vice-president of the V. B. Dental Club of St. Paul. Shortly thereafter he moved to Wisconsin, having his home in Spring Brook, but his office in Hayward, where he practiced until retirement in 1938. He then moved back to Chippewa County, buying the small farm across the river from Granite Falls where he died in 1940.

His daughter, Margret, now Mrs. George Halpin, lives in Minneapolis and is the owner of the well-known and exclusive women's apparel store, "Harolds."

In the late eighties, two new names appear, but so briefly as to merit small mention only. In the *Montevideo Commercial* of August 27, 1886, **Dr. Berens** was listed as "Physician, Surgeon, and Accoucheur," but this is the only time his name appeared. In issues of the same paper from January 7, 1887, to March 18, 1887, was a professional card reading **"Mrs. Dr. J. E. Hull**, Physician and Midwife," the wording of which would indicate she was an unlicensed practitioner.

In this connection, I feel some mention should be made of the Indian shows of the 1880's and 1890's. Many people waited for the annual visit of these shows and combined the pleasure of watching their entertainment with the business of laying in a year's supply of the various liniments for man or beast, and other medications offered. Probably the best known of these itinerant troupes in Western Minnesota was the Kickapoo Indians.

They were from a Kansas and Nebraska tribe, and the euphonious sound of the name Kickapoo, made it particularly desirable as the title of a medicine show or the name of a tonic.

Dr. Wallace Jason Collins was the next regular physician upon the local scene, and, though his stay was for only a few years, he seems to have developed a rather large practice in that short time. His early history is not very complete, but information obtained from his step-daughter, would indicate that he was born in Gardner, Maine, about 1850. She remembers hearing that his father was a sea-captain. He graduated from Bowdoin College in Brunswick, Maine, in 1886, and was licensed in Minnesota March 25, 1887. His first location was in Dawson, Minnesota, but he moved to Montevideo July 20, 1888.

One of his first patients here was Joseph Melchior, who died in September of that year. The acquaintance, made with Mrs. Melchior at the time, developed further because in the *Montevideo Leader* of April 10, 1891, we find the following: "Dr. Wallace J. Collins and Mrs. Mathilda Melchior were matrimonially united Thursday evening last week. The event was a quiet one, and the happy couple took the morning train to the Twin Cities, where they passed a few days. They are now at their home on Third Street."

Newspaper items report his having "operated upon a young man named Ekstrom for hare-lip, with a good result, performing the operation in the Sparta House."

There is also an account of an accident which was a frequent one in those days: "Jas. Franzen of Appleton sustained a terrible and perhaps fatal accident. In sliding off a grain stack, he struck on the handle of a pitch-fork, the handle perforating his rectum and bowel. Dr. Collins was called and took him at once to St. Anthony Hospital in Minneapolis."

Dr. Collins' marriage broke up, with his wife leaving him in 1894. Late in 1895 his card disappeared from the Montevideo papers. He went back East, and his named appeared in Polk's Register from Gardner, Kannebeck County, Maine, in 1896. From 1903 he was listed from Westfield, Massachusetts until his death there, November 23, 1932, from angina pectoris.

Dr. John C. Tillman lived in Chippewa County for several years. He never practiced medicine here, though he did in Germany. The following was his obituary: "Died, at Montevideo, Minnesota, June 11, 1896, Dr. John C. Tillman, aged 82 yrs., 11 mos., 24 days. He was born at Colon, (probably Cologne) on the Rhine, January 19, 1813. He graduated from the University of Bonn, served in the German army, and practiced medicine in Germany for fifteen years, before coming to the United States in 1854. He lived, for a long time, in St. Peter, Minnesota, but for the past seventeen years, has lived in or near Montevideo. He has been married twice, and leaves a widow, with six of her children and two by his first wife."

C. S. (Spurge) Tillman was his son and lived most of his life in Montevideo. Along with his other work, he prepared and dispensed Tillman Oil, which was a rather famous medication in this area in the early part of this century. Undoubtedly prepared according to a formula of his father's, it was a powerful irritant, probably composed mostly of croton oil. Applied after pricking the skin with a multiple puncture appliance, it certainly "drew the poison out" in the form of numerous pustules.

Dr. Tillman's grand-son and grand-daughter still live in Montevideo.

The next name encountered is **Dr. Russell Stewart Bacon.** Here is a man who ranks high in importance in our list of pioneer physicians. An excellent doctor, he was public-spirited, and he took an active part in many civic enterprises. His fifty-one years of continuous, active practice in Montevideo make him second only to Dr. M. A. Burns in length of service to his community.

He was born May 28, 1859, the son of Mark and Emeline Bacon, at Elroy, Wisconsin. His mother died when he was four, and his father died when he was eight years of age. From this time on, he made his own way with the help of family friends. He worked his way through elementary school and graduated from high school in Mauston, Wisconsin, in 1880. He taught school for a time at Cottage Grove, a small town near South St. Paul, and studied medicine with a doctor there. He then attended Rush Medical College in Chicago, paying his way by working on the street-car lines, graduating in 1885. He came to Minnesota at once and established his first practice in Dawson in 1886, but moved to Appleton the next year. There he remained for two years. On April 11, 1899, he came to Montevideo and set up his practice, in which he remained active until 1940.

There were three excellent and active doctors in Montevideo when he arrived, Drs. Murphy, Collins and Rogers, but he soon began to get his share of business, and newspaper items in the succeeding years frequently concerned him. I quote: "Dr. Bacon has had several calls to Milan of late. On every visit, he finds many patients awaiting him, and on one trip, there were seven patients at one house, who were brought in from the neighborhood." On March 6, 1891: "Dr. Bacon drove to Clarkfield during a blizzard Tuesday night. He spent the greater part of the night wandering around on the prairie, but finally reached his destination in safety." On October 8, 1897, an account of probably the first x-ray in the county: "On Tuesday, in Dr. Bacon's office, Roentgen rays were turned on Paul Falkenhagen's leg, broken some time ago, and a skiagraph taken. H. L. Olson developed the skiagraph."

I quote the next item, as much for the rhetoric employed as for its historical significance, from the *Montevideo Commercial* of December 8, 1893: "Married by Rev. Morse, Thanksgiving evening, at the residence of the bride's parents at Cottage Grove, Minnesota, Dr. R. S. Bacon and Alice Gallinger. Our popular townsman, Dr. Bacon, not satisfied, as most mortals would have been, with ordinary Thanksgiving cheer, reaches out toward the hymeneal altar and snatches therefrom one of the fairest flowers of Cottage Grove, and claims her as his own. Surrounded by many witnesses, he plights his faith to one, who for many years, has been the idol of his heart, and unites his future to the brave and noble army of benedicts. That our genial doctor has done well, all will hope; that he has done wisely, none will question; that his future will be bright and happy all will pray. And now that he has returned to Montevideo, bringing his bride with him, a host of friends will meet them with hearty congratulations and kindly wishes that this crowning act may ennoble, enrich and beautify their united lives."

In addition to Dr. Bacon's extensive practice, he found time for many other activities. He served on the draft board during World War I and was a member on the school board from 1909 to 1919 and secretary of the U. S. Pension Board 1890-1929. He was also a member of the board of directors of the Riverside Sanatorium 1917-1937, and director of several banks at various times. He was a member of the Masonic Lodge since 1889, serving as Worshipful Master one year; a member of Damascus Commandery of Knights Templar of St. Paul since 1890; and a life member of the Camp Release District, Minnesota State and American Medical Associations.

He was confirmed as an Episcopalian in 1882, at Basswood Grove, Minnesota, but during his residence in Montevideo, attended the Congregational Church.

He kept abreast of medical times by frequent attendance at meetings and clinics. Dr. Bacon took extended courses of post-graduate work at New York University in 1892, again in 1896, and spent most of the winter of 1900 at New Orleans Poly-Clinic. Also, for a time in 1899, he went to Minneapolis every Saturday and worked in surgery as an assistant to Dr. Fred Dunsmoor in Asbury Hospital.

He retired from active practice in September 1940 at the age of 81, and his death occurred at the Montevideo Hospital only a few months later. His wife, two sons and two grandsons survived him, but Mrs. Bacon and one grandson, Richard, have died since then. His older son, Guy S., lives in Cottonwood, Minnesota, and the younger, Arthur, with his wife, resides in Dr. Bacon's old home in Montevideo.

Dr. Bernhard Lambrechts was educated both as a Doctor of Medicine and as a veterinarian. He was born in Christiania, Norway, April 15, 1828. Under some form of government subsidy, he received his education, with expenses paid, at Wittenberg, Germany, and Copenhagen, Denmark, graduating from both medical and veterinary school. He returned to practice medicine in Christiania and conducted a free clinic during part of his time (for an hour in the forenoon and an hour in the afternoon). According to his son, from whom I obtained this information, this was objected to by the aristocracy, and, as a result, Dr. Lambrechts emigrated to America in 1867, locating in southeastern Minnesota. His practice covered a huge territory extending from La Crosse, Wisconsin, to Northfield, Minnesota. In 1870, he spent some time in St. Louis, doing post-graduate work. In 1872, he moved to a farm a mile east of Willmar, in Kandiyohi county, again practicing medicine over a large area, extending from New London and Norway Lake to Benson and Montevideo. During this period, he served as coroner of Kandiyohi County for several terms. In 1891, he sold his farm and moved to Montevideo, but after locating here did no medical work and practiced veterinary medicine and surgery only. He was a member of the State Veterinary Board for several years. He was able to speak four languages fluently, Norwegian, German, French and English.

In 1904, he moved to East Granite Falls, where he died a year later.

His wife's name was Marie Muller, and there were three children: a son Barney, a daughter, Froides and a son, Thorfin, who studied Veterinary Medicine and practiced it all his life in Montevideo until his death in 1950. Thorfin's widow lives in the old Lambrechts home on North Second Street. The mother passed away in 1888, and Froides died in Illinois two years ago. Barney is living in Twin Brooks, South Dakota.

(To be continued)

In Memoriam

FRANK TANCREDE BENOIT

Dr. Frank T. Benoit, a Life Member of the Minnesota State Medical Association, died on February 7, 1955. He was seventy-three years old.

Dr. Benoit was born in Montreal, Canada. He became a naturalized citizen while still a minor through the naturalization of his father.

Dr. Benoit received his medical education at the University of Minnesota. He graduated in 1905. From 1905 until 1908, he practiced in North Dakota. He moved to Idaho and spent two years there, returning to Minnesota in 1910. He practiced for several years at Madison, Minnesota, and Slayton, Minnesota. In 1919, he settled in Winona and remained there until his death.

He was a member of the Winona County Medical Society.

WILLIAM C. CARROLL

Dr. William C. Carroll, prominent St. Paul surgeon, died March 18, 1955. He was sixty-five years old.

For many years, Dr. Carroll was associated with the St. Paul Clinic which he and Dr. Charles W. Fogarty organized in 1920. He was an attending surgeon at St. Joseph's Hospital and Miller Hospital, and a consulting surgeon at Gillette State Hospital.

Dr. Carroll was born at Lake City, Minnesota. He received his education at St. Thomas Academy and St. Thomas College in St. Paul. He received his medical training at the University of Minnesota, graduating in 1912. He interned an Ancker Hospital in St. Paul and spent two and a half years at the Mayo Clinic, for postgraduate work.

Dr. Carroll was a member of the Ramsey County Medical Society, the Minnesota State Medical Association, the American Medical Association, the Mayo Alumni Association, and the American College of Surgeons.

In 1936, Dr. Carroll was named president of the St. Thomas Alumni Association. He was elected president of the St. Paul Surgical Society in 1938.

He is survived by his wife, Lauretta; a daughter, Mrs. Margaret Clark; a son, William C., Jr., and a sister, Nellie M. Carroll, all of St. Paul.

JAMES HARRIS HAINES

Dr. James H. Haines, who had practiced medicine in Stillwater for more than fifty years, died February 16. He was eighty-three years old.

Born in Grinnell, Iowa, Dr. Haines spent his early life there and received his B.S. degree at Grinnell College. He graduated from Rush Medical college in 1895 and moved to Stillwater. He started his career as a house physician at the Stillwater Hospital, later opening an office for general practice. He was health officer of Stillwater and Washington County coroner for many years.

In 1947, Dr. Haines became a "Fifty" Club member of the Minnesota State Medical Association. He was a member of the Washington County Medical Society.

He is survived by his wife, Josephine; a daughter, Mrs. Lloyd Nelson, and two grandchildren, all of Stillwater. He is also survived by a brother, Charles, of Denver and two sisters, Mrs. F. I. Herriott of Washington and Mrs. W. G. Ray of Des Moines.

JOHN FRANCIS MADDEN

Dr. John F. Madden, fifty-three, assistant professor of dermatology and syphilology at the University of Minnesota, and director of the dermatology department at Ancker Hospital, St. Paul, died in his sleep March 9, 1955.

Dr. Madden was born in Hillsboro, Wisconsin. He received his early education at Plentywood, Montana, and came to the University of Minnesota to obtain his medical degree. After his internship at the General Hospital in Kansas City, Missouri, he spent two years at Gibbon, Minnesota. During the years 1928 to 1930, he studied at the University of Michigan. He received a degree of Master of Science in dermatology.

Dr. Madden had offices in the Lowry Medical Arts Building in St. Paul. He was a consultant at Children's and Gillette State Hospitals. He was a member of the Ramsey County Medical Society, the Minnesota State Medical Association, the American Medical Association, the American Dermatological Association, and the American Academy of Dermatology and Syphilology.

Surviving are his wife, Elizabeth; a daughter, Mary Ellen, and a son, Peter, a student at Amherst College.

ALFRED CARLTON PETERSON

Dr. Alfred C. Peterson, a veteran Minnesota physician, died March 1, 1925, at the Meeker County Memorial Hospital, Litchfield.

Dr. Peterson was born at Cokato, Minnesota, in 1875. He graduated from the University of Minnesota College of Pharmacy in 1898. He studied medicine later and received his degree in 1903. He practiced for one year in Shevlin, Minnesota, and another in Murdock. In 1905, he went to Dassel. He remained there and was active in medical practice almost until the time of his death.

Dr. Peterson was surgeon for the Great Northern Railway, Health Officer in Dassel, member of the Litchfield Hospital staff, Director of the Tri-County Medical Society (now Kandiyohi-Swift-Meeker County Medical Society), and a member of the Minnesota State Medical Association.

He was also active in community affairs, and on

(Continued on Page 284)

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

Minnesota Academy of Occupational Medicine and Surgery, annual meeting, Minneapolis Athletic Club, May 25, 1955.

National

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

American Proctologic Society, annual meeting, Hotel Statler, New York, New York, June 1-4, 1955.

International

International College of Surgeons, twentieth anniversary congress, Palais Du Conseil General, Geneva, Switzerland, May 23-26, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examinations (Part II), oral and clinical, for all candidates for the American Board of Obstetrics and Gynecology will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from May 11 through May 20. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

AMES AWARD CONTEST FOR GASTROENTEROLOGY

The American College of Gastroenterology, in cooperation with the Ames Company, Elkhart, Indiana, announces the establishment of the Ames Award Contest for papers in gastroenterology. The contest is divided into two major classes with a first prize of $250 and a second prize of $50 in each class. One class is for fellows or residents in gastroenterology, the other for first or second-year interns. An additional prize of $100 will be given for the best paper published in the *American Journal of Gastroenterology* for which no prize has been previously awarded.

All papers submitted must represent original work in the field and must not have been previously published or presented at national meetings. The approach can be either clinical or basic science. Awards will be made at the annual meeting of the American College of Gastroenterology in Chicago in October, where the first-prize winners will present their papers in person.

All entries must be received no later than September 1 by the Research Committee, American College of Gastroenterology, 33 West 60th Street, New York 23, New York.

SCHERING AWARD COMPETITION FOR MEDICAL STUDENTS

The tenth annual Schering Award competition for medical students has opened. Its aim is to encourage medical writing and exploration of current research literature.

Titles of three subjects on which American and Canadian students are invited to submit papers this year are: Current Concepts in the Management of Osteoporosis; Prevention and Treatment of Blood Transfusion Reactions; and Recent Trends in the Clinical Use of Adrenocortical Steroids.

Both a $500 first prize and a second one of $250 will be awarded for each of the three subjects. Deadline for entry forms specifying the choice of title is July 1. Manuscripts should be mailed not later than October 1. Students may compete individually or co-operatively in teams. Information and instructions are available from the Schering Award Committee, 60 Orange Street, Bloomfield, N. J.

MINNESOTA LEAGUE FOR NURSING

"Better Patient Care for a Growing State" will be the theme for the state convention of the Minnesota League for Nursing, May 11-13, 1955. Registration for the convention, including all the meetings, will be held in the Nicollet Hotel, Minneapolis.

At the banquet to be held in the Terrace Room Thursday evening, Dr. Harold Graning, Regional Medical Director United States Public Health Service, Chicago, Illinois, will speak on "Citizen Participation for Better Health." Guests will be welcome.

Exhibits of the Upper Midwest Hospital Conference will be displayed in the Minneapolis Auditorium.

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

The Midwestern Section of the American Congress of Physical Medicine and Rehabilitation will hold a meeting in Minneapolis on May 6 in the Mayo Memorial Auditorium in the Mayo Memorial Building on the University campus. Registration will be at 8:30 a.m. without charge. In addition to scientific papers, there will be a tour of the facilities of the Department of Physical Medicine and Rehabilitation in the Mayo Memorial Building. All interested physicians are invited to attend. For further information, write to the chairman, Dr. G. K. Stillwell, 200 First Street Southwest, Rochester, Minnesota.

CONTINUATION COURSES

Introduction to Electrocardiography will be the title of a continuation course for physicians which will be presented by the University of Minnesota at the Center for Continuation Study, May 9 to 14, 1955. As its name implies, this year's course is intended primarily for physicians with little or no previous experience in electrocardiographic interpretation. Basic principles, variations in normal patterns, and frequently observed abnormalities will be stressed. Registrants will have an opportunity to interpret more than 200 electrocardiograms under supervision of qualified instructors.

The University of Minnesota announces a continuation course in *Surgery for General Surgeons* which will be held at the Center for Continuation Study from May 26 to 28. Recent important advances in surgery will be discussed. Guest speaker will be Professor Ian Aird of the University of London Postgraduate Medical School. Professor Aird will also deliver the annual E. Starr Judd Lecture on May 26. The course will be presented under the direction of Dr. O. H. Wangensteen, professor and chairman, Department of Surgery.

TECHNICAL EDITORS AND WRITERS NEEDED

The United States Civil Service Commission announces that an examination is open for technical editors and writers in the fields of physical sciences and engineering, and medical sciences. The positions pay $3,410 to $10,800 a year and are located in the Potomac River Naval Command and other Federal agencies in Washington, D. C., and vicinity.

No written test is required, but applicants must have had appropriate experience or a combination of education and experience. Further information and application forms may be obtained at many post offices throughout the country, or from the U. S. Civil Service Commission, Washington 25, D. C.

TRUDEAU SCHOOL OF TUBERCULOSIS

Despite the closing of the clinical facilities of the Trudeau Sanatorium, the forty-first session of the Trudeau School of Tuberculosis will begin June 1 and continue to June 29. The staff, facilities and skills of the Trudeau organization laboratories, of the various sanatoria in the Saranac Lake area, and of the practicing tuberculosis specialists of Saranac Lake will be called upon as in the past to present the program.

The course will cover all aspects of pulmonary tuberculosis and also certain phases of other chronic chest diseases, including those of occupational origin.

Registrations will be limited and it is suggested that those planning to attend make early application for enrollment. The tuition is $100, payable to the Trudeau School on or before the opening date, June 1. A few scholarships are available for those individuals who can qualify. Applications and more detailed information may be obtained from Secretary, Trudeau School of Tuberculosis, Box 200, Trudeau, New York.

In Memoriam

(Continued from Page 282)

March 28, 1949, the residents of Dassel honored Dr. Peterson at an Appreciation Day.

Dr. Peterson is survived by his wife, Karen Marie; two daughters, Mrs. Harold R. Havig, Owatonna, and Mrs. Seymour N. Gayner, Chaska; five grandchildren; two brothers, John of Dassel and Edward of Knapp; one sister, Mrs. Oscar Sandeen of Mound.

ERNEST G. STERNER

Dr. Ernest G. Sterner, St. Paul surgeon, died January 20, 1955, after an illness of several months. He had retired from practice less than a year before. He was seventy-two.

Dr. Sterner was born at Ohr, Sweden, in 1882. He graduated from Hamline University's medical school in 1906. He had been a resident of St. Paul for sixty-four years.

President of the Ramsey County Medical Society in 1923, Dr. Sterner was also a life member of the Minnesota State Medical Association, a member of the St. Paul Surgical Society, the American Medical Association and the Phi Rho Sigma fraternity, and past chief of staff at Bethesda Hospital.

Survivors are his wife, Elizabeth; two daughters, Mrs. Edwin Frenzel, Oaklawn, Ill., and Mrs. Charles Morscheck, Lansing, Mich,; a son, Dr. John H. Sterner, St. Paul; and nine grandchildren.

Communication

Dear Editor:

I should like to enlist your aid in bringing to the attention of Minnesota doctors information about the availability of the service of the Calvert School for the education of home-bound children of elementary school age.

Calvert is an unendowed non-profit institution in Baltimore, regularly accredited by Maryland's department of education. Since 1905 it has provided an elementary school education by mail, from kindergarten through the Ninth Grade, for children who cannot attend regular schools. It pays particular attention to the problems of the home-bound child. It is not in competition with the visiting teacher programs of the public school systems; rather, it reaches areas beyond the range of those programs.

As a non-profit organization, Calvert has a primary interest in extending its service where needed rather than in increasing its enrollment.

Very truly yours,
Donald C. Thompson

30 Rockefeller Plaza,
New York 20, N. Y.
February 23, 1955

Woman's Auxiliary

HENNEPIN AUXILIARY CONVENTION HOSTESSES

Minnesota doctors' wives are cordially invited to attend the 33rd annual meeting of the Woman's Auxiliary to the Minnesota State Medical Association in Minneapolis, May 23, 24 and 25. Headquarters will be in the Hotel Radisson.

Numerous interesting events have been planned by the Woman's Auxiliary to the Hennepin County Medical Society, with Mrs. Clyde M. Cabot and Mrs. E. T. Evans as co-chairmen for the convention.

Advance registration will begin at 1:00 p.m., Sunday, May 22, at the Hotel Radisson, and will continue at 8:00 a.m., Monday and Tuesday. The Executive Board meeting and luncheon will be at the Minneapolis Club, beginning at 9:45 a.m., Monday, May 23. In the afternoon, a tea and style show will take place at Rothschild-Young Quinlan's, beginning at 2:30 p.m. A tour of a local candy manufacturer's studio has also been planned for Monday morning.

Monday evening, doctors and their wives will attend the annual Open House entertainment, scheduled for 9:00 p.m. at the Hotel Radisson. Hosts will be the Hennepin County Medical Society—excellent entertainment has been secured in honor of the 100th anniversary of the host society.

The official annual business meeting of the State Auxiliary will begin at 9:30 a.m., Tuesday, May 24, in the Junior Ballroom, Hotel Radisson. Annual reports will be heard and officers will be elected. Following this will be the annual luncheon, where a nationally recognized performer will entertain. Installation of officers, presentation of president's pin and gavel, and greetings from the new president will take place here, followed by the new president's first post-convention board meeting.

Tuesday evening, May 24, the annual banquet of the Minnesota State Medical Association will be held in the Ballroom of the Hotel Radisson.

Final activity of the meeting will be the Wednesday morning breakfast at the home of Mrs. Harvey Nelson.

The Hennepin County Medical Auxiliary has arranged a fine array of social and business events for this meeting, and auxiliary members, and non-members also, are urged to plan now to attend.

RAMSEY COUNTY REPORTS ACTIVITIES

Mrs. L. T. Simons

The Woman's Auxiliary to the Ramsey County Medical Society assisted in the Career Festival, March 8, 9 and 10 at St. Thomas College in St. Paul. The event brought thousands of high school and college students from the entire state to gain information about various careers they may wish to follow.

The Minnesota State Medical Association sponsored a booth at the festival. Members of the auxiliary who were on hand to be of assistance were: Mesdames R. R. Grau, Wallace A. Gleason, M. D. Hilker, L. F. Freidman, H. H. Fesler, C. W. Froats, A. J. Felion, Jr., Alexander McEwan, L. T. Simons. Arrangements were made by Mrs. Douglas Kusske and Mrs. W. P. Gardner, Ramsey Auxiliary president. These members, assisted by seniors from the St. Thomas pre-medical school, gave out information and literature to students interested in a medical career.

Mrs. W. P. Gardner, president, called a board meeting on March 21 in the Medical Arts Library. During and following the meeting, cancer dressings were made.

Mrs. D. R. Gillespie, chairman for *Today's Health* subscriptions, and Mrs. Frank Milnar had charge of a booth on March 28 during the "What's Cooking?" program. Pamphlets and posters were on display, and magazines were sold by the copy and by subscription.

Our case work representative, Mrs. Alexander McEwan, attends a monthly meeting at which welfare groups are represented. Bills in the legislature and civic problems are explained and discussed.

Mrs. Wallace Gleason, press chairman, and Mrs. John Madden attended a meeting of the Historical Society on February 17. Dome Club members were guests of the society at a tea and lecture by Nobel prize winner, Dr. Philip Hench, of Rochester.

The auxiliary sent contributions to the Crusade for Freedom and also to the Civic Opera Association, the latter to be used to purchase tickets for the blind to attend the opera's presentation of "Oklahoma."

COUNTY AUXILIARIES REPORT HEALTH PROJECTS

Mrs. L. Raymond Scherer

Following a request to the presidents of the county auxiliaries, several have reported on some of their community activities in the health field.

The most popular is in the field of nurse recruitment—entertaining and informing the high school students of the possibilities of a career in that profession.

Wright, Redwood-Renville and McLeod Auxiliaries helped in the poliomyelitis, heart and cancer fund-raising campaigns. Wright and Hennepin Auxiliaries also made cancer dressings, as did Ramsey Auxiliary. Redwood-Renville Auxiliary also helped with the Red Cross Mobile Unit, Wabasha Auxiliary worked with a local PTA and arranged to bring Dr. Roy C. Burt of the Anoka State Hospital to speak for their March meeting.

Hennepin County Auxiliary gives financial support to many health and health education organizations.

St. Louis County Auxiliary arranged a most interesting meeting honoring Dr. Kate P. Newcomb, a physician from Woodruff, Wisconsin. She had been the "star" on "This Is Your Life." A result of that program was a fund of $110,000 to complete and furnish the Lakeland Memorial Hospital in Woodruff. In Duluth, 200 were present to hear her and to see the film of the national television network program on her life.

Of General Interest

Dr. E. M. Hammes, St. Paul, attended the regional research meeting of the American Psychiatric Association in Galveston, Texas, on February 18 and 19. The entire meeting, he reported, was devoted to the physiologic basis, values, limitations and hazards of pharmacologic products recently introduced in the treatment of psychiatric disorders. Dr. Hammes drove to the meeting from Florida, where he had spent his winter vacation.

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Dr. Cyril M. Smith, Duluth, St. Louis County coroner, was principal speaker at a meeting of the Duluth Association of Accident and Health Underwriters in mid-February. Dr. Smith discussed cardiovascular diseases and occupational and industrial hazards.

* * *

"Self-Medication" was the title of a radio talk given by **Dr. Donald M. Odland,** Luverne, over station KWOA, Worthington, on February 20. He discussed the dangers invited by persons who diagnose and prescribe for themselves. His talk was presented on "Your Health Hour," a program sponsored by the Southwestern Minnesota Medical Society and Auxiliary and the Minnesota Department of Health.

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Dr. Irving C. Bernstein, Minneapolis, has been elected to membership in the American Psychosomatic Society.

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Dr. Edward H. Juers, Red Wing, attended a one-week meeting of the American Academy of Orthopedic Surgery in Los Angeles early in February.

* * *

Dr. Charles H. McKenzie, Minneapolis, has announced that **Dr. Francis M. Swain** is now associated with him in the practice of gynecology and obstetrics, with offices at 1035 Medical Arts Building, Minneapolis.

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Among physicians who attended the tenth National Rural Health Conference in Milwaukee in late February was **Dr. Paul C. Leck** of Austin.

* * *

Dr. Frederic F. Wipperman, Minneapolis, was recently elected president of the Edina Optimist Club, a civic-minded organization whose motto is "Friend of the Boy." Dr. Wipperman has practiced ophthalmology in Minneapolis since 1946.

* * *

Dr. O. M. Heiberg, Worthington, presented a special lecture for senior medical students at the University of Minnesota late in February. As part of a course in "Orientation to Practice," he discussed the subject of practicing in a clinic.

Dr. Jack V. Wallinga, formerly of Minneapolis, recently attended the American Orthopsychiatric Association meeting in Chicago. Dr. Wallinga is now stationed at the U. S. Naval Hospital, Oakland, Calif., where he is clinical director of the neuropsychiatric service.

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Dr. Berry Campbell, associate professor of anatomy at the University of Minnesota, presented a series of lectures on the nervous system at medical centers in Norway, Sweden and The Netherlands in March. He returned to Minneapolis on March 25. Sponsor of his lecture tour was the University of Leiden.

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Guest speaker at a meeting of the Women's Auxiliary of Immanuel Hospital in Mankato in mid-February was **Dr. H. J. Nilson** of Mankato. He spoke on the hospital and medical situation in Sweden, where he visited last year.

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Dr. C. J. Watson, head of the department of medicine at the University of Minnesota Medical School, is visiting the Middle East during April to lecture and conduct conferences in medical schools in Lebanon, Iraq, Syria, Jordan, Egypt and Israel. His trip is sponsored by the International Education Division of the U. S. Department of State. He will return to Minneapolis on May 3.

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Dr. James A. Leigh has been appointed city health officer in East Grand Forks. He succeeds the late Dr. G. P. Kirk, who died January 20 after holding the office for more than thirty years. Dr. Leigh has practiced in East Grand Forks since 1953.

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The American College of Surgeons has awarded a $20,000 research grant to **Dr. Fletcher A. Miller,** a surgery research fellow at the University of Minnesota Medical School. The grant will cover a three-year period beginning July 1, 1955. Through his research thus far at Minnesota Dr. Miller has developed the use of a mass spectrometer to determine the level of carbon dioxide developed by a patient during surgery.

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Dr. Andrew Sinamark, Hibbing, was the principal speaker at a meeting of the Kiwanis Club at Ely late in February. He showed pictures of a trip he made last summer to the Pacific islands.

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A new surgical textbook, "Surgery of the Small and Large Intestine," by **Dr. C. W. Mayo,** Rochester, has just been published by the Year Book Publishers, Inc., of Chicago. The book, consisting of 340 pages and 94 plates, was illustrated by Russell Drake of the art studio of the Mayo Clinic.

Dr. Milton Howell, Glencoe, was the principal speaker at a meeting of the Glencoe Business and Professional Women's Club in late February. Dr. Howell discussed "Cancer in Women and Manner of Self-detection." A motion picture on the same subject preceded his talk.

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During the middle of March, **Dr. A. C. Hilding**, Duluth, was in Hollywood-by-the-Sea, Florida, attending meetings of the American Laryngological Association, the American Triological Society and the American Otological Society. He was elected to membership in the American Otological Society on March 17. On April 6 and 7, Dr. Hilding presented a series of four lectures on eye and ear research at the annual Spring Congress in Ophthalmology and Otolaryngology of the Gill Memorial Eye, Ear and Throat Hospital, Roanoke, Virginia.

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Dr. Tague C. Chisholm, Minneapolis, discussed accidents in childhood when he spoke at a meeting of the Duluth Principals and Supervisors Association and the Duluth Parent-Teachers Association Council in February.

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Principal speaker at a clinical pathological conference at the Falls Memorial Hospital, International Falls, in late February was **Dr. H. E. Law**, Virginia. He discussed cancer of the stomach. The conference is held each month, featuring a specialist in some field of medicine as guest speaker.

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Dr. Leo Furr, Bird Island, reported for a two-year tour of duty with the Air Force on March 11. He was associated in practice in Bird Island with **Dr. H. P. Hinderaker.**

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Dr. E. S. Boone, Luverne, was the principal speaker at a meeting of the Rock County 4-H leaders at Luverne in late February.

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Dr. R. N. Barr, deputy state health commissioner, discussed "How Safe Is Your Child" at a meeting of the women's section of the Industrial Safety School in Duluth on March 9.

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Among physicians attending the eighteenth annual meeting of the New Orleans Graduate Medical Assembly in New Orleans, March 7 to 10, was **Dr. R. R. Wright** of Austin.

* * *

Dr. and Mrs. J. A. Myers, Minneapolis, spent a week in mid-March on a business and pleasure trip to Puerto Rico. During the trip Dr. Myers, who is professor of preventive medicine at the University of Minnesota, gave a series of lectures at San Juan, Puerto Rico.

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While returning from a medical meeting in Chicago in mid-February, **Dr. John B. Frost**, Minneapolis, suffered minor injuries when a train on which he was riding was derailed near Waukegan, Illinois.

Dr. John B. Moyer, Duluth, has been appointed president of the Duluth Heart Council, a group which aims at disseminating programs of public and professional education.

* * *

Dr. Robert Gray, formerly of Marshall, was married to Betty Batcup of Devon, England, on January 29. The wedding took place in Nairobi, Kenya, Africa. Dr. Gray left for Africa on a Ford Foundation fellowship in August, 1954. He has completed research in health, hospital and medical practices in Kenya and will continue his research for eighteen months in Tanganyika and Uganda. Dr. Gray is the son of **Dr. and Mrs. F. D. Gray** of Marshall.

* * *

A scientific motion picture produced at the University of Minnesota has received a second-place silver medallion from the Screen Producers Guild in Los Angeles. The film, "One Victory," is a presentation of a low-temperature heart operation performed by **Dr. Richard Varco, Dr. F. John Lewis** and **Dr. Mansur Taufic.**

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Dr. W. F. Mercil, Crookston, was the principal speaker at a meeting of the Mathein Club in Crookston on March 7. He spoke on the topic, "Preventive Medicine in the Human Female."

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Dr. John M. Waugh, Rochester, was one of the speakers at a sectional meeting of the American College of Surgeons in Nashville, Tennessee, on April 4. He discussed "Various Types of Gastrectomy."

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Maj. Gen. Silas B. Hays, a native of St. Paul, has been nominated by President Eisenhower to be Surgeon General of the Army, effective June 1. Dr. Hays, who has been deputy surgeon general for the past four years, would succeed Maj. Gen. George E. Armstrong, who is scheduled to retire.

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Dr. David Allison, Litchfield, left on March 14 to begin a two-year tour of duty in the Air Force. While he is on military duty, his practice will be handled by his associate, **Dr. Gregory Olson** of Litchfield.

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At the age of ninety-two, **Dr. Fletcher W. Penhall**, Willmar, retired from active practice on March 1. Dr. Penhall, a graduate of Bellevue Medical Center in New York, began practicing in Morton in 1891. After fifty-three years of practice there, he moved to Willmar and became a member of the staff of the Willmar State Hospital in 1944. Now in retirement, Dr. Penhall is again living in Morton.

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A testimonial dinner honoring **Dr. E. E. Novak** of New Prague was held in Jordan by King Hiram Lodge, A.F. and A.M., on February 22. At the dinner, residents of the area paid tribute to Dr. Novak for his almost sixty years of service as a physician in New Prague. Representatives of numerous fraternal organizations were on hand to honor him and to present him with a watch to mark the occasion. Dr. Novak was graduated from the University of Iowa in 1895. He served as a member of the University of Minnesota Board of Regents for twenty-two years.

* * *

In Hallock three physicians have combined to form the Hallock Clinic. They are **Dr. J. E. Nord, Dr. Anthony S. Berlin** and **Dr. P. J. Parker**. The clinic will be housed in the building formerly occupied by Dr. Nord. The building has been completely remodeled and redecorated to provide proper quarters for the new medical center.

* * *

The Minnesota State Medical Association's new television series, "Doctor's Digest" appears weekly over KEYD-TV. The show, jointly sponsored by the Association and the University of Minnesota, can be viewed from 6:30 to 7:00 p.m. on Thursday evenings. **Dr. James Rogers Fox**, Minneapolis, serves as moderator and the show features regular medical tips of the week and medical news of the week, plus panels, interviews and film clips. Billed as a "medical variety show," it is hoped that "Doctor's Digest" will bring important medical and health news to the people of the state.

* * *

The alumni of the Cyrus Fishermen's College will hold its 17th annual banquet at Cyrus, Minnesota, April 29, at 7 P.M. **Dr. H. Linde**, who has practiced medicine in Cyrus nearly fifty years, is president of the college. All Minnesota doctors are cordially invited to this banquet of walleye pike.

NEW LOCATIONS

Dr. Ralph Rayner, formerly of Gibbon, has become associated in practice with **Dr. David F. Struxness at Glencoe**. A graduate of the University of Minnesota Medical School, Dr. Rayner interned at Minneapolis General Hospital. He practiced at Gibbon for the past three years.

Dr. Arthur Neumaier has returned to Glencoe to resume the practice of medicine there following a period of study at the Veterans Administration Hospital in Minneapolis.

Dr. Charles T. Louisell, who recently was released from active duty with the Navy, has opened offices for the practice of medicine in Fairmont. A graduate of the University of Minnesota in 1944, he specialized in ophthalmology and otolaryngology before beginning his military service.

Dr. John B. Maunder, who recently completed two years of duty in the Air Force, has become associated with the Oxboro Clinic in Richfield. A graduate of the University of Minnesota in 1945, he specialized in obstetrics and gynecology before entering the Air Force.

Dr. D. W. Sontag, formerly of Long Beach, Calif., has become associated in practice with **Dr. E. C. Bayley** and **Dr. B. A. Flesche** in Lake City. He is a graduate of the University of Minnesota.

HOSPITAL NEWS

The results of elections conducted recently by the medical staffs of numerous Minnesota hospitals have been announced. Among the election results reported are the following:

Fairview Hospital, Minneapolis: Dr. Glenn Petersen, chief of staff; Dr. L. J. Roberts, chief-elect; Dr. Lester W. Carlander, Jr., treasurer; Dr. Stanley Stone, secretary.

Montevideo Hospital, Montevideo: Dr. L. A. Krystosek, chief of staff; Dr. J. H. Allen, vice president; Dr. E. G. Hustad, secretary-treasurer.

Municipal Hospital, Worthington: Dr. C. R. Stanley, chairman; Dr. D. J. Halpern, vice chairman; Dr. Raymond Minge, secretary.

St. Joseph's Hospital, Brainerd: Dr. R. L. Cushing, chief of staff; Dr. E. R. Kanne, vice chief of staff; Dr. Maurice Meller, secretary-treasurer.

Doctors Memorial Hospital, Minneapolis: Dr. Allan J. Blake, chief of staff; Dr. Richard H. Beiswanger, assistant chief of staff; Dr. Fred A. Rice, secretary.

Miller Memorial Hospital, Duluth: Dr. F. T. Becker, chief of staff; Dr. John F. Schmid, vice chief of staff; Dr. John J. Dwyer, secretary-treasurer.

St. Francis Hospital, Shakopee: Dr. I. B. Heinz, chief of staff; Dr. B. F. Pearson, assistant chief of staff; Dr. Harry Simmonds, secretary-treasurer.

St. Barnabas Hospital, Minneapolis: Dr. Arthur H. Mork, chief of staff; Dr. Lloyd F. Sherman, vice chief of staff; Dr. John T. Moehn, secretary-treasurer.

St. Gabriel's Hospital, Little Falls: Dr. A. H. Benson, president; Dr. David Hoehn, vice president; Dr. G. W. Heine, secretary-treasurer.

St. Luke's Hospital, Duluth: Dr. M. F. Fellows, chief of staff; Dr. Arden L. Abraham, chief-elect; Dr. H. J. Jeronimus, secretary.

Community Hospital, Fairmont: Dr. O. E. Wandke, president; Dr. A. L. Ourada, vice president; Dr. H. A. Williamson, secretary.

Asbury Hospital, Minneapolis: Dr. Wayne S. Hagen, chief of staff; Dr. Einar Monson, vice president; Dr. M. Elizabeth Craig, secretary-treasurer.

Olmsted Community Hospital, Rochester: Dr. Theodore O. Wellner, president; Dr. James R. Doyle, vice president; Dr. R. L. Page, secretary.

St. Mary's Hospital, Minneapolis: Dr. James E. Trow, chief of staff; Dr. C. N. Borman, vice chief of staff; Dr. John Kelly, secretary-treasurer.

Community Memorial Hospital, Faribault: Dr. Ernest J. Nelson, chief of staff; Dr. Paul Stahler, vice chief of staff; Dr. Elizabeth Reischl, secretary-treasurer.

St. Joseph's Hospital, Saint Paul: Dr. J. P. Medelman, president; Dr. Bernard O'Reilley, vice president; Dr. Francis W. Lynch, secretary; Dr. C. L. Cain, treasurer.

City Hospital, Owatonna: Dr. O. W. Roberts, president; Dr. Frank C. Anderson, vice president; Dr. K. G. Henry, secretary-treasurer.

Glenwood Hills Hospital, Minneapolis: Dr. Irving Bernstein, chief of staff; Dr. John Schumacher, vice president; Dr. Jennings Peteler, secretary-treasurer.

St. Mary's Hospital, Duluth: Dr. Karl E. Johnson, chief of staff; Dr. R. E. Nutting, chief-elect; Dr. J. C. O'Neill, secretary.

MINNESOTA BLUE SHIELD - BLUE CROSS

Blue Cross

During the month of January, 1955, 11,119 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of January 31, 1955, to 1,002,760 participant subscribers.

Hospitalization expense incurred by Blue Cross subscribers during the month of January, 1955, amounted to $1,374,711.70.

Effective March 1, 1955, a Washington, D. C., office of the Blue Cross Commission will open the Artemas C. Leslie, Associate Director of the Blue Cross Commission in charge.

The reasoning for opening of the Washington office was due to the importance and frequency of contracts with our Federal Government which assures full time staff attention to Washington affairs.

Artemas C. Leslie is a Pittburgh attorney and for the past five years has been Insurance Commissioner of Pennsylvania. He was originally appointed in 1950 by Governor James H. Duff and reappointed by Governor John S. Fine. Under Leslie, many new fields of insurance activity were opened up, including new concepts of multiple-line underwriting, "package" policies, and other experimentations.

Leslie has been active in the affairs of the National Association of Insurance Commissioners. He has been a member of the Association's Executive Underwriting Powers as well as chairman of the Subcommittee on Blue Cross and Blue Shield of the Accident and Health Committee.

Leslie is senior partner in the law firm of McCloskey, Best & Leslie, of Pittsburgh. He is a member of the Allegheny Counuty, Pennsylvania, and American Bar Associations, the National Insurance Bar, the Federation of Insurance Counsel, American Law Institute, and American Judicature Society. He is admitted to practice in the State and Federal Courts, including the Supreme Court of the United States.

Blue Shield

Of increasing interest are the Blue Shield statistical data compiled annually which show the number of different types of services furnished Blue Shield subscribers by doctors of medicine. On a comparative basis, each type of service in 1954 increased numerically over the number of such services in 1953, and the average payment for each type of service also showed an increase. Such services as in-hospital medical care, emergency medical care, obstetrical, surgical and ancillary services showed a marked increase both in the number of cases in 1954 as compared with 1953, and in the amount paid by Blue Shield for such services.

During 1953, the average payment made to doctors for in-hospital medical care was $24.66 per claim. In the year 1954, this average increased to 29.01 per service and the number of such services increased 15% over 1953. More remarkable is the increase in emergency medical care payments and the increase in the number of such claims compared with the year 1953. Payments in 1953 for emergency medical care averaged $338.48 per claim. In the year 1954, this average payment increased to $43.02 per claim, or an increase of $4.54 more than the average amount paid per claim in 1953. In addition, the number of claims of this nature in 1954 almost doubled the number paid in 1953.

Obstetrical services furnished Blue Shield subscribers have always been one of the most frequent Blue Shield services. During 1953, the average cost per claim for this service was $52.52. During the year 1954, the average cost per service was $60.20. It is also noted that an increase of 698 in the total number of obstetrical services was experienced in 1954 as compared with 1953.

In reviewing the data on surgical services rendered to Blue Shield subscribers, an increase of 16 per cent was experienced in the total number of surgical services provided during 1954. Other types of services showing marked increases are those known as ancillary services. These include anesthesia, x-rays and endoscopic services. For each type of ancillary service during the year 1954 substantially more benefits were paid by Blue Shield for services rendered to subscribers.

It is apparent from these analyses that in both the number of services and the amounts paid for the different services, Blue Shield payments to doctors have grown appreciably in 1954 as compared with 1953. There is great likelihood that payments both in number and costs will be even greater in 1955.

MINNESOTA ACADEMY OF MEDICINE

(Continued from Page 275)

met. Some of these ideas and ideals have been mentioned by some of our distinguished former presidents and I simply wish to implement their fulfillment. Each of you will have time to evaluate them and I beg your indulgence to give these matters your early consideration in the coming year.

Dr. Regnier, of Minneapolis, read his address as retiring president on "Hormonal Treatment of Metastatic Carcinoma of the Breast." Lantern slides were shown.

The meeting adjourned.

Robert E. Priest, M.D., *Secretary*

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

EARLY CARE OF ACUTE SOFT TISSUE INJURIES. Committee on Trauma, American College of Surgeons. 192 pages. Price $1.00, paper cover. Chicago: American College of Surgeons, 1954.

AN OUTLINE OF THE TREATMENT OF FRACTURES. Fifth Edition. Committee on Trauma, American College of Surgeons. 93 pages. Illus. Price $1.00, paper cover. Chicago: American College of Surgeons, 1954.

ION EXCHANGE AND ADSORPTION AGENTS IN MEDICINE. Gustav J. Martin, Sc.D., Research Director, National Drug Company, Philadelphia. 333 pages. Illus. Price $7.50, cloth. Boston: Little, Brown & Co., 1955.

SURGERY OF THE SMALL AND LARGE INTESTINE. Charles W. Mayo, M.D., Section of Surgery, Mayo Clinic, Rochester, Minnesota; Professor of Surgery, Mayo Foundation, Graduate School, University of Minnesota. 340 pages. Illus. Price $9.00, cloth. Chicago: Year Book Publishers, Inc., 1955.

THE MEDICAL CARE OF THE AGED AND CHRONICALLY ILL. Freddy Homburger, M.D., Research Professor of Medicine, Tufts College Medical School. 253 pages. Illus. Price $5.75, cloth. Boston: Little, Brown & Co., 1955.

NATIONAL PLUMBING CODE. Sponsored by American Public Health Association and American Society of Mechanical Engineers. 174 pages. Price $3.50, paper. New York: American Society of Mechanical Engineers, 1955.

THE HEALTH VISITOR AND TUBERCULOSIS. By Sheena H. Buchanan, SRN, SCM, HV Cert, Assistant Superintendent Nursing Officer in Charge of Health Visitors, Northamptonshire County Council. Foreword by J. Greenwood Wilson, M.D., FRCP, DPH, Medical Officer of Health, Port of London, Formerly Medical Officer of Health, City & Port of Cardiff. Preface by G. B. Lord, M.D., Physician Superintendent, Rushden House Sanatorium, Northamptonshire. 150 pages, including facsimiles of report cards. Clothbound. Price 8s. 6d. London: The National Association for the Prevention of Tuberculosis, 1955.

Here, in concise and readable form, is a practical guide for health visitors in tuberculosis written from the author's own experience. Although she treats in detail the minute-to-minute practicalities of routine work and considers such wide questions as general and specialized health visiting, the health visitor and the chest clinic, and the changes brought about by the National Health Service Act, Miss Buchanan never forgets the human aspect. The family is the central social unit and courage and patience are the prime attributes of the good health visitor trying to help them. No one in the public health work should miss this latest contribution to the welfare of the tuberculous.

ANATOMY; REGIONAL AND APPLIED. By Raymond Jack Last, M.B., B.S. (Adel.), F.R.C.S., Professor of Applied Anatomy, Royal College of Surgeons of England. 665 pages. Illus. Price $10.00. Boston: Little, Brown and Company, 1954.

This book on human anatomy is written to help the reader understand anatomy. It is in no sense a book on surgical anatomy, in which the reader is given a short course in surgical technique rather than anatomy. The book is divided on a regional basis. The embryological and functional aspects are given. An added feature is the bibliographical notes at the end of the book. An adequate index is given and the book is well printed. It should enjoy a wide popularity.

C. E. Rea, M.D.

BOUND COPY OF BULLETIN ON RHEUMATIC DISEASES AVAILABLE

The first four volumes of *The Bulletin on Rheumatic Diseases* have been bound in soft cover and are now available to physicians.

The Bulletin, published by the Arthritis and Rheumatism Foundation, is issued nine times a year and is available without charge to all physicians and medical students who request it.

Physicians wishing to regularly receive *The Bulletin on Rheumatic Diseases* at no cost, should write to Dr. Russell L. Cecil, Medical Director, Arthritis and Rheumatism Foundation, 23 West 45th Street, New York 36, N. Y.

The bound special volume composed of all issues of *The Bulletin* of the past four years, is available from the Arthritis Foundation at the cost of $1.

The Bulletin, the only American publication in the field of the rheumatic diseases, was started in 1950 to foster greater interest in the rheumatic diseases among physicians. Its aim is to keep doctors abreast of the more important clinical advances and changing concepts of the rheumatic diseases promptly, simply and succinctly.

The Bulletin at present is being distributed to 22,500 physicians throughout the United States and tweny-five foreign countries.

"PRESCRIPTION FOR MEDICAL PARTNERSHIPS"

"Prescription for Medical Partnerships," a general information booklet on the business details of establishing and continuing a group medical practice, has been prepared by Dr. Charles Maertz, retired Medical Director of the Union Central Life Insurance Company.

A copy can be obtained by writing to "Medical Partnerships," Field Service Division, P.O. Box 179, Cincinnati 1, Ohio.

General Practice Symposium

Neurodermatitis

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THE commonest disease of the skin is eczema, and the commonest form of eczema is neurodermatitis, which is merely a word to express the relationship between the nervous system and the skin. Various forms of neurodermatitis occur, such as the exudative form characterized by blisters on the fingers or oozing widespread plaques, which may occur in childhood as well as in older persons. The commonest form, however, is the circumscribed thickened plaque called "lichen simplex chronicus." These plaques, single or multiple, may occur in various places or they may become a diffuse process to the extent of exfoliative dermatitis. The patient may be entirely incapacitated and be miserable day and night because of the outstanding symptom of itching, often paroxysmal in nature; many patients are driven to distraction and almost to suicide.

Lichen simplex chronicus occurs in certain types of persons who may have hay fever, asthma or urticarial tendencies. Thickening of the skin results from scratching. This thickening brings on exudation and nonspecific cellular reaction; the tissues are thus changed so that itching results and a vicious cycle is established.

The most familiar site for the appearance of lichen simplex chronicus is along the lateral surface of the thighs, on the shins, about the ankles and on the elbows or knees, where the lesions resemble those of psoriasis. A peculiar form occurs in the nuchal region of women almost exclusively, as well as behind and in the ears; an extremely common form of neurodermatitis, or lichen simplex chronicus, produces the symptoms of pruritus vulvae or ani.

The presence of neurodermatitis around the ankle always brings up the question of whether or not varices are present. One must distinguish neurodermatitis of the lower portion of the legs from so-called stasis dermatitis. Whenever any disturbance occurs in the lower part of the legs, hydraulic factors come into play, and any time the skin is injured, the circulation becomes stagnant in that region. It is rare to relieve these patients of their symptoms by treatment of varicose veins, although the use of an ace bandage is helpful for two reasons, namely swelling is prevented and the site is covered so that scratching is not possible.

When the disease reaches the proportions of exfoliative neurodermatitis, it is extremely difficult to arrive at a satisfactory diagnosis. The contiguous lymph nodes are invariably enlarged and present an almost irresistible demand for biopsy. Other diagnostic facilities should be utilized first, for in such patients the site from which the specimen has been removed for biopsy frequently becomes infected. The microscopic picture of the nodes is usually one of reticulum cell hyperplasia, plus the presence of pigment and fat. Pathologic diagnoses such as lipomelanoreticuloendotheliosis may be returned, although the condition is nothing but a scavenger reaction in the lymph nodes as the result of extensive chronic dermatitis. Biopsy of the skin shows a rather nonspecific picture that may be almost impossible to distinguish from that of psoriasis. A patient who has dermatitis of this magnitude is entirely incapacitated. As already indicated, some patients who have this syndrome have had asthma, eczema, hay fever and urticaria off and on since infancy.

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From the Section of Dermatology, Mayo Clinic and Mayo Foundation.

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In not a few of them, opacities in the lenses will develop that are typical of allergic cataracts. Therefore, one test in these patients should be routine screening by slit-lamp examination to detect such changes in early stages. If such lesions are found, serious restrictions are placed on the patient's activities.

The localized plaque of neurodermatitis may be treated by topical measures. Protective pads, such as an elastoplast cutout, placed over the plaque are sometimes helpful. Tying the hands at night is of aid in some of these conditions, as is any artificial device to break the cycle of the scratching reflex. Systemic measures, such as allergic surveys and dietary restrictions to remove supposedly allergenic foods, are practically worthless. A mistake is often made in prescribing ointments containing a local anesthetic agent, such as dibucaine hydrochloride (nupercaine) or benzocaine, for the relief of itching. Sooner or later, sensitivity to these agents develops and the original condition becomes aggravated and widespread. The same is true in the presence of slight infection, when the prescribing of sensitives, such as nitrofurazone (furacin) is to be condemned. The use of roentgen therapy in moderate doses is helpful; however, excessive amounts of such treatment produce actinodermatitis, which leads to no end of complications.

Dietary control is prescribed for patients who have gained weight; patients who have hypertension should have proper restrictions from the standpoint of their general health. Sometimes in women this condition first appears during the menopausal years; under these conditions, some estrogenic support is helpful. When neurodermatitis becomes widespread and the patient is extremely unstable, some restrictions should be made on the excessive activity. These people usually are hyperactive and like to improve every shining minute. The typical woman who has neurodermatitis never sweeps anything under the rug. She may be on a committee; soon she is chairwoman and soon there are no other members on the committee but this woman—she's the chairwoman and the entire committee. It is useless to attempt to find an ointment in this jar or that to correct the symptoms which are trying to tell us that such a patient is about to blow a fuse. Phenobarbital is helpful but it is also sometimes capable of provoking allergic reactions. Some favorable results have been attained by use of reserpine (serpasil), even in the absence of hypertension, because of the tranquilizing effect of this agent. Chloral hydrate is a useful bedtime sedative.

Topical applications in the acute stage include use of moist compresses of boric acid (saturated solution) or aluminum subacetate. The latter is prescribed as an 8 per cent solution that is to be diluted 1 fluidounce to a pint. It is not used as a wet poultice but as a moist dressing; it is useful in relieving the irritation.

One of the most successful but rather messy treatments is the use of tar. It can be prescribed as an ointment containing 1 or 2 per cent each of crude coal tar and salicylic acid. If tar is used in the hair, it should be incorporated in a water-soluble base.

Dermatitis of the ear canal is often neurodermatitis, and fungi are not involved. It is necessary to clean out the canal frequently. A small wick of moist dressing may be helpful. The most characteristic reaction in this location is intense itching, and the patient perpetuates the condition by scratching. In the treatment of ear conditions, nuchal eczemas and pruritus vulvae et ani, use of an ointment containing 1 per cent hydrocortisone is helpful. The effect of this steroid applied locally wears out in time unless the underlying disturbances are corrected, but it has been a most significant advance in treatment. I have no brief for those who would use cortisone or corticotropin (ACTH) systemically for the treatment of neurodermatitis, even when the condition becomes generalized, because the patient becomes dependent on it and it is difficult to discontinue treatment without a rebound reaction. Occasionally, when the condition is uncontrollable, it is useful to give 15 to 25 mg. of ACTH intravenously as an eight-hour drip in a 5 per cent solution of glucose.

As already emphasized, patients who have widespread neurodermatitis are incapacitated and really need hospitalization for immediate relief. In long-range treatment they need readjustment of their habits of living and sometimes a change of environment. Some of the mañana influence of the Southwest is helpful and the sunlight there is also beneficial.

Differential Diagnosis of Tumors of the Neck

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ANY tumor of the neck, as is one in any other place in the body, is potentially dangerous and should be treated accordingly. The only way to establish a definite diagnosis is by histologic study, which requires biopsy or removal of the lesion. The best time to carry out primary treatment of a tumor in the neck, especially if it is malignant, is at the time of biopsy. Therefore, it is important to use every possible means to establish a preoperative working diagnosis so that the physician can better explain to the patient and his relatives what might be expected in the way of definitive treatment.

It is for these reasons that one always should keep in mind the differential diagnosis of tumors in the neck when a patient is being examined. For instance, if a lesion should prove to be a squamous cell epithelioma metastatic from a primary tumor in the mouth, there is no better time to do the necessary radical dissection of the neck than when the primary lesion is removed. If one strongly suspects that a lesion is squamous cell epithelioma, possibly primary dissection of the neck should be done in preference to biopsy, because biopsy in squamous cell epithelioma may increase the chance of recurrence because of "seeding" at the operative site or persistence of the carcinoma because of incomplete removal of the tumor. Thus in the course of examination of the neck, one should always inspect the lip, the endoral cavity and the larynx, because of the potential danger of metastatic involvement of lymph nodes in the neck arising from primary carcinoma in these regions.

General Aspects of Examination

The examiner must consider a tumor of the neck from the standpoint of the history and the results of inspection, palpation and auscultation of the tumor. If the tumor has been present for many years without any evidence of recent change, that means one thing to the examiner; if the tumor has been present for ten or twenty years and in the past month has shown a sudden increase in size, that means something else; if the tumor was first noted a month ago and is painful and tender, that means yet another thing. One should also note whether or not there has been any difficulty in swallowing, or any recent evidence of hoarseness. One factor that is sometimes misleading is whether or not the tumor varies in size. The patient may say that the lesion increases and decreases in size. It is difficult to judge this because with change of the position of the neck, as in moving the head from side to side, the appearance of the tumor will change; it appears best to ignore this particular symptom.

For inspection, it is helpful to have the patient stand directly in front of the examiner, perhaps 2 or 3 feet away. This establishes whether or not asymmetry of the neck is present; if it is, the tumor is obvious and merely from inspection one should get some idea of its nature. Motion of the tumor up and down as the patient swallows gives certain information, perhaps indicating a nodular goiter. A tumor that is not easily apparent on inspection sometimes becomes prominent when the patient swallows. Also, movement of the head from side to side occasionally makes the tumor more prominent. Any dimpling or attachment of the skin over the tumor should be noted.

Palpation should not be attempted until after thorough inspection. The physician should feel the neck rather carefully, as some people are rather sensitive to such palpation. The tumor preferably should be grasped between the thumb and the fingers. Occasionally, however, one is unable to feel the tumor between the fingers and then it must be palpated against the deeper structures of the neck. At times, one can depress the structures of the neck with the opposite thumb, causing the tumor to become more prominent and more palpable. One should note whether the

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tumor is firm or soft, encapsulated or infiltrative, solid or cystic, tender or nontender, fixed to surrounding tissues or freely movable, in which direction it moves and, if possible, from which structures of the neck it arises. When the lesion is in the upper cervical region, especially the submaxillary triangle, bimanual palpation with the fingers in the mouth is of value. One should also determine the degree of hardness or firmness. A tumor that is calcified is like a rock and usually is benign, in contrast to a firm tumor that feels as though it is infiltrating adjacent tissue; the latter type of tumor frequently is malignant. One should feel for attachment to surrounding tissues.

In the course of palpation, one should notice if there is any pulsation of the tumor. Occasionally, an aneurysm or a vascular tumor might arise from structures in the neck. It is extremely important to know this before exploration of a tumor. Auscultation of the lesion may aid in this diagnosis.

Regardless of how expert the examiner is and how certain he may be that a lesion is of this type or that, the only way to prove the point is to excise the lesion for histologic diagnosis. Then the lesion can be forgotten or the indicated definitive treatment can be carried out.

Tumors of the neck may be divided into congenital lesions, primary tumors, metastatic tumors, tumors of the salivary glands and inflammatory lesions. This presentation is primarily concerned with the characteristics of these lesions that might be of diagnostic value.

Congenital Lesions

Among congenital lesions occurring in the midline of the neck are cysts of the thyroglossal duct. This tumor occurs in the midline and lies either over or below the hyoid bone. If it is much below, the cystic mass may move slightly to the right or the left of the midline. Most often this lesion is definitely cystic; however, if it is tense it is somewhat difficult to establish its cystic nature. If one grasps the tumor and creates pressure on it downward and then feels above the tumor, between the tumor and the hyoid bone, one frequently can feel the cordlike structure that represents the tract from the cyst through the hyoid bone to the base of the tongue; palpation of such a tract renders it fairly certain that the tumor is a cyst of the thyroglossal duct. If the lesion has been previously incised, or if it has previously drained, the character of the discharge is important. A discharge from a cyst of the thyroglossal duct is mucoid in type. A cyst of the thyroglossal duct may not have been noticed in childhood and may be initially discovered in patients sixty or seventy years of age.

Among the lesions in the lateral aspect of the neck are branchial cleft cysts, which occur in the submaxillary triangle, usually just anterior to the anterior border of the sternocleidomastoid muscle. The cystic nature of a branchial cleft cyst can usually be determined. It is superficial, has a smooth surface, is freely movable, unless it is large or has been previously infected, and is not attached to any of the underlying tissues. Sinuses sometimes are associated with branchial cleft cysts if the cysts have been previously operated on and not completely removed. One should always inspect for a branchial cleft sinus or fistulous opening, which occurs along the anterior border of the sternocleidomastoid muscle usually just above the clavicle. The characteristic discharge is mucoid in type. A branchial cleft fistula has no cystic component as a rule. When the course of the tract is put under tension, a cordlike structure can be felt along the edge of the muscle.

Another lateral cystic lesion is a cystic hygroma, which often occurs in the lower portion of the cervical region, especially if it is small. If it is large, it may involve the entire neck. Such a lesion is cystic, soft and multilocular. A ranula is possibly a similar structure occurring in the upper portion of the neck. A ranula frequently can be identified by inspection of the floor of the mouth, where it shows as a watery bleb that disappears when punctured. Such lesions also can be felt in the submandibular region as cystic structures, usually bilateral.

Tumors of the Thyroid

Adenomas of the thyroid are frequently encountered. One should always be suspicious of a thyroid adenoma and should palpate the lateral aspects of the neck in all cases of nodular goiter for possible metastatic lesions. My colleagues and I consider that a nodular goiter is frequently enough malignant so that all nodular goiters should be removed. Whenever a node is found in the neck that has the characteristics of a metastatic lesion, the thyroid always should be carefully palpated. In our experience, about 4 per cent of nodular goiters that are not producing symptoms will contain unsuspected carcinoma.

Occasionally confused with a cyst of the thyroglossal duct is an adenoma of the pyramidal lobe of the thyroid gland. An adenoma may or may not be firm but rarely does it feel cystic. There is no palpable tract above an adenoma as is often noted with a thyroglossal duct cyst. Adenomas usually move up and down when the patient swallows and frequently the presence of other adenomas of the thyroid gland supports the diagnosis of this midline tumor as an adenoma.

Also to be confused with an adenoma of the thyroid gland or a cyst of the thyroglossal duct is a delphian node, which also occurs in this region. A delphian node is a metastatic lesion from a primary carcinoma in the thyroid. When the carcinoma is located in or near the isthmus, one of the first areas of spread is to a lymph node located over the thyroid cartilage; such metastatic lesions, however, are firm, usually small, and freely movable.

Tumors of the Salivary Glands

Tumors of the salivary glands are important in examination of the neck. A mixed tumor of the parotid gland will have been present for a long period; it is hard but yet as a rule it is freely movable unless it is in the deep portion of the gland. If a tumor that has been present for many years shows sudden growth, one has to be suspicious of malignant degeneration. The facial nerve is rarely involved regardless of the size of the tumor unless a malignant tumor is superimposed on the mixed tumor. However, a cylindroma, an adenocarcinoma, or another type of malignant tumor of the parotid gland is not only hard but is usually fixed within the tissue; such tumors frequently cause paralysis of the facial nerve, which is extremely important in evaluating a tumor and advising the patient regarding the treatment that should be carried out. Warthin's tumor, which is cystadenoma lymphomatosum, occurs in the parotid gland; it is soft, usually presents itself below or slightly behind the parotid gland and appears to be encapsulated on palpation. One should not confuse this lesion with a mixed tumor.

Miscellaneous Tumors

Among the primary tumors of the neck are tumors of the carotid body. These lesions occur in about the same area as do branchial cleft cysts but are deeper in the neck and are not cystic. Tumors of the carotid body may be elongated, extending upward and downward; because of their fixation to the carotid vessels, they can be moved laterally but not up and down, which is an important diagnostic finding. Lymphosarcoma occurs in a similar region in the upper part of the neck and in the lower portion of the neck in the internal jugular lymph nodes. Usually lymphosarcoma in the neck involves multiple nodes, frequently matted together. These nodes are usually rubbery in consistency.

A parathyroid cyst has to be considered occasionally, although this is a rare finding. A neurofibroma might occur along the lateral aspect of the neck under the sternocleidomastoid muscle. It is an elongated tumor in contrast to a lymphosarcoma, which is round or associated with a matted group of nodes. Neurofibromas frequently are bilateral. Lipomas are seen usually in the supraclavicular region and the posterior cervical triangle. They are soft and multinodular.

Inflammatory Lesions

Inflammatory lesions of the neck include acute lymphadenitis associated with respiratory and oral infections; this condition does not create a diagnostic problem since the enlargement of the nodes is of recent origin and usually bilateral. If, however, such lymphadenopathy does not disappear within two weeks, it should be regarded with suspicion. The chronic lesions of tuberculosis, sarcoidosis or other granulomas occasionally are confusing; biopsy is required to establish the diagnosis but one should be suspicious of this type of lesion from the history.

Metastatic Tumors of the Neck

Metastatic tumors in the neck are usually of recent origin and are firm and nontender. They may be movable but, if large, may be fixed to the surrounding tissues. Again, when one is suspicious that a tumor may represent metastasis to a node, careful search should be made for a primary lesion on the lip, in the oral cavity, or in the larynx, as well as on the skin of the face or scalp. Lesions of the lip spread in an orderly fashion to the submaxillary nodes in the neck. A node that is the seat of metastasis may be misleading if it has a necrotic center and feels soft. If a

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Fractures of the Upper Extremity in Children

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FRACTURES in children are different than similar ones in adults. Ordinary good treatment produces better results in children than it does in adults. The heavy periosteum that surrounds the bone in children may help to prevent displacement; most of the time it makes for satisfactory manipulation and easier closed reduction. In preadolescent children, the potential for rapid growth will produce an excellent end result even after mediocre reduction. This same growth potential is responsible for the usual speedy union and the rarity of nonunion in children.

This generally optimistic outlook must be tempered by the knowledge that with improper treatment the complications of fractures in childhood can be disastrous. The general principles of treatment are not complex. Alignment is the chief requirement. While it is desirable to produce restoration of length and anatomic apposition, such restoration certainly is not necessary and failure to produce such a result is no indication for open reduction and internal fixation in children. As a rule, completely satisfactory results can be obtained by traction or by closed manipulation and immobilization in plaster. This rule, however, has the usual exceptions. Some fractures, notably certain types about the elbow, often require open reduction. In this discussion, I wish to consider the commoner major fractures of the upper extremity in children and to emphasize those that may be attended by complications or that require open reduction.

Fractures of the Humerus

Fractures of the humerus occur through the condyles, through the shaft, below the tubercles or through the upper epiphysis in about that order of freqency. Fractures of the shaft are rarely accompanied by palsy of the radial nerve and may be satisfactorily treated by application of a hanging cast. If the general alignment and rotation are good, one need not worry about end-to-end apposition or overriding and shortening of up to a half inch; sound union will occur and the proper length will be restored by subsequent overgrowth.

Separation of the upper humeral epiphysis may be treated the same way. Restoration of function and normal bony contours is usually rapid. If the epiphysis is completely displaced, manipulation with the patient under general anesthesia will be required, but even then one need not insist on strict anatomic reduction, and open reduction is rarely required.

Supracondylar fractures are common. Accurate reduction is difficult to achieve and even more difficult to maintain. Neural and vascular complications are more common than the physician usually suspects. Because of the close proximity of major nerves and arteries, in a typical supracondylar fracture the artery or one of the nerves may be impaled on the end of the proximal fragment or actually may be caught between the ends of the fractured bone. Once the diagnosis is established by initial roentgenograms, prompt treatment can be effective. Of prime importance is accurate evaluation of the patient's condition. Simple sensory and motor testing should be done to determine the status of the major nerves. The presence or absence of radial pulsation should be established; if it is present, one should note its quality. Closed reduction is then carried out with the patient under general anesthesia by traction in the long axis of the arm combined with flexion and pronation. It was formerly taught that these fractures should be put up in supination, but I believe the rotary components can be reduced best by putting the forearm up in pronation. Satisfactory reduction can be maintained only by means of a posterior plaster splint that holds the arm in flexion. If this causes dis-

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appearance of radial pulsation, the elbow must be extended until pulsation reappears.

One can deal with these fractures by either continuous traction in bed or open reduction and fixation of the fracture by crossed Kirschner wires. Traction in bed will produce just as good results as will surgical intervention. The greatest danger is Volkmann's ischemic contracture, which presents pain as its most significant symptom. Never disregard the complaint of pain in the hand associated with these fractures. If this pain is accompanied by swelling, coldness, cyanosis or pallor of the fingers, prompt action must be taken regardless of the hour and regardless of the interval of time since the fracture. If extension of the elbow and removal of all circular dressings do not result in a prompt satisfactory return of circulation, block of the stellate ganglion by means of procaine might be considered. However, it is preferable to deal with this situation directly by opening the antecubital fossa and exploring the artery. The artery may be contused, transected or caught between the ends of the bone. Segmental resection of the damaged portion of the vessel relieves the reflex vasopasm and usually results in adequate circulation. Vascular grafting is not indicated in such cases.

Epiphysial Injuries at the Elbow

Epiphysial injuries at the elbow usually are more serious than the clinical picture or the roentgenograms would indicate. Three main centers of ossification are present at the inferior end of the humerus. One is for the lateral condyle, or capitellum, one is for the medial condyle, or trochlea, and one is for the internal epicondyle. At times, there may be a fourth center for the lateral epicondyle. A fall on the point of the flexed elbow may displace the entire epiphysis forward. Usually, however, the fall is on the outstretched hand and one of these centers of ossification is pulled away from the others by muscular traction. When the joint is forced into the varus position, the origin of the common extensor muscles on the back of the forearm may avulse the lateral part of the epiphysis, the capitellum, together with some of the adjacent portion of the trochlea and the end of the shaft. If the aponeurosis is incompletely torn, the condyle simply may be displaced laterally and not rotated. Such a fracture will unite firmly, with good results.

However, if the aponeurosis is completely torn, the condylar fragment may be rotated completely out of the joint and turned upside down. If the fracture is allowed to heal in this position, there will be an increase in the carrying angle and sooner or later ulnar palsy will appear because of stretching of the ulnar nerve. Open reduction is usually required. After the condylar fragment has been anatomically reseated in its bed, it usually can be held there firmly by suture of the surrounding soft tissues of the aponeurosis. Internal fixation of such a fracture is not required.

The epiphysis for the internal epicondyle does not unite until about the age of eighteen; thus, injury to this structure can occur until late in adolescence. Until that time, the origin of the common flexor muscles, which attaches to the internal epicondyle, may avulse this structure when a strain in the valgus position is applied to the elbow. This is an opposite strain from the one that has just been considered. At least four grades of such a displacement may occur. The least serious is a slight separation, with minimal displacement. The most serious is avulsion of the epicondyle associated with complete outward displacement of the elbow. The more serious displacements usually are associated with some damage to the ulnar nerve. Less extensive displacements may be adequately treated by closed manipulation, with immobilization of the elbow in the flexed position. However, when the avulsed epiphysis has been drawn into the joint it is necessary to do an open reduction, securing the fragment in its bed by means of a few sutures. An important point about the roentgenograms in epiphysial injuries is that the films may not tell the story at all as these structures are composed mainly of cartilage and are not well portrayed roentgenologically.

Children who fall on the outstretched hand may impact the head of the radius against the capitellum, causing displacement of the upper radial epiphysis. An important consideration here is that the head of the radius in children must never be excised. To do so will result in radial shortening, radial deviation of the hand and disruption of the radio-ulnar joint at the wrist. The usual displacement of the radial epiphysis can be treated by closed manipulation. When the displacement is more pronounced, it may be necessary to make a short incision and restore the parts under direct vision. In most cases, the epiphysis is perfectly stable after such a procedure. Occasionally, when it is not stable and will not stay in position, a Kirschner wire can be introduced from the back of the elbow through the capitellum into the shaft of the radius to hold its position for two or three weeks until early formation of callus occurs.

Fractures of the Forearm

Fractures of both bones of the forearm occur at the distal third, at the middle third and at the proximal third in about that order of frequency. Open operation is not justified for such fractures regardless of their level. It must be emphasized that no trace of angulation should be present in fractures of the proximal or middle third, for this will not correct itself with growth. Instead, permanent limitation of pronation and supination will result. Greenstick fractures at these levels frequently will be troublesome because of persisting and recurring angulation. In such situations it may be necessary to extend the greenstick fracture and convert it into a complete simple fracture in order to prevent recurring angulation. Some overriding is permissible if good alignment is attained.

Fractures of the upper third of the forearm should be immobilized in full supination and those of the middle third should be immobilized in midposition due to contraction of the muscles. It may be necessary to maintain alignment by using traction on the fingers and one need not fear doing this in children. Perfect restoration of the contours will be obtained in a few months and function as a rule will be normal.

Fractures about the distal third of the forearm may be left unreduced if the angulation is less than 30 degrees. The hazard of leaving such fractures alone is far less than the hazard of giving the patient an anesthetic agent and manipulating the fracture in any way. The distal fragments usually are displaced dorsally. In such fractures, there is a strong tendency for angulation to return even in a well-applied cast. Immobilization of such a fracture should be in a long cast with the elbow at a right angle and the forearm pronated. Molding of the cast is a more effective way of controlling the angulation than trying to put the hand way down in pronounced flexion. There is no justification at all for open reduction.

Fractures of the Hand

Space allows discussion of only one fracture of the hand. One of the common athletic injuries in adolescence is fracture of the neck of the fifth metacarpal. Such a fracture cannot be handled with the finger extended, because with such extension the ligaments are relaxed and the small distal fragment cannot be controlled. If the fracture is allowed to heal in this position, the end of the metacarpal will be down in the palm just like a marble. This type of fracture can be easily reduced, however, by flexion of the metacarpophalangeal joint to an angle of 90 degrees; after its reduction, it can be held that way by means of a plaster slab.

Conclusion

A plea is made for conservatism in treating fractures in children. Nature will do a wonderful job in healing these fractures and normal function will be restored, as a rule, if physicians will simply assist nature to the extent of restoring alignment and providing proper immobilization for an adequate length of time. In some cases, it is almost unbelievable to what extent nature will afford correction of extremely severe fractures in spite of minimal correction obtained at the time of manipulation.

SKIN CANCER

Sunlight may be a major cause of skin cancer. Five fields of evidence point to sunlight as a possible causative factor: "(1) Cancer of the skin of laboratory animals (mice and rats) can be induced by exposure to ultraviolet radiation; (2) cancer of the skin is more common among outdoor than indoor workers; (3) cutaneous cancer is less prevalent in Negroes than in the white races, presumably because the former are less susceptible to sunlight; (4) the incidence of cancer of the skin is greater in regions of the earth that receive high insolation; (5) cancer of the skin occurs principally on parts of the body most exposed to sunlight."—KETTERMAN, H. L.: Sunlight and cancer, *Journal of Kansas Medical Society,* 41:12 (Jan.) 1955.

Management of Recent Injuries of the Hand

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MANY excellent articles have been published about the injured hand. Much of the material included in this presentation was derived from articles published by the great pioneer in reconstructive surgery of the hand, Dr. Sterling Bunnell,[1,2] from a series of bulletins produced by the American Society for Surgery of the Hand[3] and from a recent article by Godfrey.[4] The accompanying sketches outline a plan of procedure that is applicable in the early management of the majority of injuries of the hand.

Wounds of the hand comprise 36 per cent of all industrial accidents. Although no accurate statistics are available, it is probably safe to estimate that a similar incidence is present in injuries around the farm and the home. If an injured hand is to regain useful function, it must be protected from infection and from additional damage to tissues, both of which may be instrumental in converting the hand into a rigid club. This protection is furnished initially by avoidance of meddlesome interference with the wound, cleanliness of the surrounding area, application of sterile protective dressings and immobilization in the position of function. Probing, examination, cleansing and treatment of the wound itself should not be undertaken until adequate surgical facilities are available.

Bunnell noted after examining thousands of crippled hands that six main types of crippling are encountered. These are (1) stiffening of the hand in poor position, (2) flexion contractures, (3) skeletal malalignment, (4) dysfunction from injuries to tendons and nerves, (5) ischemic contractures and (6) trophic or vasomotor conditions. Although the latter two types of crippling usually but not always are due to the initial injury, the first four types are often due to sins of omission as well as commission in the treatment of the injured hand.

Stiffening in Poor Position

Edema, together with immobility, is the commonest cause of stiffness. When these two factors are combined with a position of nonfunction after injuries to the hand, it will stiffen in its poor position. Edema results from many causes but especially from infection. In the presence of edema, fibrin is precipitated about the tendons, muscles and joints. Soon the fibrin is converted into rigid fibrous tissue and the hand becomes a club rather than a finely co-ordinated and delicate piece of machinery. Edema is prevented soon after the injury by elevation and use of compression dressings. These dressings must be voluminous and snug but not too tight. Active motion must be started as soon as it is feasible. The part that is fractured, burned or otherwise damaged must be splinted, but the remainder of the hand must not be immobilized but kept active and moving. The position of function of the hand is that of moderate dorsiflexion of the wrist, moderate flexion of the fingers and moderate abduction and opposition of the thumb. When injuries of the hand are treated, this position must be borne in mind constantly. If for some unpreventable reason the hand should partially stiffen in this position, all function is not lost, whereas if the wrist is flexed, the fingers are straight or clawed and the thumb is adducted, function is lost.

There is no place in the treatment of injuries of the hand for a splint that holds or allows the fingers to be maintained in the extended position. Even in mallet or baseball fingers, although the distal joint is hyperextended in treatment, the midjoint must be flexed so as to tighten the central slip of the extensor tendon and thus approximate the avulsed extensor tendon to the distal phalanx. In an extended finger, its intercapsu-

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From the Section of Orthopaedic Surgery, Mayo Clinic and Mayo Foundation.

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lar ligaments are relaxed and soon become fibrotic in this shortened position. In a flexed finger, the ligaments are tight.

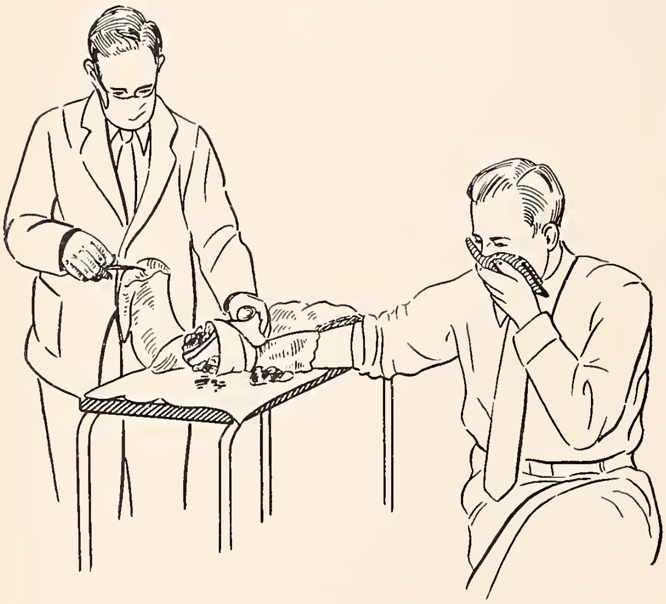

Fig. 1. Wounds of the hand should be covered immediately by a large protective sterile dressing. The nose and mouth of the physician or first-aid attendant and the patient should be covered in an attempt to prevent further contamination of the wound.

Infection as a Cause of Stiffening in Poor Position.—Infection produces edema, pain on motion and thus immobility and a poor position, which is assumed for protection. Therefore, all possible precautions should be taken to prevent infection. A mask should be worn by all who come in contact with an open wound of the hand (Fig. 1). A large protective sterile dressing should be applied to the hand immediately after injury. The wound should not be probed or fully examined until this large first-aid dressing is removed and all arrangements have been made to proceed with the necessary surgical care (Fig. 2). The patient must be taken to the hospital immediately, as time is an important factor in preventing infection.

It is unsafe to repair deep structures in a wound of the hand if bacteria have had an opportunity to multiply for more than 8 to 10 hours after injury. The wound should be gently but thoroughly cleansed with soap and water and then completely excised or débrided; tendons, nerves and bones should be carefully preserved (Fig. 3). Fresh wounds have nonvirulent contamination and heal well if circulation, closure, compression dressings and rest are adequate. Tendons and nerves should not be repaired primarily in crushing, explosive or grinding injuries.

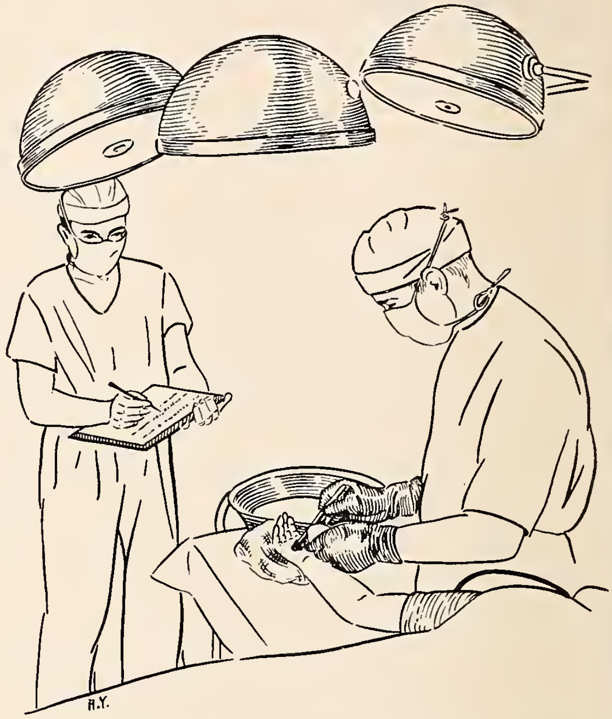

Fig. 2. The first-aid dressing is not removed nor is the wound thoroughly inspected until the patient is in the operating room and plans have been made to proceed as necessary. Careful evaluation of the injury is then made and recorded and a plan of reconstruction is decided on. The hand and forearm are shaved, washed with soap and water and the wound is thoroughly irrigated with normal saline solution. A pneumatic-cuff tourniquet is applied.

Every effort should be made to attain effective and early closure of the wound. The rotation of flaps of skin and grafting of the donor site, using the skin of a filleted finger or an immediate pedicle flap from the abdominal wall, may be necessary (Fig. 4). Thus, months of drainage from infection and its concomitant crippling may be avoided. Especially is it important to cover all vulnerable parts such as joints, bones, tendons and nerves, which are so suspectible to infection. On the other hand, the closure must not be under tension. If a wound is too dirty to close, it may be packed open for five to seven days and closed secondarily.

Flexion Contractures

Flexion contractures are due to contracted scar tissue resulting from a burn or an open infected wound. Prevention, therefore, involves utilization of all available means to close the wound. In burns, the deep bed is usually in good condition for grafting. Often the burn has become demarcated to such an extent that the destroyed skin can be excised four or five days after injury and a split-thickness skin graft can be applied at the same time or a few days later. Granulation tissue, if present, should be scraped off so as to apply the skin on a good bed that will not contract later. The borders of either a free or a pedicle graft should be zigzag and should not cross a flexion crease in a straight line. The hand and wrist must be splinted in a position of function if contractures are to be avoided (Figs. 5 and 6).

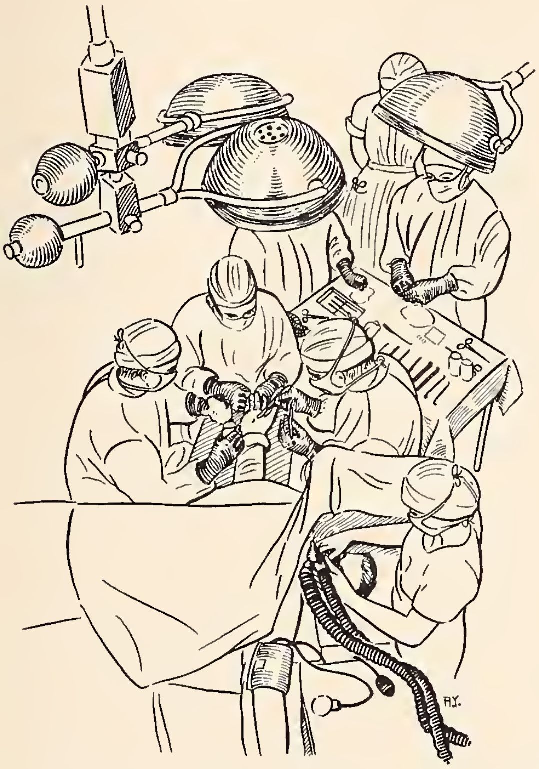

Fig. 3. Definitive treatment requires a well-equipped operating room, good lighting, adequate instruments, sufficient assistance, complete anesthesia and a bloodless field as supplied by the pneumatic tourniquet. Strict asepsis, with use of masks, gowns and gloves, helps prevent contamination of the wound with more virulent organisms. The wound is thoroughly débrided but all usable parts are saved, especially nerves and tendons. Fractures are reduced, the digits being placed in the position of function so they will be opposable.

Skeletal Malalignment

Skeletal malalignment must be prevented by restoration of normal function of the bony structures at the earliest possible time. Restoration of normal position is accomplished by manipulation, skeletal traction or open reduction. Regardless of whether a fracture or a dislocation is being manipulated, full relaxation, preferably induced by having the patient under general anesthesia, is desirable. Manual traction, pressure and molding should be performed gently and deliberately to avoid further injury to soft tissues. If reduction is not promptly successful, operative (open) treatment is in order, since less harm is done by gentle surgical procedures performed through a correct incision than by rough and forceful closed manipulation.

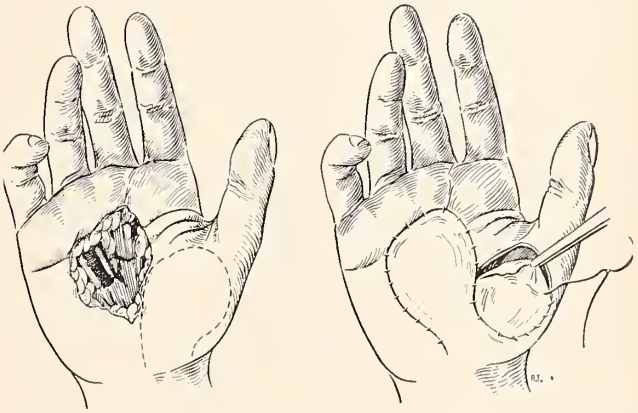

Fig. 4. A cover of skin is supplied if at all possible, and usually it is, by swinging a flap as illustrated and applying a split-thickness graft to the resultant defect, by application of a split-thickness graft directly to the defect in selected instances or by use of a direct pedicle graft from the abdominal wall. If a finger has to be discarded, its skin with vessels and nerves may be utilized for cover.

To maintain reduction, particularly of oblique or comminuted fractures of the phalanges or metacarpals or fractures into joints, control by skeletal fixation may be required. The hand and the injured finger or fingers should be supported in the position of function. Not much traction is needed for fractures of the metacarpals and phalanges. The pull must be applied with the finger held semiflexed and never straight. Use of banjo splints is to be condemned. Rotary malunion of digits is avoided by checking to see that the plane of motion of each finger passes through the navicular tubercle and by observing the plane of the nail. Stiffening of adjoining fingers is avoided by splinting only the one that is injured. All uninjured parts must be kept moving. The cast should hold the wrist in dorsiflexion; it must be molded to the metacarpal arch and should terminate at the distal flexion crease and the thenar crease in the palm so that the proximal finger joints are free to flex.

For many metacarpal fractures, temporary pinning with Kirschner wires cut off beneath the skin to avoid infection is an excellent method of fixation; the hand does not stiffen, as it is not in a cast. The wiring or plating of fractures of the bones of the hand generally is unsatisfactory. Healing of fractures of the long bones of the hand requires immobilization for three to five weeks; healing of fractures of carpal bones requires twelve to fourteen weeks. Fractures of the navicular may require four months for union. If immobilized consistently for these periods, most of these fractures will not require surgical intervention.

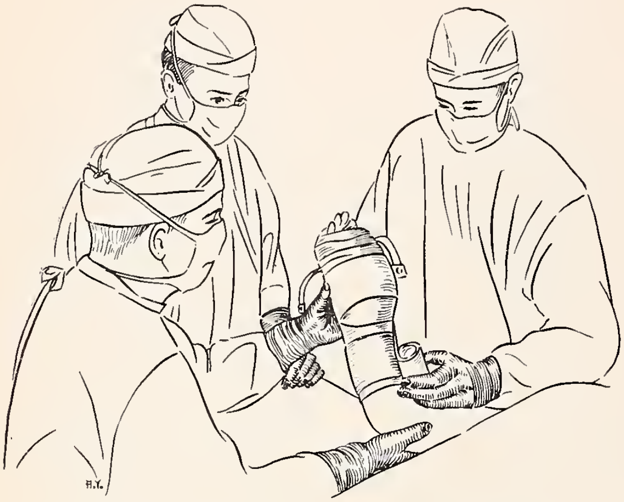

Fig. 5. Swelling is avoided by fluff compression dressings that are applied just tightly enough, but not too tightly, to the hand, which is in the position of function with the finger tips exposed so that circulation may be evaluated. Straps for elevation are incorporated in the dressing.

Injuries to Tendons and Nerves

The dysfunction from injuries to tendons and nerves is best prevented by repairing these structures early, within a few hours, and establishing a good cover of skin. Success depends on obtaining primary healing. In wounds more than eight to twelve hours old or in badly contaminated, dirty or explosive wounds, primary repair of tendons and deep structures must be omitted or serious infection will follow. The surgical repair must be done through proper incisions, with minimal trauma. The best conditions for healing are accomplished by avoidance of dead spaces, hematomas, buried sutures, especially catgut, and tight suturing. For the average surgeon it is best to unite the ends of nerves with one suture of stainless-steel wire or silk to prevent retraction of the ends, thus facilitating secondary resuture under the best of conditions later. However, when primary suture of a nerve is performed expertly, the regeneration is sooner and better than that obtained after late repair. Nerves can and should be successfully repaired down to the distal flexion crease in the finger, including even the tiny motor branches within the hand. Injury to the nerves in the arm or brachial plexus is just as crippling as is injury to the hand itself and repair of these nerves should take precedence over that of bones and joints. When this is not done, the paralyzed muscles and distal portions of the nerves undergo such irreversible fibrous atrophy that restoration of function is impossible. A paralyzed hand needs to be kept mobile and in the position of function or it becomes a useless claw in a position of nonfunction. The patient should be instructed to keep the paralyzed hand in the position of function by gently manipulating it with the good hand. Painful and tender neuromas are avoided by minimizing formation of scar tissue. Amputation should be done in noninfected fields and the endings of nerves placed in good tissue.

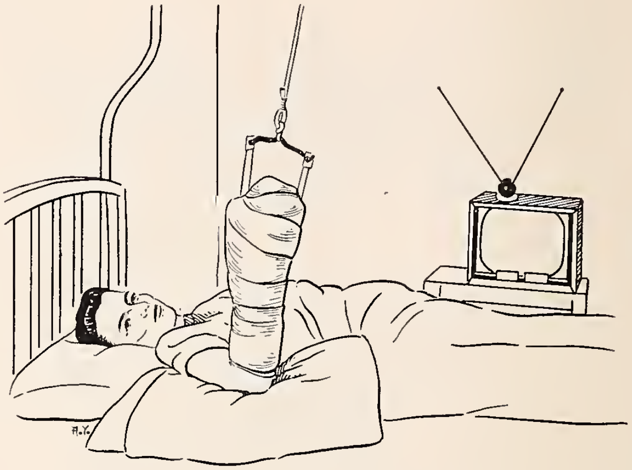

Fig. 6. The bandaged hand is elevated and rest is enforced until the acute reaction of the injury is past, which usually takes about three days.

Ischemic Contractures

Ischemic contractures, both of the Volkmann type and the local type in the hand, usually are preventable by avoidance of tight casts or dressings. If the patient continues to complain of severe pain a few minutes after the cast or dressing is loosened and if there is evidence of impaired circulation in the hand, emergency surgical treatment is necessary if the severe crippling that results from prolonged ischemia is to be avoided.

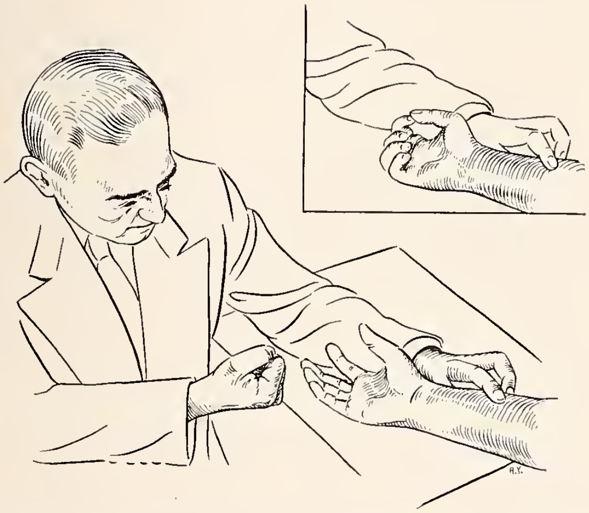

Fig. 7. Supervised active, not passive, motion not only of the uninjured parts but also of the injured part must be started as soon as healing will permit.

Trophic or Vasomotor Conditions

Some patients appear more inclined to experience so-called Sudeck's atrophy and the tendency to causalgia than do others. These persons usually have unstable sympathetic nervous systems. Early recognition of such patients and encouragement in whatever activity may be required to make these people use their hands during every waking moment usually will abort the development of this extremely painful and disabling complication (Fig. 7).

Summary

Review of the six main types of crippling of the hand, namely stiffening in poor position, flexion contractures, skeletal malalignment, loss of motion and sensation from injuries to tendons and nerves, ischemic contractures and trophic or vasomotor conditions, leads to the conclusion that many of these complications can be ascribed to treatment and, therefore, are preventable.

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TUMORS OF THE NECK

(Continued from Page 295)

tumor is invading surrounding structures, it may be tender and make the examiner think of an inflammatory lesion.

If a patient has a hard, nontender node or nodes in the lower deep jugular region or supraclavicular region (Virchow's, or sentinel, node) on the left, one must be suspicious that these are secondary to a primary tumor of the lung, stomach or kidney.

Incision for Lesions of the Neck

In removal of a tumor of the neck for examination, it is extremely important always to use a transverse rather than a vertical incision. A transverse incision heals nicely and is not a cosmetic problem later. Any incision that is made vertically in the neck crosses the natural cutaneous lines and results in a puckered scar. For a large tumor in the neck or when an extensive resection is being done, such as a radical dissection of the neck, a vertical component to the horizontal incision is usually necessary.

Comment

Regardless of how careful the examiner may be, his impression of a tumor of the neck is just a guess until he proves the diagnosis by biopsy. Since many of these tumors are malignant, cosmetically deforming and potentially infected, all should be surgically investigated when discovered. The attitude, "The tumor is not bothering you, why bother it?" should not prevail.

Precautions in the Use of Cortisone for Treatment of Rheumatic Diseases

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CORTISONE and related hormones, such as hydrocortisone and corticotropin, are the most potent antirheumatic agents known at present.* Unfortunately, these hormones also are capable of producing metabolic effects that often are undesirable and, indeed, sometimes temporarily harmful, especially if excessive doses are employed.

For optimal results, the use of these hormones in the treatment of rheumatic diseases must be attended by certain precautions, which can be divided into three convenient categories: (1) selection of the proper disease for treatment, (2) selection of the proper patient having one of these diseases, and (3) selection of proper dosage and attention to other details of management during treatment.

General Precautionary Measures

Selection of the Rheumatic Disease.—Cortisone is most commonly indicated in selected cases of the following rheumatic diseases:

1. Rheumatoid arthritis and its variants, such as rheumatoid spondylitis, Still's disease, Felty's syndrome, Reiter's syndrome, psoriatic arthritis and the arthritis associated with chronic ulcerative colitis.
2. Acute rheumatic fever.
3. Systemic lupus erythematosus.
4. Periarteritis nodosa.

In a second group of diseases cortisone occasionally may be helpful but its systemic administration is usually not indicated, since other and simpler measures ordinarily suffice. These diseases include:

1. Arthritis of hypersensitivity states, such as serum sickness, in which antihistaminic drugs, epinephrine, salicylates, local application of heat, and rest for the affected joints usually are adequate.
2. Acute gouty arthritis, for which colchicine is the drug of choice.
3. Acute nonspecific bursitis, tendinitis and the shoulder-hand syndrome; for these conditions, physical medicine, salicylates and, in certain cases, roentgen therapy or local injection of hydrocortisone generally are sufficient.

Cortisone should not be employed for:

1. Specific infectious (septic) arthritis.
2. Osteoarthritis.
3. Primary fibrositis.
4. Psychogenic rheumatism.

In osteoarthritis of peripheral joints, intra-articular injection of hydrocortisone may produce temporary symptomatic relief and is often useful, especially for acute or subacute flare-ups, but prolonged systemic administration of cortisone should be avoided.

Selection of the Patient.—If it has been established that a co-operative patient has a disease for which cortisone may be indicated, the next decision concerns the suitability of its use in the particular patient. Two principal considerations arise in this regard. The first is whether the disease in this particular instance is of sufficient activity and severity to warrant the use and expense of hormonal therapy or whether the patient would respond equally well to simpler treatment. For example, many patients who have mild rheumatoid arthritis can be managed satisfactorily by a general program, such as physical therapy, salicylates, extra rest, protection of joints and other supportive measures. Secondly, does the patient have some other condition that might be aggravated by cortisone and that, therefore,

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From the Section of Medicine, Mayo Clinic.

*Reference will be made primarily to cortisone in this presentation, although the remarks apply generally to hydrocortisone or corticotropin.

might contraindicate its use? Absolute contraindications to prolonged usage are psychoses, Cushing's syndrome, and tuberculosis and certain other infections not readily susceptible to control by currently available therapeutic agents, as acute poliomyelitis. Relative contraindications include renal and cardiovascular insufficiency, peptic ulcer, psychoneuroses, osteoporosis, diabetes mellitus, convulsive disorders and certain severe intercurrent infections.

The decision of whether or not to employ cortisone in the presence of one of these relative contraindications requires balancing the possible benefits of cortisone against its possible hazards in each particular patient. For example, one might elect to avoid use of cortisone if a patient has a moderate degree of rheumatoid arthritis but a peptic ulcer that is difficult to control, whereas one might employ cortisone for severe, incapacitating active rheumatoid arthritis in the presence of a peptic ulcer that could be controlled by careful medical management. Occasionally, the pros and cons are so evenly balanced that only cautious trial of treatment may allow a definite decision.

Selection of Dosage and General Management. Studies Prior to Treatment.—Before cortisone is used, the patient should receive a complete medical examination, a roentgenogram of the thorax, serologic tests for syphilis, urinalysis, determination of hemoglobin and leukocytes in the blood and other tests pertinent to evaluation of the disease or complicating conditions. The physician should discuss thoroughly with the patient the potentialities and limitations of treatment with cortisone and should stress the necessity for frequent rechecks and co-operation in following directions carefully.

Choice of Hormone.—Doses of cortisone, hydrocortisone or corticotropin required to produce equivalent antirheumatic effects differ somewhat. On the average, the required amount of hydrocortisone given orally may be about 20 per cent less than that of cortisone, although in practice individual variations are considerable.[1,2] It is even more difficult to estimate comparative doses of corticotropin owing to differences that result from such factors as the preparation used, the route of administration and the variable activity of the adrenal cortices. However, increased antirheumatic activity of these hormones is accompanied by increased metabolic activity, affecting electrolytes, water, protein, fat and carbohydrate. No one of these three hormones has proved consistently superior to the others in the avoidance of hypercortisonism.

Examination During Treatment.—During administration of the hormones, the patient should be examined periodically with regard to the effect of treatment on control of the disease and to the presence or absence of hypercortisonism. The frequency of periodic rechecks depends on the disease and the individual circumstances. The patient who is treated for acute systemic lupus erythematosus with large doses of cortisone needs to be checked clinically at least daily; laboratory tests, such as urinalysis and measurement of serum electrolytes, sedimentation rate and hemoglobin, may be required frequently. On the other hand, the rheumatoid patient treated with small doses of cortisone may need to be seen only at weekly intervals initially, then later at intervals of two to four weeks, at which time the clinical status should be evaluated carefully and weight and blood pressure recorded; necessary laboratory tests may be minimal, such as urinalysis and measurement of hemoglobin and leukocytes every two or three months, and roentgenograms of the thorax every six to twelve months.

Careful clinical observation of the patient is of more importance than are laboratory tests in the management of the patient receiving cortisone, especially in the detection of hypercortisonism. Such evidence of hypercortisonism as mental stimulation, increased appetite, gain in weight, facial rounding, retention of fluid and appearance of supraclavicular fat pads usually occurs long before any laboratory signs of hypercortisonism.

Regulation of Dosage.—Regulation of dosage is the most important phase of careful treatment. Attitude toward dosage should be conditioned to a great extent by the disease under treatment and the anticipated duration of hormonal therapy.

For acute self-limited but potentially damaging diseases, such as acute rheumatic fever, relatively large doses are employed to accomplish quick suppression of the inflammatory reaction; then a gradually lowered but completely suppressive dose is maintained for the relatively brief course of the disease. For example, one might give to a young adult with acute rheumatic fever 200 or 300 mg. of cortisone daily for a few days until clinical signs of acute rheumatic fever have disappeared, after which the dose might be reduced to about 100 to 150 mg. daily for the next two or three weeks. Then slightly lower doses, perhaps 50 to 100 mg. daily, might be employed for the next few weeks. Thereafter the dose might be tapered off in a period of another week or two or longer if necessary. Recurrence of activity may call for temporary increase in doses. Signs of hypercortisonism in the presence of apparently complete suppression of the disease would indicate the probable desirability of some reduction in dosage. Obviously, dosages and duration of treatment should be adjusted to the needs of the individual patient; the variation in severity and duration of the disease does not permit a routine schedule of treatment. When such large doses as just outlined are employed, a low-salt diet and the administration of potassium chloride (for example, 2 gm. three times daily) are advisable.

For subacute and chronic diseases typified by occasional acute, potentially fatal flares, as for example in systemic lupus erythematosus, extremely large doses of 300 to 1,000 mg. or more per day may be employed temporarily during the acute stages; subsequently the dose is tapered off so that during the chronic stages little or no cortisone is employed. If the use of cortisone is necessary during the less acute stages, the dosage should be regulated to avoid hypercortisonism in much the same manner as will be described in the discussion of rheumatoid arthritis.

For chronic diseases requiring indefinitely long treatment, as in chronic rheumatoid arthritis, the dose should be kept low enough to avoid hypercortisonism.

Dosage in Rheumatoid Arthritis

The objective in the treatment of rheumatoid arthritis with cortisone or related hormones should be to achieve as much antirheumatic effect as possible without producing hypercortisonism.

Problems of Chronic Hypercortisonism.—If large doses of cortisone, hydrocortisone or corticotropin are administered to a rheumatoid patient, antirheumatic effects may be maintained whether or not signs of hypercortisonism appear. However, in some patients maintenance of excessive doses producing hypercortisonism eventually leads to serious complications, such as osteoporosis with fractures, peptic ulcers, pronounced emotional disturbances and excessive retention of fluid. Extreme fatigability, emotional lability and depression and diffuse aches and pains unlike but often confused with rheumatoid symptoms may be noted.[4] Reactions simulating systemic lupus erythematosus or periarteritis nodosa have occurred in several cases.[5] At present, one cannot predict in which patients significant complications will develop if hypercortisonism is permitted to persist. Therefore, doses should be regulated to avoid hypercortisonism in all patients.

Policy on Dosage.—A policy of restriction of dosage so as to avoid hypercortisonism implies that doses will be smaller than those employed previously by many physicians. As a consequence, less than complete antirheumatic effects may result in many cases. Necessary compromises in dosage may permit suppression of only 50 to 75 per cent of symptoms. Relief of this or greater magnitude generally can be achieved in at least 50 per cent of cases, often more easily in men than in women. In about 35 per cent of cases satisfactory regulation of dosage may be more difficult, while in about 15 per cent continuous adequate control of rheumatoid arthritis with tolerable doses is difficult or unsatisfactory.

Initial Doses.—My colleagues and I virtually have eliminated the use of large initial priming doses in our present treatment of rheumatoid arthritis. Starting doses are the approximate amount estimated as reasonable for prolonged maintenance, governed by the activity of the inflammatory reaction and the patient's expected susceptibility to hypercortisonism. The following daily doses of cortisone often are employed initially: for men, 40 to 60 mg.; for postmenopausal women, 20 to 30 mg.; for other women, 25 to 40 mg. Doses of hydrocortisone are slightly less. Sometimes the chosen initial dose may prove insufficient, in which case a small increment (5 to 10 mg.) may be made temporarily.

Reduction of Dose.—As soon as antirheumatic response is definite, gradual reduction of dose is attempted. Decrements generally are from 2.5 to 5 mg. when the dose is 50 mg. or less, or from

5 to 10 mg. when the daily dose is more than 50 mg. If a previous reduction has been well tolerated, the dose may be reduced again within a week or so. In some patients, reduction must be made less often. If the response to the previous reduction has been less favorable, another decrement may be delayed for several weeks or as long as necessary for the patient to readjust to the new dose, unless further reduction is dictated by other circumstances, such as the presence of hypercortisonism.

Maintenance Doses.—The maintenance dose is not a fixed dose. Ideally, it is that variable and usually decreasing amount required to maintain relief without producing hypercortisonism. Thus maintenance refers to relief, not dose. The proper dose for maintenance of relief varies from patient to patient and from time to time in the same patient; hence, frequent individual attention is required. Occasional and repeated attempts at reduction should be made even though the patient appears to be well stabilized at a given dose, since a smaller dose may accomplish as much. If the reduced dose proves insufficient, the former dose can be reinstated, perhaps for one to three weeks or so, before reduction is again attempted. Eventual discontinuation of medication by gradual reduction should be the aim.

For mild or moderate flares, the dose often can remain unchanged; extra amounts of acetylsalicylic acid, physical therapy and rest may suffice. If necessary the dose may be increased by 5 mg. for mild or moderate flares and 10 mg. for more severe flares; a second increment may be given later if required. The dose is reduced again as soon as the flare begins to recede.

Experience suggests that daily doses for prolonged treatment generally should not exceed the following limits: for men, 50 to 60 mg.; for postmenopausal women, 30 to 35 mg.; for other women, 40 mg. Hypercortisonism develops in some patients who receive less than these doses; others can tolerate slightly larger doses. In case of increased stress or pronounced articular flare, temporary use of larger doses may be required.

Division and Spacing of Daily Oral Doses.—For most patients the daily oral dose of cortisone should be divided into four more or less equal parts and one part should be given every six hours; a typical schedule might be 10 mg. at 6 a.m., noon, 6 p.m., and midnight. The duration of antirheumatic effect of cortisone sometimes is sufficiently long to permit the patient to take the last dose of the day at bedtime rather than having to be roused from sleep to take the final dose at a later time.

Other patients may be treated more satisfactorily by varying the size of individual doses during the twenty-four-hour period so as to provide a little more cortisone for times when symptoms are likely to be worse. Thus, for a man whose symptoms are particularly severe during the morning but much less severe in the afternoon and evening, a program of 20 mg. at 6 a.m. and 10 mg. at noon, 6 p.m. and midnight may achieve better results than a program of 12.5 mg. every six hours, although the total dose would be the same in each instance.

Flexible Program of Dosage.—Many rheumatoid patients experience varying degrees of symptoms from day to day during an average week. For some such patients it may be better to employ a flexible program in which the patient takes a slightly different amount for "average" days, "good" days and "bad" days. Thus, instead of a daily maintenance dose of 45 mg. during the week (total weekly dose of 315 mg.), such a patient might more effectively take 40 mg. daily for three average days, 35 mg. daily for two good days and 45 mg. daily for two bad days (total weekly dose of 280 mg.). Such a program not only supplies more cortisone when needed but also reduces the amount given at times when less is required. In this way smaller total doses may be successfully employed. This plan requires especially intelligent co-operation by the patient, as well as careful check by the physician to insure that supplemental doses are not employed excessively or unnecessarily. The patient is not permitted to regulate his own dosage but rather is permitted a choice within a slight range of dosage predetermined by the physician.

Supplementary Measures.—Appropriate supplementary treatment, such as physical therapy, salicylates, adequate rest, protection of involved joints and other supportive measures, should be continued during administration of cortisone.

Management in Case of Stress

The pituitary-adrenocortical mechanism is inhibited by long-term treatment with cortisone or corticotropin and, therefore, it cannot react normally to produce increased quantities of needed adrenocortical hormones in case of stress. Thus, in order to prevent the serious consequences of adrenocortical insufficiency in severe stress, sufficient additional hormone must be administered.[3,6,7] For minor stress, such as a mild intercurrent infection or a minor surgical procedure performed with the patient under local anesthesia, the maintenance dose of cortisone should be continued or rarely increased slightly, perhaps by 25 to 50 mg., for the duration of the stress; careful clinical observation should reveal any need for additional cortisone.

In major stress, such as a major surgical operation, serious injury or severe intercurrent infection, even more additional cortisone must be given. In elective major surgical treatment, my colleagues and I give 200 mg. of cortisone intramuscularly forty-eight hours, twenty-four hours and one hour before operation. On the day after operation, 100 mg. is usually given if the patient's condition is satisfactory; within the next two to three days the dose is gradually reduced to the preoperative maintenance level or its use is discontinued. Oral administration is resumed postoperatively whenever it is conveniently possible.

For more urgent surgical procedures or other emergencies, 200 mg. or more of cortisone is given intramuscularly as soon as possible; this dose is repeated after operation and as needed thereafter. In addition, a dose of 100 to 200 mg. of hydrocortisone in solution is given intravenously in 250 cc. of isotonic solution of sodium chloride during operation or for rapid effect in other emergencies, to be repeated as needed during or after operation.

Other precautions also are advisable to minimize the effects of major stress in these cases. Operation should be performed early in the day to avoid prolonged fasting and other stress incident to waiting. If intravenous administration of fluids is required, the use of glucose alone in distilled water should be avoided; isotonic solution of sodium chloride or such a solution containing 5 per cent glucose should be used instead. Morphine is poorly tolerated by patients who have adrenocortical insufficiency; the use of meperidine hydrochloride (demerol hydrochloride) appears preferable.

During and after operation or other stress, the patient should be watched closely. Blood pressure and pulse are checked at least hourly for twenty-four to thirty-six hours. If signs of adrenocortical insufficiency develop, such as weakness, tachycardia, hypotension, vascular collapse or respiratory failure, 100 to 200 mg. of hydrocortisone should be promptly administered intravenously in 250 cc. of isotonic solution of sodium chloride; this injection should be repeated as often as necessary to maintain normal blood pressure. A dose of 200 mg. of cortisone should also be given intramuscularly for later supportive effect. Oxygen, blood transfusions and vasopressor drugs, such as norepinephrine, 4 mg. in isotonic solution of sodium chloride, may be employed as indicated if shock develops.

The afore-mentioned precautions are employed not only for patients currently under treatment with cortisone but also for those who have received significant amounts of the hormone within the past three to six months, or even within the past year if hypercortisonism has been present. In patients treated with cortisone less recently, prophylactic use of cortisone often is deemed unnecessary. However, in such cases the other precautions just described are observed just as in the case of the patient who receives cortisone, and a solution of hydrocortisone is kept available for emergency intravenous use.

Patients treated with cortisone or related hormones should carry cards similar to those carried by diabetic patients to inform those who might care for them in an emergency that special care and additional doses of hormone may be required.

Conclusion

This presentation purposely has stressed the possible hazards and necessary precautions during administration of cortisone in the treatment of certain rheumatic diseases. In viewing these hazards, one must not overlook the benefits that result from use of cortisone in certain carefully selected, properly managed patients who have one or another of the rheumatic diseases for which

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Digestive Ailments of Older Patients

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AN AGING population is rapidly developing and more patients are living longer and remaining in better health at present than at any time in history. Among this group, gastrointestinal complaints are extremely common and varied. The literature bearing on the field of geriatric gastroenterology is scanty and individual experience is limited. Since degenerative cardiovascular renal disease and cancer loom so large as causes of death, it is natural that the bulk of the literature on the aging process has concerned itself with these two problems while the gastrointestinal tract has been almost forgotten. Only in recent years have gastroenterologists become interested in this problem. Those who have written about it have agreed in emphasizing (1) the fact that many of the gastrointestinal complaints are due to curable or correctable lesions, (2) the variation in symptomatology from the usually accepted classic clinical syndrome of the disease in question and (3) the opportunity which the family physician has in dealing with this group of patients.

The Pathologic Physiology of Aging

The changes which occur in various organs of the body as a result of the aging process have been fairly well documented, the exception being the gastrointestinal tract. There are minor degenerative changes of the salivary glands with a reduction of ptyalin secretion. Since digestion of starch begins in the mouth, these changes may be reflected in the development of fermentative diarrhea. The esophagus itself is usually seen to resist the passage of time but in some very old persons there may be either atony or unco-ordinated peristalsis with resulting dysphagia. The lowering of gastric acidity with age has been well studied and it is probable that about 10 to 12 per cent of older persons have permanent anacidity. Some atrophy of the gastric mucosa has been reported but this finding has been contradicted by recent gastroscopic biopsy studies that show essentially normal mucosal structure in the stomachs of many older people. Radiologists tell me that there is little evidence of a loss of gastric tone or motor activity in the aged. So far as the small intestine is concerned, the little that is known about it would indicate that no significant changes take place in it as a result of aging. The same statement cannot be made of the colon, in which loss of muscle tone and propulsive activity is frequently seen. This problem is frequently compounded in older persons by the development of rectal lesions that interfere further with normal intestinal function.

So far as the accessory organs of digestion are concerned, some falling off in organ weights may occur but little else has been observed. The liver may show some abnormal pigmentation of its cells and fibrosis around the portal spaces but the cell mass as a whole appears to be normal and such liver functional data as exist would indicate that the physiologic capacity of the liver is not greatly disturbed. Much the same statement can be made with regard to the pancreas.

With regard to the abdominal vasculature, aging may be a basis for such lesions as mesenteric thrombosis. However, existing evidence appears to indicate that degenerative vascular disease of the abdominal aorta and its branches lags somewhat behind the same changes in other portions of the body.

I shall have relatively little to say about cancer and will, in the main, deal with the benign gastrointestinal diseases, for many of which there are satisfactory and acceptable forms of treatment.

Case-taking in Geriatric Gastrointestinal Disease

As in pediatrics, diagnoses in old patients must be based largely upon objective evidence and upon a sound general knowledge of what is likely

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Read at the meeting of the Minnesota Academy of General Practice, Rochester, Minnesota, October 20, 1954. From the Palo Alto Clinic.

to be wrong. The histories in older patients, almost all of whom have some digestive complaints, may be grossly misleading. Many elderly individuals are willing to blame their complaints on "something I ate," upon supposed allergies and upon other ailments that they have been known to have for a long time. Many of them probably minimize their symptoms because they are afraid of what might be found. Finally, many of them are unwilling to discuss the personal, social and economic difficulties which may play a large part in the production of their symptoms. For this reason one may recommend the general practice of first getting acquainted with the patient, then seeing what else is wrong with him (since multiple diagnoses are the rule in the aged), and finally considering the gastrointestinal complaints.

The well-established practice of obtaining a history of the complaints for a single typical day is helpful; of even greater value is the presence of a relative or friend who may often supply the pertinent details. Finally, it is wise to make an attempt to see any acute episodes of distress which these oldsters may have. A visit to the old man or woman in the patient's own home and the observation of an episode of the illness at first hand may be of greater value than even the most detailed studies in the hospital. The advantages that the family physician has in respect to these factors are obvious.

Common Gastrointestinal Diseases of the Aged

I shall attempt to refrain from statistics so far as is possible. However, I have drawn freely upon Monroe's experiences at the Geriatric Clinic of the Peter Bent Brigham Hospital, upon some observations kindly made for me by Dr. Roger Egeberg and his colleagues at Wadsworth Hospital in Los Angeles and upon the personal experiences of some of my own colleagues. It seems well to begin at the entrance to the digestive tube.

Diseases of the Esophagus.—There are probably more erroneous diagnoses made in respect to esophageal disease than any other field of gastroenterology, the reason probably being that it is not often recognized how frequently esophageal disorders will produce symptoms referable to the abdomen. Cardiospasm is a relatively rare condition in older persons, but carcinoma is common enough and should be the first consideration in any elderly person with a short history of dysphagia. Both of the former are overshadowed in importance by one common ailment, namely, hiatus hernia, with or without peptic esophagitis. Such hernias are common in old people; in fact, evidence indicates that there may be a relative relaxation of the esophageal hiatus which increases with age. The diagnosis frequently can be made from the history alone, the most common feature of which is nocturnal or postural pyrosis and regurgitation of food, along with varying degrees of transitory dysphagia and of pain in the epigastrium and left upper quadrant. The diagnosis is clinched by roentgenologic study. If one is to get maximal help from the radiologist, however, one must tell him what is suspected.

It is remarkable how often older patients with hiatus hernias are admitted to the hospital with a history of bleeding; probably at least a third are anemic when first seen. Actual esophageal ulcers are not infrequently seen when they are sought for, usually in the herniated portion of the stomach or in the distal portion of the esophagus. Probably patients can bleed from esophagitis and gastritis alone without gross ulceration.

Diverticula of the esophagus are not especially rare. It is noteworthy that they infrequently cause symptoms until the later years of life. The important diverticula to identify are those at the pharyngo-esophageal juncture. Such diverticula, when filled with food, may set off a chain reaction of aerophagia and belching; hyperventilation may be associated with the condition as well. Auscultation of the neck during the act of deglutition may be of diagnostic value.

Traction diverticula of the middle third of the gullet are of lesser importance, while those of its distal portion are extremely rare. It is important to exclude esophageal lesions in patients who have supposed cardiac disease. Barium studies, so widely used by radiologists in studying the outlines of the heart and aorta, have in many instances served to establish an entirely different diagnosis, namely, that of esophageal disease.

Diseases of the Stomach and Duodenum.—Since gastric acidity decreases with advancing age, it was the general belief about 30 years ago that ulcers of the stomach and duodenum would, therefore, disappear or become quiescent. The situation is different today and it is now recognized that either acute or reactivated ulcerative disease of the stomach and duodenum is relatively common in patients more than 60 years of age. In his afore-mentioned studies, Monroe found that 6.4 per cent of the more than 8,000 admissions to the Geriatric Clinic were for peptic ulcer; in only about half these cases was the usual long and classic history obtained. In one home operated by the Veterans Administration, 60 per cent of all gastrointestinal admissions from the home to the hospital are because of ulcer problems involving obstruction, bleeding or penetration and almost universally requiring surgical consultation and study.

Some interesting points are found with regard to the location of peptic ulcers in older patients. In the population as a whole there are probably ten duodenal ulcers to every gastric ulcer. In the aged, this ratio falls to about 3.5:1. Some correlation may exist between gastric ulcer and degenerative arterial disease, which may be of clinical importance, especially in connection with the cause of gastric ulcers and the seriousness of bleeding from them.

The patient with a long-standing ulcer history who experiences reactivation of his ulcer diathesis in later years presents no particular problem. There is a sizable group of old patients, however, who present their first ulcer symptoms in the seventh decade of life. In such patients bizarre symptoms appear to be the rule rather than the exeception.

The usual sequence of pain with an empty stomach and ease with food often does not appear, being replaced by symptoms having little relationship to those usually described by ulcer patients. A common story is that of isolated attacks of severe pain unrelieved by anything short of narcotics and not infrequently associated with vomiting and bleeding. Other patients may complain of pain aggravated by the taking of food or of periodic vomiting.

Complications, as already indicated, are almost the rule in the aged. Of these the most serious is bleeding, which not infrequently comes out of a clear sky. Duodenal ulcers apparently bleed a little less often than do ulcers on the gastric side. Men have more hemorrhages than women. Both the incidence and gravity of hemorrhage appear to increase with age, with a corresponding effect upon mortality. The true incidence of hemorrhage is difficult to determine, figures of from 5 to 30 per cent appearing in the literature; in Monroe's series, the latter percentage obtained. It is difficult to estimate the severity of hemorrhage in the aged, and a high incidence of cardiac and cerebral thrombosis follows hemorrhage, even that of moderate degree.

Obstruction is noted in about 10 to 15 per cent of old patients with ulcer, slightly more often with gastric than with duodenal lesions. Many of these patients have symptoms and roentgenologic findings suggestive of cancer. Obstruction may appear suddenly and with little in the way of antecedent history. Persons who have acute obstruction are a particular problem and may need hospital study with repeated checks of gastric retention and radiologic studies before a final diagnosis can be made. It should be pointed out that imbalance of electrolytes and azotemia develop with extreme rapidity in older patients after either hemorrhage or obstruction and that neglect of fluid balance for as little as a day or so may result in serious complications. Most of these older patients with pyloric obstruction can be handled well by simple surgical procedures, such as gastroenterostomy with or without excision of the ulcer. Fortunately, perforation occurs only about half as often as obstruction. A pronounced sex difference is noted, perforation being five times as common in men as in women.

What impresses gastroenterologists in dealing with the older ulcer patient is the bizarre symptomatology, the high incidence of complications and the relatively high mortality and morbidity rates; the persistence of the ulcer diathesis beyond the seventh decade of life is also impressive. Obviously, we should make roentgenologic studies of the stomach on more old patients even if the history is bizarre; we must be particularly quick to investigate a history of gastrointestinal bleeding, even if the amounts of blood lost are small. Finally, if the patient is seen for the first time with hemorrhage or other complications which might be attributed to ulcer, immediate investigation is compulsory.

One final point is stressed by gastroenterologists who have dealt extensively with ulcer in older patients. Treatment should never be continued for any length of time on the basis of symptomatology alone without confirmatory roentgenologic studies. Even after examination has revealed a lesion that appears to explain the complaint, and even if symptoms are benefited by medical management, further roentgenologic studies after a lapse of time are necessary. The high incidence of neoplastic disease in the stomach should warn us not to be misled by a solitary roentgenogram that shows a supposedly benign lesion.

Diseases of the Gallbladder.—Thirty years ago, when disease of the gallbladder was suspected in all middle-aged patients who had any sort of dyspepsia, a good many unnecessary cholecystectomies were done. Over the years the pendulum has swung in the opposite direction and today even cholecystography is often omitted unless the patient has had fairly classic symptoms referable to the biliary tract. As a result, a great many persons are going into the later years of life carrying gallstones which are not suspected until a catastrophe of some sort occurs.

The increasing incidence of gallstones with age requires only brief mention. Women apparently form most of their gallstones between their middle twenties and the menopause. In men the process starts later and continues into the eighth and ninth decades of life. The end result is that the incidence of stones in the two sexes becomes nearly equal in the very old. Many of these stones remain completely asymptomatic for life; others produce their first and only symptoms in advanced age. It is well to remember that gallstones are probably the commonest single cause of serious abdominal pain in patients more than 65 years of age and that neither the location nor the exact character of the pain is of much diagnostic value.

Should the accidentally discovered calculous gallbladder be removed in older patients? Is a single bout of pain sufficient indication for radical treatment? These questions are practical and the answer is not always simple. When statistically considered, the answer must be in the affirmative.

If one checks present hospital mortality rates from gallstones against the generally accepted surgical risk, it will be found that the mortality rate of stones in older patient is much higher than the least optimistic figures on surgical risk. In old patients the complications of gallstones and associated disease are jointly responsible. In Monroe's series there were many patients with cholecystenteric fistulas, gallstone ileus, common-duct stones and obstructive cirrhosis, to say nothing of associated pancreatitis; in the same series, the incidence of vascular complications was high. About 44 per cent of the patients who had gallstones had associated nonvalvular heart disease and 10 per cent had had previous cerebrovascular accidents.

Thus, gallstones are a distinct hazard to life which increases rapidly with age. Surgical mortality rates run a parallel course, although as a rule the old patient with gallstones stands operation well and there are few contraindications. The mortality rate of all patients with gallstones, according to Monroe, is materially increased above the hospital mortality rate of all old patients by at least 10 per cent. In his series, 4.5 per cent of the "medically treated" patients with gallstones died in the hospital solely as a consequence of their biliary disease and 8.5 per cent died from gallstones with complicating disease elsewhere in the body.

Diseases of the Small Intestine.—Regional ileitis is rare at all ages and decidedly uncommon among the old. The disease appears to present no special features in the later years of life. Whether healed ulcerative or granulomatous disease in early years leaves any residues that persist into the later years of life is not known. I recall three instances of intestinal strictures from my own experience; one caused obstructive symptoms of some severity while the others caused difficulty of a relatively mild degree. In two the origin was unknown; the third was associated with polyarteritis.

Diverticula in the small intestine, not uncommon in the aged, interest the radiologist but are rarely productive of serious symptoms. Duodenal diverticula in particular are probably of no great clinical importance, although a few are known to have produced mechanical difficulties chiefly because of their large size. Meckel's diverticula are rarely considered in diagnosis, yet I know of a recent case in which one produced obstruction in a 92-year-old man. This lesion was successfully resected and the obstruction relieved.

Diseases of the Colon.—If one arbitrarily excludes cancer from the discussion, only a few organic conditions involving the colon remain to be considered in the older age group. Ulcerative colitis is rare in geriatrics, yet an occasional case is found even in the aged.

One sees on histories a great many diagnoses of diverticulitis of the colon. Diverticula themselves are as common after 50 years of age as gray hair or wrinkles; they rarely produce symptoms until they are called to the patient's attention. In private practice a really significant attack of diverticulitis is something of a rarity.

One sees frequently on radiologic examination of older persons a narrowed cicatricial strip of intestine beginning in the descending colon and extending into the sigmoid. These lesions are probably truly the consequences of previous diverticulitis. They may simulate cancer, although their length and the rather indefinite terminal margins they present argue against such a diagnosis. In a few patients, cicatricial obstruction of the region may occur which can be relieved only by surgical excision of the diseased segment of intestine. It has been said that lesions of this type do not bleed, yet I am sure that some must do so. One woman, aged 83, with this radiologic picture bled from the intestine for years in quantities such as to require frequent transfusions. In spite of many roentgenologic studies and two laparotomies, no other cause for the bleeding point was ever found.

Much has been said and written about polyps in the colon. The small multiple polyps so frequently seen during sigmoidoscopy are easily fulgurated and removed. Larger single, adenomatous polyps, which produce symptoms by mechanical blockage of the intestine or by ulceration and hemorrhage, are not uncommon in older patients. Since many of them are potentially malignant, their removal must be advised, but in my experience the expected cure of the vague abdominal complaints so commonly associated with them does not materialize.

Some special mention must be made of appendicitis in the aged. Dr. Egeberg has commented on the frequency of appendiceal rupture in old patients associated with only moderate inflammatory reaction. These ruptures are apparently caused by distal obstruction incidental to cancer, the cicatricial residues of diverticulitis, volvulus or perhaps even constipation. There is no more difficult diagnosis than that of appendicitis in an old person. The symptoms are bizarre and the peritoneal reaction minimal; suspicion frequently is the only possible basis for diagnosis. All too frequently a diagnosis is not suspected until an abscess develops.

The major problem referable to the colon in old patients is constipation. No bulk formers, lubricants or antispasmodics will get much of a performance out of the old colon. What one should attempt to do is persuade the patient that the intestine need not be emptied every day and that no physical harm will result from an overdue stool. Such arguments leave most old people unmoved, and it is a rare person who has not worked out his own salvation by means of various laxatives, suppositories or enemas. It takes a hardy physician to attempt to correct some of these notions and no amount of talking can divert some oldsters from their preoccupation with their bowel habits.

The diarrheas of old people are troublesome. In some instances, they are associated with achlorhydria and probably also with deficient intestinal absorptive capacity. Such difficulty may be relieved in part by parenteral administration of liver extract and use of vitamin concentrates. Orally administered antibiotics may be responsible for setting the stage in many such patients. In others, no apparent explanation is available and one has little to offer except purely symptomatic measures. The nature of such diarrheas is obscure; much further study of the subject is needed.

Hepatic and Pancreatic Disease.—Cirrhosis of the liver does not frequently present itself as a clinical problem in the later years of life. This is probably due to the fact that the peak incidence of cirrhosis as a cause of death is past at age 60. Also, many types of cirrhosis are inconsistent with long life. Hemochromatosis and biliary cirrhosis (often described as xanthomatous) usually have run their course long before the seventh decade. Somewhat the same statement may be made in regard to chronic hepatic atrophy, an especially lethal ailment in postmenopausal women. What remains, therefore, is chiefly nutritional cirrhosis and the cause is, in most instances, alcohol plus malnutrition. The incidence of the disease varies with the location and dietary habits of the community.

As might be expected, the complications of cirrhosis, chiefly hemorrhage from varices and episodes of hepatic necrosis, are highly fatal in old persons. Some fortunate patients, however, appear to achieve a balance between cellular destruction and repair, with the result that the aged patient may carry on successfully. I recall a number of older patients with cirrhosis who presented every evidence of gross hepatic damage and yet survived for years in a reasonable state of health. One man to whom a fatal prognosis was given at the time of his admission to the hospital lived for 10 years, dying of arteriosclerosis. One should not despair of the older cirrhotic; he deserves as vigorous treatment as the younger patient and not infrequently will reward these efforts by a long and reasonably comfortable life.

Viral hepatitis is no respecter of age. Epidemiologic study appears to indicate that persons in middle life and beyond have a fairly solid immunity to the naturally acquired disease but this conveys no immunity to the serum-borne type. Probably because of this fact and the likelihood that hepatitis in the aged may be due to virus SH, by far the more dangerous type, the prognosis in the old patient with hepatitis should be guarded. The state of the patient's nutrition and the presence or absence of associated disease elsewhere in the body may be determining factors in the outcome. It is wise to study the old person with jaundice with great care, since the difficulty of diagnosis between obstructive and parenchymatous jaundice apparently increases with age. The patient who has a definitely obstructed biliary tree cannot be explored too promptly, whereas the patient with primary hepatic cellular disease must be kept out of the operating room.

Chronic relapsing pancreatitis appears to be relatively rare in older persons but it does occur often enough to keep one constantly reminded of its presence. Pancreatic edema associated with common-duct stone is, of course, frequently seen and is relieved when the offending calculus is removed. Two patients recently seen with episodes of hemorrhagic pancreatic necrosis were in the seventh and eighth decades of life. Both recovered after a long and stormy illness characterized by imbalance of electrolytes and azotemia. It is encouraging to be able to tell these patients that only about half of those who have this syndrome will have a recurrence. Surgery in such patients is probably better avoided, but one must make an exception for the patient with a pancreatic cyst. The suspicion of cancer is ever present in this group of individuals but it appears well established that the more violent episodes of illness are most often associated with the benign form of pancreatic disease.

Functional Complaints of the Aged.—These are legion even in persons who have an underlying organic disorder. In these patients one has to pick up the trail of an organic disorder out of the maze of bizarre symptoms. One must beware of the alleged functional complaint in patients more than 45 years of age; this statement is doubly true for patients more than 60 years of age. One learns to be cautious in commitments made to these patients and to advise re-examination if the character of the symptoms changes.

There are, however, innumerable older persons who always have had digestive disturbances that simply grow in number and magnitude with the passing of years. Among these, certain types stand out, such as the bolters of food and the swallowers of air, who usually can be distinguished by their ability to belch at any time and as frequently as desired; another type is the confused oldster who has himself imagined or has been led to believe that he has food allergies or sensitivities and who as a result has restricted his diet severely. To convince these people of the error of their ways and to get them back on a full and proper diet require diplomatic skill of the highest order. A third group, and perhaps the commonest of all, are persons with so-called mucous colitis. It is a fortunate physician who does not have a group of these unfortunate patients under his care. To the best of my knowledge there is no dietetic program nor any combination of drugs which will restore peristaltic activity to normal in the colons of these individuals. With persuasion and guidance, some of them improve, but more often they drift disconsolately from one physician to the next, obtaining little relief from any.

Finally, some comment must be made about the organic psychoses of old age and their tendency to produce digestive symptoms. Minor cerebrovascular accidents, encephalomalacia of arteriosclerotic origin and even simple reactive depression may produce somatic digestive symptoms of a most bizarre type. The depressive episode may pass and the digestive complaints disappear, but with organic cerebral damage, prognosis for recovery is not good. If any help is to be obtained for these older persons with functional digestive complaints, it is usually at the hands of the wise family physician who knows

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Management of Acute Abdominal Diseases

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THE diagnosis and management of acute diseases of the abdomen continue to be among the most perplexing problems that confront the surgeon today. Regardless of the tremendous strides in modern medicine, considerable room for improvement exists in this field. It must be remembered that the first successful closure of a perforated peptic ulcer was done less than 50 years ago and that removal of a diseased appendix has been a practical surgical procedure only for about the same time. Laparotomy for intestinal obstruction was regarded until comparatively recently as a procedure of last resort.

Needless to say, the most important and also one of the most difficult problems is that of arriving at the correct diagnosis. While it is not within the scope of this paper to discuss the taking of a history and the performance of the physical examination, it should be emphasized that extreme care is needed at this crucial stage. Among the several diseases that simulate an acute surgical condition of the abdomen are cardiac derangements, chronic interstitial nephritis, pneumonia, porphyria, hydronephrosis, typhoid fever, tuberculous peritonitis and tabes dorsalis. The latter three diseases are not seen nearly so often as formerly. For some time it has been the policy of my surgical colleagues and me to require as routine on all candidates for emergency surgical treatment a certain minimum of laboratory studies, including a complete blood count, urinalysis, thoracic roentgenogram, a scout (preliminary survey) film of the abdomen when indicated, and determination of the blood group and Rh factor. Other studies may be done depending on the particular problem at hand; these include such things as roentgenograms of the abdomen with the patient upright or in the lateral decubitus position to determine the presence of free air, and chemical determination of values for serum amylase and lipase. Such studies are of considerable value when used in the proper situation, but they should not be ordered indiscriminately as they are time-consuming and expensive.

Stomach and Duodenum

Acute hemorrhagic peptic ulceration, either gastric or duodenal, is a most formidable problem. Constant observation of the pulse, blood pressure and value for hemoglobin is necessary in addition to the usual administration of blood and other supportive fluids. Massive bleeding must be brought under control without delay. The patient who has such hemorrhage presents a considerably different problem than does the usual type of bleeder. The mere induction of anesthesia may prove fatal as a result of aspiration of large quantities of regurgitated blood. During the past two years my surgical colleagues and I have worked in close co-operation with the anesthesiologists and have established a routine method of management in these difficult situations. Since its inception, the surgical mortality rate has been greatly reduced.

Our present method of surgical management of bleeding peptic ulcer is as follows: two 15-gauge needles are inserted into the veins even if it is necessary to cut down on the veins to accomplish this. It is thought that complete and adequate control of two veins is of utmost importance in the maintenance of an adequate circulating blood volume. The systolic blood pressure should be maintained at more than 80 millimeters of mercury in order to be well above the minimal pressure for renal filtration and thereby forestall development of lower nephron nephrosis. The abdomen is opened after local infiltration with procaine has been accomplished. The stomach will be greatly distended and filled with blood and clots; therefore, the stomach is opened and evacuated immediately so that the patient can-

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From the Section of Surgery, Mayo Clinic and Mayo Foundation.

The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

not regurgitate large quantities of blood. The anesthesiologist then puts the patient to sleep and inserts an intratracheal cuffed tube. Thus, with complete control of both the circulatory system and the airway, the surgeon can proceed with the operation with relative ease and considerably less risk to the patient.

The next problem is to bring the bleeding point under control by either suture or ligature; once this is accomplished, the condition of the patient will become stabilized fairly well. Of course, blood is being replaced all the time, and after the bleeding has been stopped the surgeon can proceed with an adequate and satisfactory gastric resection of whatever type is preferred.

Perforated peptic ulcers have had considerable attention recently due to the advocacy of conservative methods of management by some workers. For the most part, my colleagues and I continue to treat perforated ulcers surgically. At present no attempt is made to close a perforated gastric ulcer, since it cannot be determined immediately whether such a lesion is an ulcer or a carcinoma. Therefore, these lesions should be resected whenever possible.

Perforated duodenal ulcer presents a somewhat different problem. The diagnosis of an acute perforated duodenal ulcer is made from the criteria of previous ulcer history, sudden onset of acute abdominal distress, free air in the abdomen and an increased value for serum amylase. The serum amylase is increased usually to 500 or 1,000 units in perforated duodenal ulcer; this finding is often important in differentiating this disease from acute pancreatitis, in which the value for serum amylase is considerably higher, often ranging from 3,000 to 4,000 units. As soon as the diagnosis of perforated peptic ulcer is made, the patient should be prepared for operation unless very definite localizing signs are demonstrable.

Such preparation consists of immediate gastric intubation to rid the stomach of its contents and prevent further leakage into the abdomen. In addition to this, the patient receives parenterally large doses of atropine or methantheline bromide (banthine) to further inhibit gastric secretion and also to inhibit pancreatic secretion. The patient should receive large doses of antibiotics, with morphine or some similar narcotic to allay the pain; intravenous administration of fluid or blood tranfusions are given as necessary to treat shock or to restore proper hydration. The perforation is then promptly closed.

Many times the abdominal cavity will be greatly soiled with contents from the stomach and duodenum, and severe chemical peritonitis will be present as a result of the gastric acid, bile and pancreatic juice liberated into the peritoneal cavity. Under such conditions, the peritoneal cavity should be lavaged profusely with large quantities of saline to remove such material and reduce the extent of chemical peritonitis. A mixture of penicillin and dihydrostreptomycin is left in the abdomen to combat the growth of any bacteria that might have been introduced into the cavity. As the result of use of this method, there have been no deaths at the Mayo Clinic in the last two years from perforated peptic ulcer.

In addition to this regimen, more perforated duodenal ulcers are now being resected than before. Many patients who have had simple closure of a perforated duodenal ulcer will return later because of reperforation, obstruction or hemorrhage. If primary resection will spare the patient a second major surgical procedure, the surgeon probably is obligated to do this whenever possible. However, primary resection should not be used indiscriminately, and candidates for the procedure should be picked with great care.

A number of factors enter into the selection of patients for primary resection of a perforated duodenal ulcer. The age of the patient should come into consideration, but no hard and fast rule can be set; a 65-year-old patient may be in unusually good condition for his age. The general condition of the patient is important; the physician must use his clinical judgment in evaluating the condition of each patient. The amount of soilage in the abdomen should be considered. If the patient is having a great amount of shock resulting from severe chemical peritonitis, the surgeon should not add to that shock by attempting a long procedure, such as gastric resection. The duration of the perforation is also to be considered. If the perforation has been present for some time, much contamination has occurred and the patient's condition may not be good; under such conditions, mere closure of the ulcer should be done. The condition of the duodenum is of prime importance. This will determine whether or not adequate closure of the duodenal stump can be done. If the tissues are in poor condition, friable, or edematous, if much fore-shortening is present, or if the perforation is extremely large and occupies a major portion of the wall of the anterior surface of the duodenum, then it is not wise to attempt resection and run the risk of a draining duodenal stump in addition to the many other associated problems.

Gallbladder

Difference of opinion continues as to when operation should be performed in acute disease of the gallbladder. Patients with this disease must be carefully individualized. Emergency surgical intervention does not have to be considered for patients who are merely having minor attacks. Patients having attacks severe enough to necessitate admission to a hospital or repeated injection of narcotics for relief of pain present a considerably different problem. Such patients may be evaluated on the basis of several factors, among the most important of which are (1) the duration of the present attack, (2) the severity of the present attack, (3) the number of previous attacks and (4) the association of pancreatitis.

If the duration of the present attack has been as short as twenty-four hours or so, it is advisable to observe the patient for a while longer, perhaps an additional twelve to twenty-four hours. This allows time to show improvement, and in the meantime proper medical management can be instituted. At the end of this period if the pain and other symptoms are less and the temperature is on the decline, then the program should be continued. If, however, the condition of the patient has not improved or has become worse, then surgical intervention is indicated. At the end of this period, the attack has not been of sufficient duration for the surgical procedure to be technically difficult because of inflammatory reaction. On the other hand, if the attack has lasted five or six days, the affected tissue by this time has become so brawny and ligneous that cholecystectomy is extremely hazardous and difficult and should not be attempted if it can possibly be avoided. Thus a duration of five or six days is the upper margin of safety beyond which operation is not recommended until after the attack has subsided.

The number of previous attacks may play an important role in determining the best form of treatment for a particular patient. If the patient is seen in a severe attack and has not had previous gallbladder disease, that patient should be operated on immediately. It is largely among this group that free perforation of the gallbladder is found, associated with bile peritonitis. This is due primarily to the fact that the adjacent abdominal structures have not yet had an opportunity through repeated minor attacks and resultant inflammation to wall off the area around the gallbladder and thus prevent so-called free perforation. Approximately 4 per cent of all acutely inflamed gallbladders will perforate.

In the event of associated pancreatitis, which is evidenced by pain penetrating through to the back and by an increase of serum amylase, surgical intervention should be postponed if at all possible. This is especially true since these patients may have a stone in the common bile duct, which would necessitate exploration of the duct. This is technically difficult in the presence of severe inflammation; therefore, operation should be delayed if at all possible until after the attack has subsided sufficiently to allow the inflammation to subside, which usually takes two or three weeks after onset of the disease.

Pancreas

Acute pancreatitis continues to be a most difficult and often lethal problem. A rigid abdomen, a shocklike picture and greatly increased values for serum amylase and lipase, in the absence of free air in the abdomen, lead to the diagnosis of acute pancreatitis. In the past, several procedures have been offered for use in surgical management of this disease. Among these are drainage of the common bile duct, drainage of the region around the pancreas and splitting of the capsule of the pancreas. These methods have not proved to be of sufficient value to recommend their routine use. Instead, a properly managed conservative regimen appears to offer considerably more than does surgical intervention in this disease and does not add to the already existing shock of the patient.

As soon as the diagnosis of pancreatitis is made, all efforts should be directed toward the suppression of pancreatic function, general support of the patient and the relief of pain. The patient should receive nothing by mouth and continuous gastric suction should be instituted. This removes the gastric acid as a source of pancreatic stimulation. Use of morphine and codeine should be avoided as these drugs frequently may cause spasm of the sphincter of Oddi, thus hindering pancreatic drainage. Other drugs for the relief of pain should be used, however, in sufficient doses to give adequate relief. Large doses of atropine or banthine given parenterally should be used to suppress gastric as well as pancreatic function. Massive doses of broad-spectrum antibiotics should be given, as sterile abscesses frequently form from necrosis of tissue and pancreatic digestion and these may be secondarily infected by bacterial invaders. Adequate amounts of fluid and blood should be given parenterally as indicated. This program has proved to be satisfactory and has greatly reduced mortality rates in the management of this disease.

Small Intestine

Emergency problems of the small intestine may be divided into three main groups, namely (1) those related to Meckel's diverticulum, (2) those resulting from mesenteric vascular occlusion as the result of either thrombosis or embolism and (3) obstruction from such conditions as adhesions, congenital bands, volvulus, tumors (either benign or malignant, such as polyps, lipomas, myomas, adenocarcinomas and carcinoids), and foreign bodies, such as ingested articles and gallstones.

Meckel's diverticulum may give symptoms from bleeding, obstruction or perforation. The diagnosis usually is made by exclusion, as there are no good pathognomonic signs to indicate the lesion.

Mesenteric thrombosis may be acute or insidious in onset depending on the causative situation. The existence of ventricular fibrillation often may give a clue but this is not necessarily a prerequisite for mesenteric vascular occlusion. Venous thrombosis may be a more frequent cause of mesenteric vascular occlusion than is arterial embolism; the former may occur on the basis of cardiac failure with resultant decompensation and diminished venous return. The affected tissue must be resected; otherwise the outlook is extremely poor.

The diagnosis of intestinal obstruction may be made on the following criteria: intermittent crampy pain, borborygmi at the height of the pain, nausea, vomiting, interference with bowel movements, abdominal distention, localized abdominal tenderness and roentgenologic evidence. Certain factors contribute to a late diagnosis in acute intestinal obstruction. These include failure to recognize intestinal colic, failure to auscultate the abdomen, frequent lack of local findings, confidence in enemas, apparent effect of laxatives and premature use of morphine.

Whenever possible it is always well initially to try conservative methods in the management of simple intestinal obstruction. Such methods include giving nothing by mouth, intestinal intubation, parenteral administration of fluids, hot abdominal stupes, inhalation of oxygen, blood transfusion when necessary, warm gentle rectal irrigation and close observation, including blood chemical determinations and roentgenograms of the abdomen.

Obstruction of the small intestine presents many varied problems. Adhesions and congenital bands are the cause in the great majority of cases. In the case of adhesions, medical decompression by means of a Miller-Abbott tube or some other type of long intubation tube, if possible, is preferable to surgical procedures because the latter only produce more adhesions.

Volvulus of the small intestine presents an extremely serious problem. Often an indication to the correct diagnosis may be noted on a scout film of the abdomen. A characteristic roentgenologic picture is the so-called almond-shaped deformity, which is pathognomonic of a closed-loop obstruction of the small intestine, or so-called volvulus. Once this picture is seen, the patient should be operated on immediately; otherwise gangrene of the intestinal loop will develop, necessitating a much more extensive procedure, with resection of a segment of the small intestine.

Tumors that produce obstruction must be removed surgically. It is best, however, to decompress the intestine as much as possible beforehand, as this will make the operative procedure considerably easier for both the surgeon and patient. Resection of the small intestine may be unusually dangerous, especially when one is forced to attempt anastomosis in a distended edematous friable segment. In order to put the tube into the small intestine as far down and as rapidly as possible, it is often necessary to push the tube into the small intestine through the pylorus with the aid of fluoroscopic examination. Tubal decompression may greatly improve the integrity of the tissues with which the surgeon is forced to work.

Foreign bodies occasionally may cause intestinal obstruction; many of them can be noted on a scout film of the abdomen. Ileus produced by a gallstone, however, is an extremely difficult problem to diagnose preoperatively at times, yet to the careful observer the diagnosis frequently may be made from the scout film. The stone itself seldom may be seen; however, the presence of free air in the biliary tree is pathognomonic of gallstone ileus. This finding means that the patient should be operated on immediately, because it is indicative of air that has escaped into the biliary tree through the fistula between the gallbladder or the common bile duct and the small intestine. The stone usually will lodge in the distal 5 or 6 cm. of the ileum, with a marked amount of inflammatory reaction around it and a considerable degree of distention above it. When the stone is found it should be milked proximad away from the inflamed and edematous intestine up to a normal segment, where a small incision can be made through the wall of the intestine, the stone removed and the incision closed with minimal difficulty. One per cent of all obstructions of the small intestine are due to gallstones.

Large Intestine

Acute emergencies involving the large intestine may be generally divided into three main groups, namely (1) those resulting from appendicitis, (2) those resulting from diverticulitis and (3) those resulting from obstruction from other causes. Appendicitis will not be considered in this presentation because relatively little recent change has occurred in the management of this disease.

Diverticulitis is primarily a disease of middle and old age. Five to 10 per cent of persons more than forty years of age have but do not suffer from diverticulosis. Of these persons, one in 300 experiences diverticulitis. Diverticulosis is twice as common among males as among females but diverticulitis has an equal sex incidence. Diverticulitis is located in the sigmoid in 75 per cent of cases. Emergency surgical procedures for this condition are indicated only in the presence of free perforation or obstruction. A transverse loop colostomy to divert the fecal stream is usually all that should be done initially, and more definitive surgical treatment should be planned for a later date. Rarely the affected region may be exteriorized. Primary resection of the perforated obstructed segment is difficult because of the severe inflammatory reaction and seldom can be accomplished. Other conditions of the colon that necessitate emergency surgical procedures are volvulus and closed-loop obstruction.

Carcinoma of the sigmoid and rectosigmoid appears to be the commonest cause of closed-loop obstruction in the presence of a competent ileocecal valve. A closed-loop obstruction may be readily recognized on a scout film of the abdomen by the severe distention of the colon, with apparent absence of gas in the small intestine. These patients should be followed with extreme care since perforation of the cecum may be imminent. Impending cecal perforation may be diagnosed by the following criteria: progressive cecal enlargement as evidenced by roentgenologic studies, pain and tenderness over the cecal region, rebound tenderness over the cecal region and an increase in pulse rate. Many times in the presence of closed-loop obstruction, the first symptom may be pain in the right lower quadrant, which will draw attention away from a lesion in the sigmoid or rectosigmoid and may cause it to be overlooked. The reason for such pain with an obstructing lesion in the sigmoid or rectosigmoid is that the cecum is the thinnest and most easily distensible part of the intestine, which is why it also is the commonest point of perforation.

When the afore-mentioned findings are noted, immediate surgical decompression is compulsory. In our experience the procedure of choice in this instance is transverse loop colostomy. Some surgeons favor use of appendicostomy or cecostomy. It has been our experience that an appreciable number of the stomas established by these latter procedures do not function in a satisfactory manner and failure to function at this crucial time may prove fatal.

Thousands of people are abroad in the country with tuberculosis which is arrested or inactive following treatment. Antibiotics have played a large part; and we hope these people will remain well for many years, but how many will relapse, we do not know. How many may at some time produce tubercle bacilli, which may be a danger to the community, we do not know. Will the antibiotics lose their effectiveness as the number of resistant cases increases in the community? Again, we do not know. It is such uncertainties that lead the pessimists to say that tuberculosis, like the poor, will be always with us.—George J. Wherrett, M.D., National Tuberculosis Association Transactions, 1954.

Hematuria

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MODERN physicians are fully aware of the potential seriousness of hematuria and advise patients who have such a condition to undergo prompt and thorough examination. The tragedies that occur from delayed diagnosis of malignant lesions of the genitourinary tract often can be ascribed to the patient, who either does not seek advice immediately after hematuria appears or ignores the counsel of his physician. The public must be educated to the seriousness of hematuria.

Hematuria always demands a qualifying adjective, namely either gross or microscopic. Gross hematuria always means an abnormal or pathologic situation. On the other hand, the finding of microscopic hematuria always brings up the unanswered question with regard to whether or not a few erythrocytes may be present in the urine of normal persons. The bulk of evidence accumulated over the years would indicate that, with ordinary techniques of urinalysis, the presence of more than two erythrocytes per high-power microscopic field in a centrifuged specimen is abnormal. Of course, many variables enter into this conclusion, such as the accuracy of the technician, the speed of the centrifuge and the specific gravity of the urine. Dilute urine contains fewer erythrocytes than does an equal volume of concentrated urine.

Sites of Origin of Hematuria

There are three main possible sites of origin of hematuria, namely lesions occurring as part of a systemic disease, lesions in organs adjacent to the genitourinary tract and lesions in the genitourinary tract itself. Among the systemic diseases that may cause erythrocytes to appear in the urine are (1) acute or chronic nephritis; (2) diseases of the blood-forming organs, such as thrombocytopenic purpura, leukemia, Hodgkin's disease, hemophilia and polycythemia vera; (3) cardiac disease, as in congestive cardiac failure or renal infarction resulting from rheumatic carditis and auricular fibrillation; (4) acute exanthematous diseases and (5) deficiency diseases, such as deficiency of ascorbic acid or vitamin K. Drugs and chemicals, such as methenamine, carbolic acid, turpentine and sulfonamides, may produce hematuria, as may administration of anticoagulants, such as dicumarol or heparin.

Lesions in organs adjacent to the urinary tract can be disposed of briefly by simply stating that the possibilities are unlimited. Among such lesions that may produce hematuria by irritation or invasion of the genitourinary tract are appendicitis, salpingitis, diverticulitis and carcinoma of the colon.

The remainder of this presentation will be devoted to a consideration of lesions of the genitourinary tract itself.

Microscopic Versus Gross Hematuria

The presence of asymptomatic microscopic hematuria is perhaps more troublesome to the physician than is gross hematuria because it is encountered more frequently and is associated with a much smaller incidence of serious disease; yet the physician does not dare neglect such a finding. Significant lesions will be found in approximately 5 per cent of such cases; thus the clinician would like the urologist to furnish a rapid and simple screening procedure that would eliminate the 95 per cent of cases in which such minimal hematuria is insignificant. To date such a procedure is not available and it is necessary to subject 100 patients to such procedures as excretory urography, cystoscopy and retrograde pyelography to discover the five or six who have significant lesions. In some of the cases in which no lesions are discovered to explain the hematuria, the condition simply may be normal erythrocyturia. In some cases hematuria may originate from trauma as a result of digital rectal or pelvic examination

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before the urine was obtained. In other cases, subclinical nephritis or a small angioma of the kidney may be present. It is apparent that asymptomatic microscopic hematuria is a worrisome problem whose solution is not always satisfactory.

Gross hematuria is a less troublesome problem, at least to the physician, simply because it is associated with such a high incidence of significant and serious lesions that a complete urologic examination is compulsory.

The commonest cause of gross hematuria in a woman is inflammation, usually cystitis or pyelonephritis. The commonest cause of gross hematuria in a man is a tumor of the bladder, with hypertrophy of the prostate in second place. An important point to emphasize is that benign enlargement of the prostate causes hematuria much more frequently than does carcinoma of the prostate.

General Clinical Aspects of Hematuria

When the physician is confronted with a patient who has gross hematuria he first considers the age of the patient. Hematuria is rare in infancy and childhood; usually it is the result of infection in such instances. Such infections usually are produced by stasis, which usually is caused by congenital obstruction. The latter is ordinarily at one of three chief points, namely the ureteropelvic juncture, the vesical neck or, in the male, the urethral meatus. Tumors of the genitourinary tract in infancy are rare and are chiefly Wilms' tumors and sarcomas. Such lesions rarely cause hematuria; they are diagnosed because they present themselves as abdominal masses.

With regard to gross hematuria in adults up to 40 years of age, the common causes include infection, cystitis, pyelonephritis, tuberculosis and calculi. Tumors are relatively uncommon in this age group. However, in patients more than 40 years of age, the incidence of neoplasms and prostatic lesions, both benign and malignant, becomes increasingly common.

The type of hematuria may give some clue as to its origin. Initial and terminal hematuria usually is considered to indicate a lesion in the neck of the bladder or the prostatic portion of the urethra. If it is present in the same degree throughout urination and if the blood is dark, the lesion is thought to be in the upper part of the urinary tract, whereas if the blood is fresh and bright, it supposedly comes from the bladder. However, it is not wise to place too much reliance on this finding. Blood that emerges directly from the urethra and soils the clothes of either a man or a woman originates distad to the external sphincter. It is well to be cautious about a story of bleeding in a woman. The physician must take a careful history and be extremely patient with a woman in order to ascertain whether she is talking about vaginal bleeding, about urethral bleeding or about cystitis; this is because of embarrassment and because many women do not understand the terms used. Sometimes a complete urologic and gynecologic examination is required to uncover the true situation.

The general physical examination may not be too productive in the diagnosis of hematuria but it should not be overlooked. Palpable renal enlargement may indicate neoplasm, hydronephrosis or a cyst of the kidney. Tenderness in the flank may indicate a stone, infection or hydronephrosis. Enlargement of a supraclavicular lymph node may herald hypernephroma. Acute left varicocele in a man past 40 years of age often means a left hypernephroma with invasion of the left renal vein, because the spermatic vein empties into the left renal vein. Digital rectal examination, which is so important, can aid in the diagnosis of carcinoma of the prostate, benign hypertrophy of the prostate or prostatic calculi. Vaginal examination may reveal infiltration at the base of the bladder from a tumor.

Excretory Urography

Emphasis should be placed on the value of excretory urography, including the plain roentgenogram of the kidney-ureter-bladder region that accompanies this procedure. This examination is of great importance in the diagnosis of urologic lesions because it is one of the greatest tests of renal function, indicating the comparative function of the two kidneys. The other factor of importance with regard to excretory urography is that it focuses attention on the part of the urinary tract that is involved, which is of great aid in cystoscopy.

Excretory urography can be of assistance in the localization of urinary obstruction, demonstrating whether such obstruction may be at the ureteropelvic junction or lower in the urinary tract. It can accurately localize calculi in the kidneys, pelves or ureters. It may demonstrate filling defects at the base of the bladder as the result of prostatic hypertrophy. Other filling defects may indicate infiltrating tumors of the bladder. Pedunculated tumors of the bladder may be outlined by the way in which the opaque medium surrounds them. Hypernephromas may be indicated by characteristic distortion of calyces. Tumors of the renal pelvis may produce typical filling defects.

Cystoscopy

It is usually necessary to perform cystoscopy to complete a urologic diagnosis because it is easier and more accurate to study lesions in the bladder under direct vision than to rely on urography entirely. Also, it is often necessary to do retrograde pyelography to supplement excretory urography. It is of importance to examine the patient urologically while bleeding is still present. So often a patient bleeds grossly for 2 or 3 days and by the time he gets ready for urologic examination the bleeding has stopped, making it difficult to determine the source of the hemorrhage.

Another element of importance in cystoscopy is to examine the ureteral orifices and watch the urine as it spurts from them. That is the only way to determine whether or not blood is coming from a kidney. Examination of a specimen of ureteral urine taken from a ureteral catheter will not necessarily indicate whether or not blood is coming from the kidney in question because the trauma caused by passage of a ureteral catheter in most cases will cause either microscopic or gross hematuria. However, the appearance of bloody urine flowing out from a ureter that has not been catheterized will localize the kidney that is bleeding.

In conclusion I wish to emphasize that the Bladder Tumor Registry indicates that 50 per cent of tumors of the bladder are not diagnosed within one year after the first attack of hematuria. This is a tragic situation; it means that all physicians must be on the alert in their practice and community to educate patients and the public to the seriousness of hematuria and the advisability of seeking medical advice immediately when it occurs.

CORTISONE IN RHEUMATIC DISEASES

(Continued from Page 308)

such treatment is indicated. Until better therapeutic measures are developed, cortisone and related hormones will continue to be important and valuable agents in the management of such patients.

Addendum

Since this paper was prepared, another potent antirheumatic steroid, namely metacortandracin, or 1-dehydrocortisone, has been synthesized. Although this steroid has less tendency than does cortisone to cause retention of sodium, chloride and water, and to produce excretion of potassium, it does cause many other cortisonelike metabolic effects. The dose of 1-dehydrocortisone (metacortandracin) necessary to produce an equivalent antirheumatic effect appears to be only a third or a fourth that of cortisone, perhaps even less in some cases. The use of this steroid should be attended by the precautions indicated during treatment with cortisone; however, restriction of intake of sodium and supplementation of potassium usually have not been required, at least not in rheumatoid patients treated for periods up to several months with doses of 5 to 20 mg. per day.

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The Technique and Interpretation of the Vaginal Examination

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MOST of us learn best by doing, and so it is fortunate indeed that the technique of the pelvic examination can be attained more by doing than by reading or hearing about it. What I have to say, therefore, is directed chiefly toward actual performance of this examination, with some attention to interpretation of what the examiner finds.

The conditions to which the physician's attention is called most often by women include inflammatory and infectious disease, new growths, sequelae of labor and endocrine dysfunctions which produce aberrations of menstrual function. Often the patient will seek your counsel because of abnormal vaginal secretions, genital bleeding or pelvic pain. Less often, she may come to you because of protruding masses or generalized pelvic or abdominal discomfort. Still others may visit your office because the persistent "cancer drives" have made them apprehensive, and they wish to reassure themselves by such routine measures of investigation as you are able to provide. Whatever the cause, it is certainly true that the number of such patients in the office of the gynecologist is increasing. The net result is to place greater responsibility on the physician, for if mass education sends more patients to him, he will be expected more and more often to detect malignant processes in earlier stages, where treatment can be swift and effective, and lives can be saved.

The Physical Examination

A general physical examination should follow the history and should, whenever it is possible, precede the pelvic examination.

What to Look For.—In the general examination of the patient much can be learned: her habitus, whether masculine or feminine, robust or frail, hirsute or balding—all may be noted with a glance. The temperature, pulse rate and blood pressure should be recorded. The breasts should be examined, for they are secondary sex characters and as such they share in many changes and physiologic conditions within the pelvis. In the abdominal examination the physician should note the presence or absence of striae indicative of rapid loss of weight, and evidence of past pregnancies or endocrine dysfunctions. Tender areas should be carefully palpated, and distinction should be made between rigidity and normal muscular defense reaction.

Much can be learned from an adequate abdominal examination, and although it is neglected by many, it may actually bring to the fore the primary difficulty at hand, especially if the patient is acutely ill or apprehensive. In passing, let us remember that a full bladder at times has deceived the shrewdest of examiners. For this reason, some gynecologists have suggested that the patient void immediately before examination to forestall such a diagnostic pitfall.

The Pelvic Examination

Whatever is learned after the history and the physical examination must be gained tactually, and must be correlated with information gained from those two procedures. The only way I know of to develop the tactile sense is to do enough pelvic examinations to acquire the faculty of instant recognition not only of the normal anatomic relationships but also the minor aberrations which are the hallmarks of pelvic disease. One should become familiar with the nodular tender areas involving the uterosacral ligaments and posterior uterine surface so characteristic of endometriosis; also the thickened, tender and bulbous swelling of the tubes portraying the aftermath of pelvic inflammatory disease.

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Equipment.—All the necessary equipment for the proper performance of a pelvic examination should be at hand before the examination is begun. This would include drapes, hand protection, lubricants, light, material for taking smears, and a table which will offer the examiner every advantage.

Since the speculum is an indispensable instrument to the gynecologist, a word should be said regarding the various types available. For most purposes, the bivalve speculum is perfectly satisfactory. It is made in several sizes, and the examiner selects that size which can be introduced easily and does not cause discomfort to the patient. In children, the most satisfactory speculum is the tubular cystoscope, which is used with the patient in the knee-chest position. The tubular speculum likewise is available in a variety of sizes. For some patients the flat Sims speculum may be used to advantage. The physician should become familiar with the various speculums and their sizes to facilitate and insure maximal comfort to the patient.

Rapprochement With the Patient.—Establishing the patient's confidence is the greatest single factor in promoting ease of examination. Unconsidered remarks or chance actions which engender fear, resentment or anxiety may result in a tense, disturbed or apprehensive patient. Such a patient is rigid and ill at ease, and in a state that may make a pelvic examination impossible or seriously unproductive. In creating confidence, gentleness is the first essential. Relaxation may be encouraged by asking the patient to breathe through her mouth. Constant reassurance is helpful. No violation of modesty should enter the pelvic examination, but exposure should be consistent with thoroughness. The presence of a nurse or an assistant may aid in this respect, although it is not entirely necessary. Most of all, the physician should maintain an attitude of kindly and impersonal thoroughness. A pelvic examination is not a pleasant experience for any woman, and the success with which it is conducted will depend as much on the attitude of the physician and his assistant as upon the actual situation in the pelvic region.

What to Look For.—Inspection of the external genitalia is done with the patient in the lithotomy position, with the physician standing between the patient's knees. The vulva is inspected for dermal lesions, excessive secretions and tumor masses. Since vulvar neoplasms frequently metastasize to the inguinal glands, these glands should be palpated for tenderness or enlargement. Small, shotty inguinal glands are not unusual, especially in young women, and should cause no concern unless they are associated with definite lesions.

After examination of the vulva, the labia should be gently parted, and the size, shape and dermal changes, if present, should be noted carefully. Inspection for kraurosis vulvae, lichen sclerosus et atrophicus and leukoplakia should be made. If it is suspected that one such condition is present, the counsel of a dermatologist is of inestimable help in identification. If a discharge is present, the examiner should determine whether it is bloody, serosanguineous, purulent or mucoid. Normally, Bartholin's glands should not be palpable and Skene's glands should not be tender. If the hymen is intact, examination of the pelvic organs may be completed rectoabdominally. Careful note should be made of the caliber of introitus. By pressure exerted downward against the perineal body, more space may be obtained with less discomfort to the patient.

The condition of the pelvic floor is then determined. To determine the presence or absence of rectocele is not difficult, but may be rendered easier by pressure exerted upward on the posterior vaginal wall, through the rectum. The size, shape, consistency and position of the cervix should then be determined by palpation. A normal cervix is said to have the consistency of the end of the nose, whereas a cervix invaded by a malignant process generally has a hard or gritty consistency.

At this point, examination with the speculum is begun. It is well to recall at this point that the axis of the vagina is directed posteriorly, while the long axis of the introitus is anteroposterior. It is well, then, to introduce the speculum of the bivalve type with its long axis vertical, to conform to the shape of the vaginal orifice. This is preceded by separating the vulva and pressure on the perineal body. When it is well past the entrance of the vagina, the speculum is turned so that the blades lie transversely, with the tip of the speculum pointed posteriorly toward the vaginal floor when the blades are opened. The common practice of using soap or lubricants is not advisable, since soap alters the chemical reaction of the vaginal secretions and interferes with staining and cultural reactions. Lubricants also frequently make interpretation of Papanicolaou smears for malignant cells more difficult or even impossible. Rather, it is better to wet the gloved hand and speculum with warm water, thereby decreasing the shock to the patient and offering adequate lubrication.

With the aid of a strong light, the cervix is now visualized directly. Size, position and length of the cervix, as well as the nature of its secretions, are noted. This is the moment at which an old adage becomes most significant: "Examine the cervix with a strong light and with a suspicious mind." A smear for study by the Papanicolaou technique may be taken; secretion should be taken from both the internal os and the vaginal pool. Samples may be taken with the use of either a wooden spatula or a cotton applicator. The secretion is spread on a clean glass slide which is dropped immediately into a solution of 95 per cent alcohol. Because of the danger of explosion, ether should not be added to the solution of alcohol stored about the offices.

Next, the cervix is inspected for evidence of cystic change, lacerations or erosions. A specimen of tissue can be taken for biopsy, if biopsy is indicated. A specimen of any abnormality that is seen should be taken for biopsy before definitive therapy is offered. Such a specimen should be obtained in the presence of all cervical erosions, and care should be exercised to obtain adequate tissue from the squamocolumnar junction. This should always be done before cervical cautery is attempted. The application of Lugol's solution will demarcate those areas most applicable for biopsy.

You will recall that normal cervical and vaginal epithelium contains glycogen, whereas abnormal epithelium, such as that found in erosions or a malignant lesion, contains little or none at all. Hence, by applying a weak solution of iodine (¼-strength tincture of iodine) to these areas, a marked differentiation may be seen rapidly; normal tissue taking a deep mahogany brown, the pathologic a pink. It is from the pink or light areas that specimens for biopsy should be taken. One must be cognizant of the fact that the Schiller or iodine test is not specific for any type of lesion, nor does it distinguish malignant from benign tissue. All it does is to demarcate the areas from which specimens of tissue for biopsy should be taken. There is no special time in the menstrual cycle when the specimen for biopsy should be taken; the important factor is to do it when the patient is seen. In this regard, the endocervix should not be neglected, because the introduction of a small sound or cotton applicator within the cervical canal (the so-called Clark test) will many times disclose a pathologic process which otherwise might have been missed.

Many women present themselves with bleeding after subtotal hysterectomy. Under such circumstances, a small endocervical curet may be used to obtain tissue for examination. If small endocervical polyps are the cause of the bleeding, this curettage may be therapeutic as well as diagnostic. It is always well to submit all such material to a competent pathologist for careful examination and evaluation.

It is also a wise practice to remove all polyps which may be found extruding from the cervix. Polyps can be removed easily by torsion; this should be followed by fulguration of the bases of the polyps. All polyps should be examined by a competent pathologist. Before the speculum is removed, the condition of the pelvic walls should be observed, with attention to the presence or absence of excoriations or new growths.

The bimanual examination, which would better be known as the "vaginal-abdominal examination," can be done with either the left hand or the right within the vagina. From a practical standpoint, and especially in the case of those physicians who practice obstetrics, I believe it is useful to develop ambidexterity in the performance of this examination. With the examiner's fingers resting against the pelvic floor, the cervix is palpated, while the examiner's other hand is placed flat on the lower part of the patient's abdomen. By elevating the palm and using the tactile sense in the balls of the fingers rather than in the tips, the various organs are located, steadied and evaluated. The size, shape and consistency of each structure can be determined, and if tumors project into the superior strait, their outlines can be noted.

After the cervix has been palpated, the presence or absence of pelvic pain on motion is determined. The position of the uterus is ascertained by locating the body of the uterus. When the uterine fundus lies in its normal relationship, it is usually in an anteflexed position. Retrocession or retroflexion occurs normally in a high percentage of women. The mobility of the uterus may be then thoroughly tested. Immobility or excessive pain on uterine motion may be indicative of chronic infection, acute exacerbation of chronic infection, adhesions or endometriosis. When the median part of the pelvis has been palpated and the condition of the uterus has been determined, the examining fingers are now slid into one of the fornices lateral to the uterus. The abdominal hand is directed in a like plane, and is moved slowly and deliberately.

Next, the examiner's fingers in the vagina are pushed out into the lateral fornix, while the hand resting on the abdomen is directed in a like plane. The ovary is then palpated between the tips of both fingers. A normal ovary is sensitive and mobile. Ovaries which are retrocessed within the pelvis are best examined later by rectoabdominal approach. The physician should become familiar with the normal size of an ovary and should keep in mind its tendency to enlarge after contralateral oöphorectomy and hysterectomy. The normal ovary feels like an almond; it is about 4 cm. long and 2 to 3 cm. wide. Normally, it will move within a limited range. Occasionally, its mobility may become abnormal and it may be situated immediately lateral to the cervix, within the cul-de-sac, or high on the lateral pelvic wall.

Normal fallopian tubes usually cannot be palpated through the vagina; however, if they are thickened or are the sites of chronic residual changes from infection, they may be sensed as masses of hornlike shape which occasionally are fluctuant, and many times are tender, firm and resistant.

Occasionally, it is possible to palpate the ureters, lying as they do on the lateral pelvic wall. If pyelonephritis is present, the ureters may be thickened; if tuberculosis is present, they may be nodular. Ureteral calculi occasionally can be palpated; inflammatory conditions within the ureters may provide their own clues by making the ureters unusually tender.

Rectal examination should be done for all patients who complain of difficulties referable to the pelvis, and it is especially indicated for young women with an intact hymen. When a pelvic malignant process is present, the rectoabdominal examination gives perhaps more information than any other. The necessity for an empty bowel is clear. Care should be taken not to exert too much pressure against the anterior wall of the bowel, for that structure may be extremely tender. Should any abnormalities be noted, proctoscopic examination is indicated.

Lesions within the vagina and cervix occasionally may be seen best by examination with the patient in the knee-chest position. The vagina is easily distended with air, making the vaginal rugae disappear, and allowing the walls of the vagina to be seen clearly. Examination of children and young girls is best done with the patients in this position, and with the aid of a Kelly cystoscope.

At times, it may be necessary to carry out pelvic examination with the patient anesthetized. This is indicated only after repeated pelvic examinations have been entirely unsuccessful, sometimes with several days intervening. We should be aware not only of the usefulness of this procedure, but also of its limitations. Naturally, examination of the pelvis with the patient under anesthesia has no value under conditions in which the cooperation of the patient is needed; for example, to locate areas of pain or minimal discomfort. In general, the more complete and accurate the pelvic examination is, the less frequently need one resort to anesthesia in gynecologic diagnosis.

Laboratory Aids.—A paper on the pelvic examination of course is not complete without brief mention of some of the laboratory facilities which aid in the diagnosis. These should include urinalysis, complete blood counts, determination of the sedimentation rate, serologic tests for syphilis, smears and culture of specimens of urethral and cervical secretions, and finally, the taking of special smears for the Papanicolaou technique of detection of malignant cells. Specimens of cervical tissue should be taken for biopsy if the examiner believes this laboratory procedure is indicated.

It is well to emphasize again that all laboratory procedures should be supplemental to clinical observation and carefully performed bimanual examination. Preceded by a carefully taken history, the value of any laboratory test is dependent upon the clinician's ability to interpret the findings and correlate them with what he has found by direct examination of the patient.

The Treatment of Anemia

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IN ANY consideration of the treatment of anemia, it is important to recognize that anemia is not a disease in itself but merely a symptom resulting from some underlying cause. Therefore, one must establish the etiologic factor involved in the anemia prior to therapy.

Hypochromic (Iron-Deficiency) Anemia

The great majority of anemias are of the iron-deficiency type and usually are secondary to chronic loss of blood, to inadequate intake or inadequate absorption of iron, or to the so-called physiologic deficiency of iron. Morphologic examination of the erythrocytes reveals a decrease in hemoglobin within the cell, with resultant hypochromasia. The erythrocytes are usually normal in size, although some regenerative macrocytes may be present; in some instances the erythrocytes may be smaller than normal. Regenerative macrocytosis with increased polychromatophilia may be noted, particularly if the loss of blood has been severe. A proportionate reduction in the number of erythrocytes usually occurs in acute hemorrhage; however, in this case the cells rarely show a loss of hemoglobin unless an associated hypochromic anemia of some duration has been present prior to the onset of the acute hemorrhage.

It is important to ascertain, insofar as possible, whether any loss of blood has occurred in a person who has hypochromic anemia. Loss of blood with menses frequently may produce a rather pronounced hypochromic anemia. Bleeding from the gastrointestinal tract is relatively common. Such bleeding may originate from ulcers, carcinoma, polyps, varices or hemorrhoids; various hemorrhagic diseases may be manifested initially by bleeding from the gastrointestinal tract. In a patient who has an obscure anemia, one should examine the stools for evidence of occult blood and a thorough gastrointestinal investigation should be done.

Under modern conditions, anemia associated with an inadequate intake of iron is relatively rare. A well-balanced diet in the northern part of the United States contains approximately 10 mg. of iron daily. The gastrointestinal tract is able to absorb approximately 0.5 to 1.5 mg. of iron daily; this absorption occurs at a constant rate even when there may be unusual demands for iron by the body. The normal excretion of iron is approximately 1 mg. daily. In certain instances it is well to obtain a detailed description of the patient's diet if other etiologic factors cannot be elicited in the investigation of hypochromic anemia.

Anemia due to physiologic deficiency of iron may occur in several conditions. In infancy there is usually an inadequate intake of iron as well as a period of rapid growth with a rapid increase in blood volume; consequently, the relative deficiency of iron results in hypochromic anemia. This same condition may prevail during periods of rapid growth in later years, particularly at the time of puberty. At such times the great increase in blood volume may be accompanied by a relative decrease in the amount of iron available, with development of hypochromic anemia. At the onset of menstrual flow, blood may be lost at a time when rapid growth is still occurring, producing a double demand for iron; in such instances, rather severe degrees of hypochromic anemia may appear. The so-called physiologic anemia of pregnancy is due in part to the fact that within a relatively short period a great increase in blood volume occurs with a relatively constant intake of iron; thus, iron-deficiency anemia may occur.

Practically all persons can absorb iron from the gastrointestinal tract. The iron is absorbed in the form of ferrous salts and the chief site of absorption is the duodenum. As already indicated, an extremely small amount of the iron in-

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From the Section of Medicine, Mayo Clinic and Mayo Foundation.

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gested is actually absorbed and, after hematologic values have been restored to normal by administration of iron, it is well to continue the treatment for a month or so in an attempt to supply the body with sufficient stores of iron. Protracted treatment after that time is usually not advisable.

When an anemic patient takes iron, the peak of the response of reticulated erythrocytes occurs in about eight to twelve days, which is about the same time at which it occurs in the treatment of pernicious anemia with effective fractions of liver or vitamin B_{12}. Since administration of iron is the treatment of choice in iron-deficiency anemia, the use of liver extract, vitamin B_{12}, folic acid and other similar substances is not indicated.

Practically all patients respond to the oral use of iron. However, an occasional patient cannot tolerate iron given by mouth. Also, in certain severe hypochromic anemias it may be desirable to obtain a rapid increase in the value of hemoglobin. In such situations, the intravenous injection of iron is of value. Several formulas for the calculation of the amount of iron needed intravenously to bring the hemoglobin to a normal level can be found in the literature. The simplest one is based on the fact that approximately 25 mg. of metallic iron is required to increase the value for hemoglobin by 1 per cent. This provides an easy method of calculating the amount necessary. The preparation used is a solution of saccharated iron oxide in a dose of 100 mg. injected intravenously once or twice daily. Care should be taken to determine that the material is not unduly toxic for a given patient and a small initial dose should be used. If an untoward reaction does not occur, the patient usually will tolerate larger doses. Actually it is rarely necessary to resort to intravenous therapy, as practically all patients respond satisfactorily to iron given orally.

Macrocytic Anemia

Among the various types of macrocytic anemia, often termed "megaloblastic anemia," the commonest is pernicious anemia. Fortunately this form of anemia responds well to adequate therapy. The clinical findings in pernicious anemia may vary but in general they include anemia, glossitis with atrophy of the mucous membrane of the tongue, a history of pernicious anemia in the family, early gray hair, evidence of combined system disease, absence of free hydrochloric acid in the gastric contents, and intestinal manifestations, which may be characterized by loose watery stools or by constipation alternating with loose stools. As a rule, patients who have pernicious anemia do not lose weight and may have a relatively abundant amount of subcutaneous fat. They often display a surprising amount of energy for the degree of anemia that is present; this is probably due to the fact that, because of the gradual onset of anemia, the body becomes accustomed to the anoxemia.

Examination of blood smears in pernicious anemia reveals generalized macrocytosis, with the erythrocytes usually containing normal amounts of hemoglobin. There is usually an increase in poikilocytosis or the appearance of pear-shaped erythrocytes, which is thought to be due to loss of elasticity of the cellular membrane. The leukocyte count is usually normal but may be decreased or even increased. Megaloblasts or normoblasts may be present in severe degrees of pernicious anemia and thrombocytopenia occasionally may be noted. More rarely, a leukemoid reaction may occur in severe degrees of pernicious anemia. An occasional patient who has pernicious anemia in crisis may have a leukocyte count as high as 40,000 or more cells per cubic millimeter of blood, with a great increase in immaturity, so much so that the presence of acute leukemia may be suspected in an occasional case. Examination of the bone marrow reveals the presence of megaloblasts, which, together with the clinical findings, usually enables one to establish the diagnosis of pernicious anemia. The polymorphonuclear neutrophils usually exhibit thinning of the individual lobes, with stranding between the lobes. Hyperlobulation may occur, with as many as six to twelve lobes, or even more, being present. This type of neutrophil, although not absolutely diagnostic, is the so-called pernicious-anemia neutrophil and its appearance, with other findings in the peripheral blood, would make one suspicious of the presence of this disease.

After the diagnosis of pernicious anemia is established, the treatment is relatively simple. The most important factor is to give effective doses of liver extract or vitamin B_{12}. Depending somewhat on the degree of anemia, I prefer to start treatment with fairly large doses. If concentrated liver extract is used, an initial dose of 3 cc. (45 units) is given; if vitamin B_{12} is used, the initial dose is 60 micrograms. This dose is given intramuscularly once daily for three successive days. Then 1 cc. of liver (15 units) or a dose of 30 micrograms of vitamin B_{12} given once a week usually restores the blood to a normal level, after which time a maintenance dose may be worked out. In the uncomplicated case, this dose averages about 15 units of liver extract or 30 micrograms of vitamin B_{12} every two to four weeks. Some persons require more than others. In the presence of combined system disease, a larger dose of an effective fraction may be necessary, such as 30 units of liver extract or 60 micrograms of vitamin B_{12} twice a week, which may be continued for a number of months depending on the degree of improvement in the combined system disease.

Proper maintenance therapy consists in giving a sufficient amount of an effective fraction to keep hematologic values at normal levels, to avoid macrocytosis and to prevent any advancement of neurologic symptoms or to improve them, if it is at all possible. Sometimes a patient may be receiving maintenance doses of liver extract and getting along satisfactorily; in spite of this, the symptoms and findings of combined sclerosis will develop. In such instances, larger doses of the effective agent are required.

There is practically no difference between the response obtained from vitamin B_{12} and that obtained from liver extract. One slight difference is that liver extract exerts a slight effect on the coagulation of blood that is not produced by vitamin B_{12}. This is relatively unimportant and is of no consequence with regard to the management of pernicious anemia. The main factors are to give sufficient material for control of the neurologic symptoms and to keep the patient on sustained and steady treatment for the rest of his life.

Parenteral administration of liver extract or vitamin B_{12} is in general the treatment of choice. As a rule this method is cheaper, it permits the physician to keep the patient under observation so that he can determine whether or not the treatment is adequate, it impresses on the patient the importance of regular therapy and it assures that the material is absorbed. Vitamin B_{12} has what might be considered some advantages over liver extract in that it is a purified crystalline substance of constant potency, it may be used in patients who are sensitive to liver extract and it produces a minimal local reaction. Oral therapy for pernicious anemia produces a satisfactory remission and patients may be maintained satisfactorily by such treatment. However, at present it is usually expensive, absorption may vary greatly depending on the condition of the gastrointestinal tract at a given time and in general it is difficult to maintain adequate control of the patient by such therapy.

Folic acid is known to be important in the maturation of erythrocytes. It will convert a megaloblastic bone marrow to a normoblastic state and will correct the hematologic abnormalities associated with pernicious anemia. However, glossitis and the symptoms of combined sclerosis may develop in many patients while folic acid is being given. Consequently its use is not advisable although it is not contraindicated. Essentially the same is true for citrovorum factor, or so-called folinic acid, another substance that has been shown to be necessary for the conversion from a megaloblast to a normoblast. Folic acid and citrovorum factor are effective either orally or parenterally but neither of them exerts any beneficial effect on combined system disease. They usually have no effect on the gastrointestinal symptoms in pernicious anemia. Citrovorum factor, however, is important in that it is of great value in combating the toxic reactions to antifolic substances that have been used in the treatment of certain leukemias. Combination therapy with liver extract and iron is rarely needed because in most instances the stores of iron in pernicious anemia are at a high level. Administration of hydrochloric acid as a rule is not necessary in the management of pernicious anemia.

The treatment of other types of megaloblastic anemia is often extremely difficult. It usually may be summed up as a method of trial and error, utilizing various substances that are essential for hematopoiesis. Megaloblastic anemia other than pernicious anemia may occur as the result of gastrointestinal disease, total gastrectomy, intestinal strictures and fistulas, or short-circuiting operations; nutritional macrocytic anemia is seen in the tropics and with sprue, either tropical or nontropical. Megaloblastic anemia also may be associated with pregnancy; this is the so-called pernicious anemia of pregnancy, which is usually due to a deficiency of folic acid. Use of liver extract or vitamin B_{12} may produce improvement; otherwise, folic acid may be of value. The megaloblastic anemia of infancy usually can be corrected by use of folic acid, although in some instances liver extract or vitamin B_{12} may prove effective. The megaloblastic anemia associated with infestation by the fish tapeworm shows a variable response to the administration of vitamin B_{12} or folic acid.

The treatment of megaloblastic anemia other than pernicious anemia is probably best begun by use of vitamin B_{12} in the same dosage as would be used in treatment of pernicious anemia. If no response occurs after two or three weeks, use of folic acid may be tried. If there is still no response, folinic acid or citrovorum factor can be used. Transfusion may be necessary in some cases because of lack of response to any of these substances. Occasionally, a rare macrocytic anemia may occur with a relatively aplastic bone marrow that will respond to administration of cortisone, following which the blood responds to use of folic acid or vitamin B_{12}. In refractory anemias, such treatment might be tried.

Anemia in Systemic Diseases

Anemia is a frequent finding in many systemic conditions for which treatment is of practically no value. Examples of such anemias are those associated with metastatic malignant lesions involving the bone marrow, nephritis with azotemia and the anemia associated with infection. In azotemia, if it is possible to decrease the blood urea, the patient's blood usually will respond automatically. In true hypoplastic or aplastic anemia, treatment is of little or no value. The same is true of the leukemias and lymphoblastomas. The anemia associated with rheumatoid arthritis rarely responds to any of the usual hematinics. The anemia associated with myxedema responds to administration of desiccated thyroid but usually fails to respond to any other substance.

In conclusion, it is important to establish the diagnosis and the etiologic factor responsible for the anemia before treatment is started. In certain instances, treatment of anemia may mask an underlying serious disease whose early diagnosis would save the patient's life.

DIGESTIVE AILMENTS OF OLDER PATIENTS

(Continued from Page 314)

the background of the patient, his habits and his way of life, and who can, by persuasion and advice, do much to ameliorate these symptoms.

Summary

An attempt has been made to show the frequency, distribution and severity of benign digestive disorders in the aged. In many instances, the complaints, however bizarre and atypical, are likely to have an organic background. The diagnosis of the organic disease is often far from easy, chiefly because the usual clues which lead to diagnosis are absent or distorted. If the history is brief, full investigation is compulsory. If it is a continuation of one which has plagued the patient all his life, investigation is still essential, if only to provide the necessary reassurance and support essential to treatment. Symptomatic treatment without full investigation is at its best a risky business and temporizing on such a basis is the cause of much disability and many fatalities in the aged. It should not be forgotten that persons who reach the eighth decade of life are of a rather special pattern and mold, and not infrequently are made of better biologic materials than their juniors. Their tolerance to surgery may be amazingly good and their recovery from even major procedures rapid. Here is a field in which all physicians can participate in reducing mortality and morbidity rates, and in making life more bearable for many people who have passed their prime.

Laboratory Aids

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George G. Stilwell, Editor

CARCINOMA IN SITU OF THE UTERINE CERVIX

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Carcinoma *in situ* is a lesion in which epithelium in its normal anatomic location possesses the cytologic criteria for malignancy. It has not broken through the anatomic barrier that separates the epithelium from the underlying connective tissue. Such preinvasive or intraepithelial carcinoma has been observed in many sites, including the skin, the mammary ducts and acini, colon, stomach, bronchi, larynx and uterine cervix.

Intraepithelial carcinoma of the cervix has been recognized for more than thirty years. However, large series of cases have accumulated only during the past decade, as a result of development of intensive programs for the early detection of cancer. Investigators have established some facts concerning this phase of cervical carcinoma and have emphasized some other problems relating to it that as yet are unsettled.

Knowledge of the established facts is necessary to an understanding of the controversies. Carcinoma *in situ* is ordinarily invisible. Most of these early malignant lesions are discovered when a smear or a specimen for biopsy is procured from a grossly normal cervix or one that has an erosion, an eversion, leukoplakia or "cervicitis." Application of Lugol's solution may outline the neoplastic lesion when it is otherwise invisible; normal cervical mucosa stains dark brown owing to a high glycogen content of the cells, whereas little or no staining occurs in the malignant cells, which are deficient in glycogen.

Carcinoma *in situ* nearly always begins at the squamocolumnar junctional zone. This line is usually at the external cervical os but may lie well out on the portio vaginalis or up in the cervical canal. Those *in situ* carcinomas associated with leukoplakia may arise any place on the cervix and even in the vagina. Downgrowth of this surface carcinoma into the mucous glands of the cervix is common and may be extensive. Such spread does not impart a metastasizing potential in the absence of true stromal invasion, because the basement membrane of replaced glands acts as a barrier to lymphatic and hematogenous dissemination.

In situ carcinoma may be as small as a square millimeter and involve only a minor portion of the circumference of the squamocolumnar junctional zone. However, it may spread to involve large regions of the portio vaginalis and even, though rarely, the upper part of the vagina. This surface lesion also may extend 2 or 3 cm. up to the endocervical canal.

The accessibility of the uterine cervix to inspection and for procurement of specimens adapts it well to cytologic techniques for early detection of cancer. *In situ* or early infiltrating cervical malignant tumors that are invisible can be unmasked with a high degree of accuracy by study of stained smears of scrapings from the exocervix and endocervix. Tissue for biopsy that includes the squamocolumnar junctional zone taken from each quadrant of the cervix provides a similarly great accuracy of detection.

Cervical carcinoma *in situ* appears to be present in about 1 per cent of adult females. Practically equal numbers of these lesions can be detected by good cytologic screening, by careful biopsy techniques or by a combination of the two. Sizable variations from this usual rate of prevalence indicate differences in the criteria for carcinoma accepted by some pathologists or in the efficiency of the detection program.

Debates concerning some of the controversial phases of this subject can hardly add real knowl-

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From the Section of Surgical Pathology, Mayo Clinic. This is the twenty-seventh in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

edge, although they do bring known facts into focus. Scientific statistical evaluations of the type contributed by Dunn[1] likely will supply the ultimate answers. Unwillingness to submit human beings to periods of observation of intraepithelial carcinoma hampers acquisition of knowledge. Evaluation of recorded studies is clouded by the fact that biopsy sometimes eliminates the entire lesion, as can local cautery.

Does uterine carcinoma *in situ* usually progress to infiltrating carcinoma and is the latter ordinarily preceded by the former? More than forty instances of such progression have been documented, and a lesser number of these preinvasive carcinomas reputedly have regressed.[2] Limited data suggest that a preinvasive phase usually precedes clinical carcinoma of the cervix.

How long is the latent period, the time from which intraepithelial carcinoma is histologically recognizable until it produces clinical carcinoma? The average difference in age of patients at the time of detection of each form of lesion indicates an interval of about eight years. Since detection of the asymptomatic and invisible carcinoma *in situ* is a matter of chance, the average lesion in a screened population is found at about the midpoint in its evolution from histologic recognizability to clinical invasiveness. Accordingly, it is likely that the preinvasive phase lasts about twice as long as the ages at detection would indicate, or about fifteen years.

The likelihood that preinvasive carcinoma incubates for more than a decade lends credence to its significance. Statistically, the average prevalence of the *in situ* lesion and the recorded incidence of development of clinical cervical malignancy are consistent with the view that the former is the precursor of the latter. Additional validity for the concept of carcinoma *in situ* is afforded by other facts. Jewish women enjoy the same relative immunity for this incipient lesion that they are known to have for clinical carcinoma of the cervix. Recognizable malignant cells appear in smears in both stages of the disease. Typical intraepithelial carcinoma is frequently found at the periphery of ulcerating cervical carcinoma. The full-blown picture of carcinoma in situ probably is valid diagnostically during pregnancy, although some investigators caution against overdiagnosis of epithelial hyperplasia in the pregnant state.

Controversial criteria have been suggested for establishing that a given carcinoma is still *in situ.* The experience of my associates and me[3,4] indicates that study of a section made from each of about twelve serial blocks of the entire squamocolumnar region at right angles to the junctional zone is adequate. Minimal evidence for true invasion includes obvious vascular invasion or stromal permeation, the latter often accompanied by a scirrhous reaction to the penetrating cells.

What treatment should be given for carcinoma *in situ?* Hysterectomy alone is curative except for the rare case in which surface spread past the line of excision in the upper part of the vagina has occurred. Some physicians consider that conization of the junctional region or extensive cautery is adequate. Extensive surface spread to the exocervix or vagina or up the cervical canal imposes obvious limitations on either of these procedures. The more radical procedures, including Wertheim hysterectomy with dissection of the pelvic lymph nodes and conventional radiation therapy, are unnecessary.

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Case Reports

HEMOBILIA FOLLOWING BLUNT TRAUMA TO THE LIVER

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Hemobilia is a term used to designate hemorrhage into the biliary tract as a result of trauma to the liver. Fourteen cases are reported in the collected literature which has been reviewed by Sandblom,[3] by Sparkman,[4] and by Sparkman and Fogelman.[5] We wish to report two additional cases.

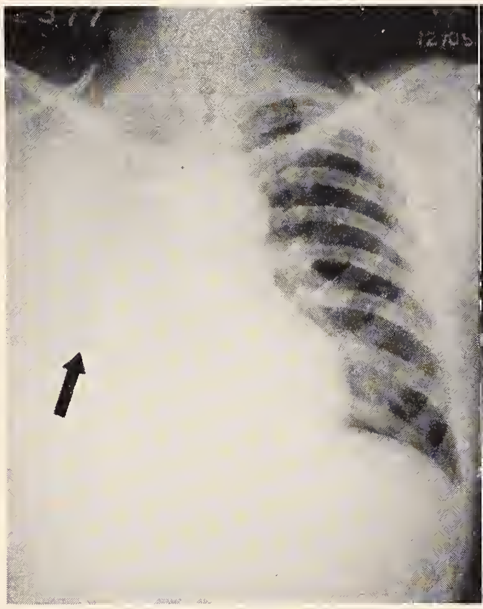

Fig 1. Case 1. Postoperative chest roentgenogram taken on December 10, 1953, showing a right pleural effusion with an air fluid collection under the right diaphragm.

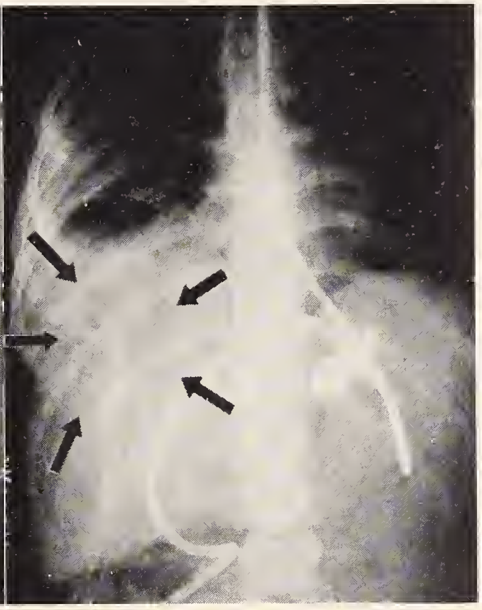

Fig. 2. Case 1. Postoperative roentgenogram of the abdomen after choledochostomy. This view shows the developing area of emphysematous tissue under the right diaphragm and a large air bubble present in that region.

Case Reports

Case 1.—D. U., a twenty-five-year-old white man was admitted to the Minneapolis Veterans Hospital on November 22, 1953, sixteen hours after having been in an automobile accident. The patient suffered no apparent injury at the time of the accident and was taken home by a passing motorist. Shortly after arriving home, he developed abdominal pain and a physician was called. When examined by the doctor the patient was in shock. A transfusion of 1000 cc. of whole blood was given at the local hospital and he was transferred to this hospital for definitive treatment.

Physical Examination.—On admission, the blood pressure was 90/60 and the pulse was 120 beats per minute. Examination of the head, neck and chest was normal. The abdomen was moderately distended and generalized rebound tenderness and spasm were present. There were no abrasions of the thorax or abdomen.

Laboratory Examination.—The hemoglobin was 12 Gm./100 cc. blood. The urine was normal. The flat and upright roentgenograms of the abdomen were not diagnostic.

Hospital Course.—Shortly after admission, the patient vomited 2000 cc. of grossly bloodly fluid. A transfusion of 1500 cc. of whole blood was given rapidly, after which the hemoglobin was reported as 11.7 grams. The blood pressure rose from 90/60 to 130/80 but the pulse remained at 130.

The patient was operated upon. Approximately 3000 cc. of blood was present in the peritoneal cavity. The capsule was torn free from an area of the dome of the right lobe of the liver. The laceration was estimated as 8x12 cm. in dimension. Liver tissue and blood clots were scooped out to a depth of 3 or 4 cm. The wound was

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packed with gelfoam. Two large rubber tube drains to the area were brought out in the right flank posteriorly under the margin of the twelfth rib. Postoperatively, the patient's temperature remained at 100-101 degrees F. There was a large amount of bile drainage (Fig. 1). It was suggested that, since most of his bile was draining through the posterior drain, a decompression of the biliary tract be performed. On December 18, 1953, a choledochostomy was performed. This was followed by a marked decrease in the bile drainage from the wound.

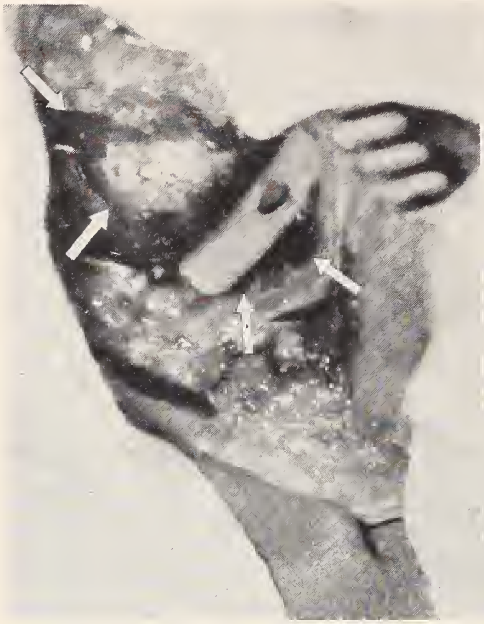

Fig. 3. Case 1. The large cavity is seen on roentgenogram in Figure 2 as an emphysematous area. The cavity is filled with necrotic liver tissue which is shown in Figure 4.

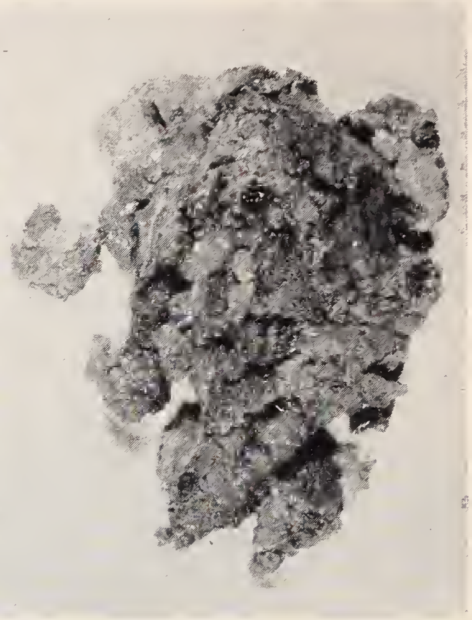

Fig. 4. Case 1. The resected specimen of necrotic liver tissue from the right lobe. This tissue is seen on the roentgenogram in Figure 2, represented by the area of emphysematous reaction.

On December 24, 1953, a peculiar emphysematous mass with a fluid level was noted under the right diaphragm on the roentgenogram (Fig. 2). This mass was explored through an incision traversing the tenth rib bed and diaphragm, and a sequestrum of necrotic liver tissue, 10 cm. in diameter was removed. The sequestrum was separated from the viable liver tissue by a fibrotic layer. It was removed by finger dissection. Brisk bleeding was controlled by a gelfoam pack. It was estimated at the time of surgery that about 30 per cent of the right lobe of the liver had sequestrated (Figs. 3 and 4).

Postoperatively, the patient developed a stenosing esophagitis involving all the esophagus beyond the aortic arch. This was satisfactorily controlled by dilatation. Healing of the liver defect was slow but progressive and was complete after four months.

This patient has been followed for one year. Liver function tests were within normal limits on December 13, 1954. The patient is completely asymptomatic and is working.

Case 2.—P. H., a twenty-year-old white woman was admitted to the Minneapolis General Hospital on June 19, 1954, following an automobile accident. On admission, she was irrational and uncooperative.

Physical Examination.—Blood pressure was 94/54, the pulse was 100 and the temperature was 98 degrees F. Findings included a large hematoma of the forehead, tenderness over the right lower chest, and abdominal rigidity with marked tenderness present in the right upper quadrant. Bowel sounds were absent.

Laboratory findings were essentially normal with the exception of a hemoglobin of 10 Gm./100 cc. blood.

Hospital Course.—A peritoneal tap performed on admission was negative. After the patient had received 1000 cc. of whole blood, her blood pressure stabilized at 130/80, and a laparotomy was performed. The peritoneal cavity contained approximately 2000 cc. of blood. Examination of the liver revealed a 5 x 10 cm. stellate laceration of the dome of the right lobe.

The laceration of the liver was packed with gauze and a large Penrose drain was placed to Morison's pouch and to the area of laceration.

The drains and pack were removed and the patient was asymptomatic by July 4, 1954.

On July 12, 1954, the patient became febrile and complained of right upper quadrant pain. This was followed by one episode of hematemesis, after which the hemoglobin was 8.8 Gm. A transfusion of 1000 cc. of whole blood was administered, and the hemoglobin was then 12 Gm. The pain in the right upper quadrant continued intermittently until July 20, 1954, when it became severe and radiated to the back. On that day the hemoglobin dropped to 8.0 Gm.

On July 21, 1954, after the patient had been given a transfusion, a laparotomy was performed. The gall bladder was filled with clotted blood. The subphrenic space was explored and contained a "deep purplish" exudate. Brisk bleeding occurred from the lacerated area of the liver and was controlled by repacking with gauze. A cholecystostomy was performed and the abdomen was drained.

The pack was gradually removed over a two-week period. The patient was asymptomatic until August 10, 1954, when mild right upper quadrant pain occurred. Three hours later the pain had become severe and the patient began to bleed profusely from the site of the previously removed subphrenic drain and through the cholecystostomy tube. In a two-and-one-half-hour period, the patient received 7500 cc. of whole blood. The patient died in the operating room from shock before definitive surgery could be accomplished.

(Continued on Page 336)

ASSOCIATION OF INTRACRANIAL MENINGIOMA WITH PITUITARY ADENOMA

Report of Successfully Treated Case

J. GRAFTON LOVE, M.D., and
CHARLES M. BLACKBURN, M.D.
Rochester, Minnesota

IN SPITE of our present-day accuracy of neurologic and ophthalmologic diagnosis, a neurologic surgeon must be prepared for unexpected developments when he opens a patient's skull.

The diagnosis of a chromophobe adenoma of the pituitary gland usually is not difficult. The visual loss which causes the patient with such a lesion to consult a neurosurgeon ordinarily is rather classic, and the roentgenologic finding of an enlarged and eroded sella turcica is most often pathognomonic of a pituitary tumor, yet occasionally the surgeon uncovers a lesion that is entirely unexpected. The following report is illustrative of the unexpected situation that may be encountered once in a while.

Case Report

A sixty-five-year-old woman came to the Mayo Clinic complaining chiefly of poor vision in her right eye. The decreased vision on the right had been noticed for about five months. A local ophthalmologist had prescribed "drops"; these had been used without improvement. Two weeks before coming to the clinic this patient saw another ophthalmologist, who examined her eyes and advised roentgenologic examination of her head, which was done. The patient said he told her she had a "boil" in her head, and that it needed to be "dried out" with x-ray treatments. A neurosurgeon saw her and was said to have concurred in the diagnosis and treatment. During the two weeks prior to the patient's visit to the clinic the husband had noted a slightly increased prominence of her right eye.

In addition, the patient complained of mild bitemporal and biparietal headache. There had been no symptoms to suggest endocrine dysfunction.

The patient was 59¼ inches tall and weighed 148½ pounds without her clothes. There were no physical signs of pituitary insufficiency. The blood pressure was 178 systolic and 108 diastolic, measured in millimeters of mercury. The uterus was surgically absent (hysterectomy had been done in 1930). Vision was recorded as 6/20 on the left and 1/60 on the right. There was 1 mm. of proptosis of the right eye. The palpebral fissure was 2 mm. wider on the right than on the left. Extra-ocular motions were generally restricted in all directions in the right eye. The tension in the right eye was 27, whereas in the left eye it was 20. The ocular fundi revealed a moderate degree of pallor, and the arterioles of the retina showed the changes of chronic hypertension.

The fields of vision when plotted revealed bitemporal hemianopsia, with involvement of the right optic nerve and right side of the optic chiasm.

Neurologic examination did not disclose any additional abnormalities. Roentgenologic examination of the head demonstrated erosion and enlargement of the sella turcica, which condition was interpreted as being due to a large intrasellar tumor (Fig. 1). Roentgenograms of the optic canals showed them as normal. The superior orbital fissure on the left was slightly larger than that on the right. This was thought to be of no clinical significance.

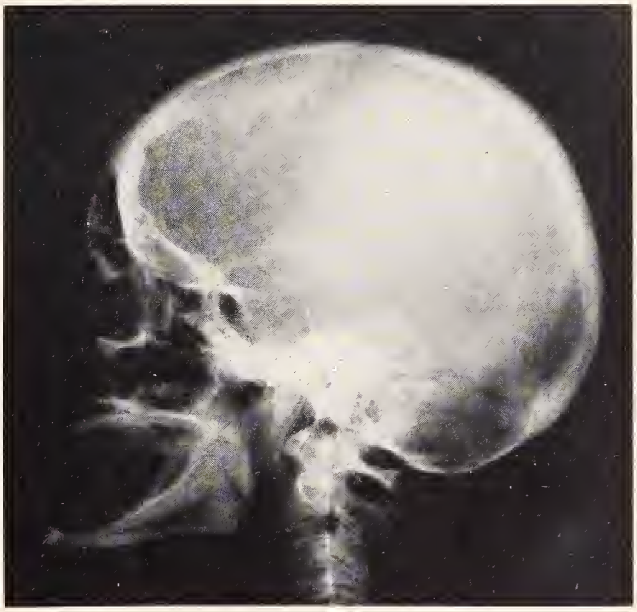

Fig. 1. Lateral roentgenogram showing enlargement and erosion of the sella turcica, such as is seen in the presence of a pituitary tumor.

The basal metabolic rate was within normal limits (+6), as were results of blood counts and urinalysis.

We knew we were dealing with a progressive lesion involving the sella turcica and the visual apparatus. We felt the lesion was neoplastic and not an aneurysm of the carotid artery or the circle of Willis. In an attempt to explain all the ophthalmic signs and the eroded sella turcica we considered three different neoplasms which might be conceived to be present, and we assigned the following percentages, in terms of likelihood, to each: meningioma, 50 per cent; pituitary adenoma, 40 per cent; pituitary adamantinoma, 10 per cent.

Operation for the removal of a tumor involving the visual apparatus was advised and accepted.

Dr. Love is in the Section of Neurologic Surgery and Dr. Blackburn is in the Section of Medicine at the Mayo Clinic and Mayo Foundation. The Mayo Foundation in Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

Although there was no clinical evidence of pituitary insufficiency, the patient was given 200 mg. of cortisone intramuscularly forty-eight, twenty-four and two hours prior to craniotomy. It was thought advisable to supply the patient with such a depot of cortisone in the event that operation resulted in acute anterior pituitary insufficiency, either temporary or permanent. After the operation the administration of cortisone was gradually reduced over a period of several days.

Right transfrontal craniotomy was performed with difficulty with a malleable needle. It was necessary to convert two of the perforator openings into burr openings, but we still were unable to turn the flap without lacerating the dura. There was sharp bleeding at the anterior temporal opening, and ultimately we realized why. After the flap had been turned, and as we were inspecting the lacerated dura, we noted a hard, firm, nodular mass in the anterior temporal hole and a thickened dura; this mass proved to be a typical meningioma of the Sylvian fissure, and it was of very respectable size. For a moment, it was thought that this process might be a lateral extension of a meningioma of the sphenoid ridge. However, this entire tumor mass was removed in one piece after the nutrient artery had been closed with silver clips and there seemed to be no deep extension.

Yet it was obvious that this tumor was not producing the defect in the visual fields or causing the erosion of the sella turcica. We then stripped the adherent dura from the roof of the right orbit and exposed the visual apparatus. As we did so, we saw a large, thick-walled, nonpulsating tumor projecting above the sella and extending under the right optic nerve and internal carotid artery, both structures being displaced upward. The tumor presenting between the optic nerves and anterior to the chiasm was aspirated without obtaining blood. Then it was incised and typical pituitary adenomatous tissue was obtained. A considerable amount of tissue was saved for microscopic examination. Most of the tissue within the capsule was removed with a sucker.

A large adenomatous extension under the right optic nerve and internal carotid artery was pulled within the sella and removed. Extensive resection of the capsule was carried out; a piece along the floor was left. The bleeding was under control. The lacerated dura was covered with a sheet of animal membrane. The bone was wired in place with two pieces of wire. The wound was closed in layers without drainage. The 30 cc. of fluid which had been removed through the malleable needle was replaced in the spinal canal.

The pathologist's report identified these lesions as (1) a meningioma of the Sylvian fissure, and (2) a chromophobe pituitary adenoma which was intrasellar, suprasellar and extrasellar (supratentorial) in extent.

The patient received 500 cc. of blood during these procedures.

The patient's postoperative convalescence was uneventful and very gratifying. For one twenty-four-hour period (the second) after operation the intake and output of fluids was markedly increased (4,800 cc. intake and 5,300 cc. output). However, this condition spontaneously subsided without the employment of posterior pituitary substance.

The patient was dismissed from the hospital on the eighth day after operation, and from the care of the Mayo Clinic four days later. At the time of dismissal there had been some improvement in vision in the temporal field of the right eye and ocular motions were normal.

HEMOBILIA

(Continued from Page 334)

Autopsy revealed an area of liver necrosis measuring 5 x 6 cm. There was an erosion of a branch of the right hepatic artery deep in the substance of the right lobe of the liver.

Discussion

The sparsity of reports in the literature would suggest that bleeding into the gastrointestinal tract as a result of liver injury is a rare condition.[1] It is more likely that bleeding is often mild or masked by the associated problems presented by other injuries.[2] Hemobilia may be an early or late complication of liver injury as in the two cases reported.

Central rupture of the liver occurs by compression of the inelastic and fragile liver parenchyma. When a tear occurs, arteries, veins, and bile ducts are ruptured and the cavity fills with blood and bile. This process produces necrosis which in turn produces secondary hemorrhage. The only exit of drainage in central laceration is into the bile ducts or the blood vessels. Autolyzing ferments at the site of injury, sequestration of damaged liver tissue and infection of devitalized tissue are other causative factors in the etiology of hemobilia.

Usually, hemobilia is an insidious process since the central portion of the liver has no sensory nerve endings. Only after the liver enlarges enough to distend the capsule or after a tear occurs in the capsule will pain be present. Shortly after the occurrence of bleeding into the bile ducts, the symptoms of typical biliary colic become manifest. This occurred in the second case reported.

Sparkman gives the symptoms of hemobilia as a triad of abdominal injury, pain, simulating biliary colic and gastrointestinal hemorrhage. In the second case reported, the patient experienced pain prior to the onset of hemorrhage and could predict the episodes.

Summary

Two cases of hemobilia are reported. In the first patient, the hemobilia occurred at the time of the injury, and in the second it occurred twenty-one days after injury.

Knowledge of this entity and the possibility of segmental necrosis of the liver substance should permit earlier debridement and decrease the chances of secondary hemorrhage.

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CASE PRESENTATION

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Duluth, Minnesota

The patient, a white nun, was originally admitted to St. Mary's Hospital in 1945 at the age of sixty-one for multiple contusions suffered when she fell while hanging curtains. Except for a fracture of a metacarpal bone, there were no other significant findings or complaints. At that time, the hemoglobin was 12 gm. per cent, red blood count 4,210,000/cu. mm. and white blood count 6,600/cu. mm. Recovery was uneventful.

The second admission was in 1946, at the age of sixty-two, for "strain of lumbar spine." While cleaning windows, she strained her back. She was admitted with severe pain in the lower back over the lumbar and sacral spine. There was local tenderness to deep pressure. Except for hyperactive tendon reflexes throughout, neurologic examination gave negative findings. The hemoglobin was 12.8 gm. per cent, white blood count 8,700/cu. mm., red blood count 4,320,000/cu. mm. Complete urinalysis showed no abnormalities. The spinal fluid showed 7 white cells/cu. mm., 64 mg. per cent sugar, 740 mg. per cent chlorides and 25 mg. per cent total protein. The colloidal gold curve and serology were normal. X-rays of the area were not remarkable except for slight wedging of the first and fourth lumbar vertebrae with hypertrophic changes which might represent old compression type fractures. The spinogram was negative for herniated intervertebral disc. The patient was fitted with a lumbosacral support and recovered uneventfully.

The final admission in February, 1954, occurred when the patient was sixty-nine. While sitting in a chair, she bent over, fell and struck the right hip, suffering immediate, severe pain in that area. Past history included pulmonary tuberculosis in 1907 for two months and a few minor surgical operations. System review revealed no abnormal findings except for blindness in the left eye for two years. Physical examination showed an obese white woman with severe pain in the right hip. A mature cataract was present in the left eye. The tongue was in the midline, and the pharynx showed no unusual changes. The chest was examined only anteriorly but showed no abnormalities. The blood pressure was 156/86 mm. of mercury. No other cardiac physical findings were present. The abdomen showed no abnormalities. There was an obvious fracture of the neck of the right femur. X-ray examination confirmed the fracture. A portable chest x-ray on the second hospital day was unsatisfactory, suggesting possible atelectasis on the right and an elevated right diaphragm. An electrocardiogram was normal. On the third hospital day, the hemoglobin was 16 Gm. per cent, the white blood count 14,500/cu. mm. with 73 per cent neutrophiles, 24 per cent lymphocytes. On the fourth hospital day, the hemoglobin was 14 Gm. per cent, platelets 135,300/cu. mm. A preoperative physical examination on the second hospital day showed a blood pressure of 150/80 mm. of mercury, pulse of 96, respirations 20/minute and temperature 100.2°; otherwise, there had been no change. The urine, which showed a specific gravity of 1.026, was otherwise normal. On the fourth hospital day, a hip-nailing procedure was carried out without difficulty. The patient progressed satisfactorily, and on the seventeenth hospital day she began to be up in a chair. On March 19, 1954, (thirty-third hospital day), the patient complained of a thick feeling in the tongue and difficulty in speaking. No other changes were found on examination. On March 22, she developed slurred speech as well as trouble in chewing and swallowing. The tongue deviated to the left. On March 24, she had two sudden episodes of dyspnea lasting about ten minutes each. The lungs were dry and the heart rhythm regular. On March 25, panting respirations were noted. An electrocardiogram repeated on this day was again normal. Neurological examination showed a nasal voice tone. Hypalgesia was present in the fifth finger and less in the fourth finger of the right hand. Deep reflexes were moderately hyperactive. Speech was difficult. Regurgitation of liquids occurred through the nose. No cerebellar signs were found. On March 26, a spinal puncture showed a pressure of 410 mm. water, and 4.5 cc. of clear colorless spinal fluid was removed. This contained no cells, but 45 mg. per cent sugar, 14 mgm. per cent total protein and 755 mg. per cent chlorides were present in the fluid. Pandy's test was negative, as were the gold curve and Wassermann tests. On this day the white blood count was 7,750/cu. mm., the hemoglobin 11.8 mg. per cent. On March 26, there was little change with no facial weakness detectable and an active gag reflex was present. On March 27, examination of the ear, nose and throat gave essentially negative findings. Pharyngeal muscle function was difficult to evaluate but all muscles moved as did the vocal cords. On March 29, the previous symptoms increased. On March 30, physical signs of diaphragmatic paralysis bilaterally were confirmed by fluoroscopy. The tongue became uncontrollable, and there was much respiratory distress. A tracheotomy was performed. Thoracic muscle action was poor. The patient was placed in a Drinker respirator. On March 31, her condition

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From the files of the Department of Pathology, St. Mary's Hospital, Duluth, Minnesota. Discussed on June 25, 1954.

was grave with obvious signs of insufficient oxygenation. Slight nystagmus was apparent with the gaze to either side. The left palpebral fissure was narrower than the right. The left corneal reflex was less active than the right, and there was slight deviation of the tongue to the left. Her condition progressed to complete coma and death that night. The white blood count the day of death was 21,500/cu. mm., with 82 per cent neutrophiles. The temperature was normal throughout her illness until the day of death when it rose to 101 degrees.

Differential Diagnosis

Dr. L. R. Gowan: Review of the protocol as well as the nurse's notes indicate a rather marked fluctuation in the severity of symptoms from hour to hour. The past history also includes several falls with resultant fractures (ribs, ankle, wrist, hip). The x-rays served primarily to corroborate the clinical impression of diaphragmatic paralysis and also show no bone signs of hyperparathyroidism.

A broad view of the problem would demand consideration of many entities including:

1. Anterior horn disease
2. Bulbar palsy (arteriosclerotic)
3. Pseudobulbar palsy
4. Basilar artery thrombosis
5. Vascular anomaly (aneurysm of basilar artery)
6. Tumor (pontine)
7. Hyperparathyroidism
8. Myasthenia gravis
9. Guillian-Barre's syndrome
10. Bulbar multiple sclerosis
11. Multiple CNS metastases
12. Toxic encephalitis (CO, As, etc.)

All of these conditions may produce bulbar signs. In the process of elimination, the negative spinal fluid findings rule out poliomyelitis, or encephalitis or other anterior horn-cell disease not already ruled out by the history. The absence of fibrillation and vacillation of the clinical course are not characteristics of bulbar palsy. Basilar artery thrombosis causes quadriplegia and death in about twenty-four hours, if complete; if incomplete, I would expect more neurological signs as a result of brain stem and cranial nerve nuclear involvement; the same arguments apply to aneurysms. Pontine tumors are rare at this age; furthermore, the patient had no headaches, dizziness or visual disturbance. Hyperparathyroidism must be considered, especially with the long history of bone aches, demineralization and leg weakness; the x-rays show no cystic changes, but these are not invariably present. Guillian-Barre's disease is a diagnosis which, if made at all, should be considered only in a patient with a fairly characteristic picture. The patient's age is the biggest argument against multiple sclerosis, although this disease could begin at this age. I would expect more neurological findings in the extremities, however; furthermore, even apoplectic onsets of multiple sclerosis usually produce a history of previous symptoms upon specific questioning. There is nothing to suggest a primary tumor, and multiple metastases would not have produced such a fluctuating clinical picture. No exposure to a toxin makes toxic encephalitis untenable as well as lack of more widespread evidence of brain involvement.

Myasthenia gravis and pseudobulbar palsy must be considered seriously. There is a past history of hypertension; therefore, vascular disease is possible. There is no atrophy; pyramidal tract involvement is common in pseudobulbar palsy, absent in this case. Past history of a stroke is also common, again lacking here.

Myasthenia gravis may appear in older people. The clinical vacillation in severity of symptoms is quite consistent with this diagnosis. Respiratory muscle weakness is common in myasthenia gravis. Clinically, myasthenia gravis usually falls into three groups: (a) bulbar, (b) ocular, (c) myasthenia of the extremities. This case was primarily bulbar in type with minimal ocular signs. Of course, most cases are much more gradual in their onset than was this case, but such sudden onsets occur and may be an exacerbation of a subclinical form of the disease. Unfortunately, we have no therapeutic test (prostigmine) recorded in this case. I believe this patient died of an acute form of myasthenia gravis but cannot definitely rule out pseudobulbar palsy. The other possibilities are improbable for the reasons mentioned above.

Dr. I. T. Clark: The chest x-ray shows high diaphragms and right lower lobe atalectasis. No evidence of thymoma is seen.

Doctor Gowan's Diagnosis

1. Myasthenia gravis
2. Possible pseudobulbar palsy

Anatomic Diagnosis

1. Benign Thymoma
2. Acute myasthenia gravis

Dr. A. C. Aufderheide: The heart weighed 380 grams. It was otherwise normal; except for lower lobe atelectasis on both sides, the lungs were not abnormal. The gastrointestinal tract showed no pathologic lesion. A small benign fibroma was present in the right ovary. A benign cortical adenoma (3 cm. in diameter) was found in the right adrenal gland. Osteoporosis of the lumbar vertebrae was apparent. Four parathyroids were grossly and microscopically normal. The parenchymal and vascular tissue of the brain and spinal cord showed no gross or microscopic changes. Microscopically, the skeletal muscle of the diaphragm was not abnormal. Situated in the superior anterior mediastium in the region of the thymus gland was an encapsulated, lobulated, hard tumor measuring 4 x 3 x 3.5 cm., which on cut section was white, trabeculated with a few hemorrhagic areas. Microscopically, the tumor consisted of larger epithelial-appearing cells mixed with smaller cells indistinguishable from lymphocytes (Fig. 1). The picture is that of a benign thymoma.

The absence of central nervous system lesions, the presence of a benign thymoma and the clinical history led us to conclude that this was a case of acute myasthenia gravis.

Discussion

Dr. H. Fisketti: Are malignant tumors of the thymus ever associated with myasthenia gravis?

Dr. A. C. Aufderheide: Rarely, a few cases have been reported, but with these few exceptions, thymomas accompanying myasthenia gravis are benign.

Dr. A. C. Aufderheide: About 50 per cent of myasthenic patients have a thymoma or hyperplasia of the thymus on x-ray examination.

Dr. J. Coll: Johns Hopkins University has reported up to 80 per cent at autopsy, if a careful search is made.

Intern: What is the current status of thymectomy in myasthenia gravis?

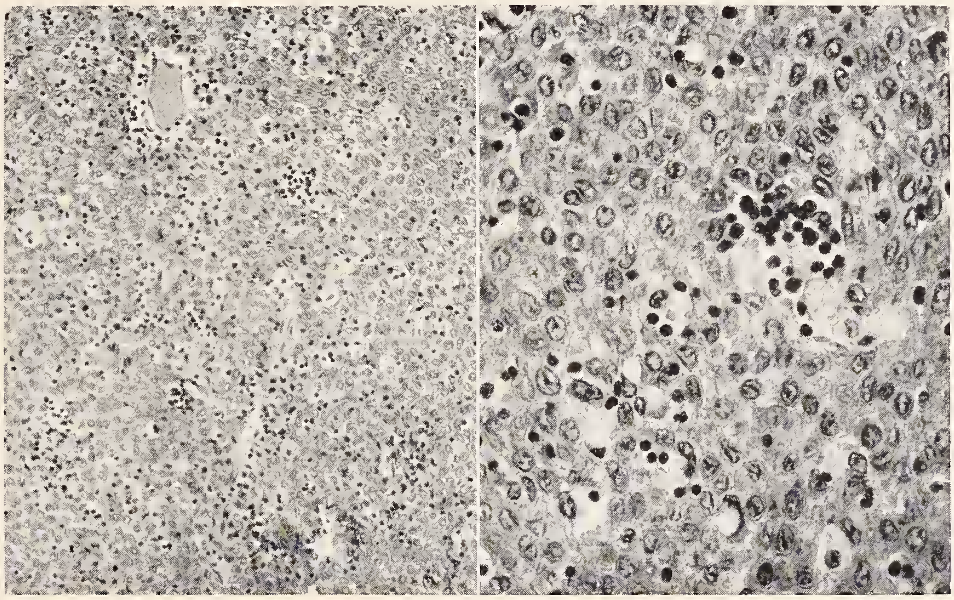

Fig. 1. (A) Low power photomicrograph of the thymic tumor. Note the cellularity. (B) High power photomicrograph of the thymic tumor showing the two types of cells. The smaller, dark-staining cells resemble lymphocytes while the others are much larger and stain less deeply.

Dr. J. K. Butler: What is the treatment of choice?

Dr. J. B. Moyer: Neostigmine tablets are the most popular; the methyl sulphate form of neostigmine is available in ampule form for emergencies. Other products are also available; I haven't had much experience with them but I understand they are quite toxic. It is important that *enough* drug be given; too many myasthenic patients are undertreated by their physician.

Dr. N. E. A. Leppo: What is the incidence of thymoma in myasthenia gravis?

Dr. Gowan: Debatable at present. The Lahey Clinic staff feels that their series shows definite benefit from thymectomy; the Mayo Clinic staff felt otherwise but recently supported the eastern school after re-evaluating their results. Thymectomy usually produces a severe postoperative transient exacerbation of the disease so that treatment must be vigorous during that phase.

I think this patient had had subclinical myasthenia gravis which probably caused enough muscle weakness to result in her frequent falls and that the present episode represents an acute exacerbation of the disease.

POSTOPERATIVE WALKING

Although having surgical patients ambulate soon after their operation has been widely practiced since World War II, Palumbo believes it should be followed even more extensively than it is today. He feels that hesitancy on the part of many surgeons is due to fears that certain undesirable effects such as wound disruption may occur. The author disagrees and notes that "Through disturbances in metabolism, prolonged bed rest produces weakness and loss of tone of both smooth and skeletal muscles, loss of calcium and nitrogen from the body, reduced vital capacity, decreased cardiac output, slowing of circulation, and a delay in wound healing. These changes may result in an increased incidence of complications of the gastrointestinal tract, as evidenced by ileus, constipation, nausea, vomiting, and/or decrease or loss of appetite." Conversely, "Early ambulation enhances wound healing and reduces other complications which may occur in the gastrointestinal tract, in the cardiopulmonary and in the peripheral-vascular system." —Palumbo, L. T.: Early postsurgical ambulation, *Journal of the Iowa State Medical Society,* 45:12 (Jan.) 1955.

President's Letter

IN APPRECIATION

This issue of Minnesota Medicine should reach you about the time of the state meeting, and the impact of the work of your state association, plus the work of your local arrangements committees in bringing to you this excellent annual meeting, should be fresh in your mind.

On occasion, I have been asked by members of local units of medical societies, "What does my county medical society do for me?" At such times, even though I could talk indefinitely on the subject, I have often felt completely tongue-tied and frustrated. Perhaps I have felt somewhat as you would feel if, after you had successfully seen a patient through a critical illness, he were to say to you, "I guess I would have done just as well if I had gone to bed and seen it through without you, eh, Doctor?"

That the physician can ask such a question about his medical society, is evidence that he is not even a member of the society in his own heart. If he had helped to cook the meal, he would inevitably have tasted some of the bounties of society membership in the process.

So many members have participated in the work of our state association that to single out any one man or group would be an injustice. The work of the Hennepin County Medical Society, our hosts at the state meeting; the Insurance Liaison Committee; the Committee on Malpractice; the committee studying our functions in civil defense; Maternal and Child Health Committees; and the editing, publishing, and editorial boards of Minnesota Medicine, to name only a few, have been so outstanding that our membership owes them a debt of gratitude beyond their ability to repay.

It is, indeed, a source of satisfaction to your officers and Council that when help and advice is needed, it is freely given. Time and efforts seem of little consequence to these devoted members of our Association when they are asked to contribute to the profession of our state.

In the light of the accomplishments of the committees, on both scientific and non-scientific work, can any one of our members seriously ask, "What does my society do for me?" Rather let us ask, "How can I aid in the dedicated work of our Association, whose object is the improvement of public health, the advancement and dissemination of scientific knowledge, and the protection and advancement of our profession in the accomplishment of these objectives?"

Arnold O. Swenson

President, Minnesota State Medical Association

Editorial

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

GENERAL PRACTICE SYMPOSIUM

This number of Minnesota Medicine contains material presented on October 20, 1954, at the Fourth Annual Refresher Course of the Minnesota Academy of General Practice in Rochester.

This meeting was representative of what I feel to be one of the richest heritages of American medicine. Five hundred general practitioners registered and attended the meetings wherein fourteen specialists gave of their time and knowledge. The meeting demonstrated a co-operation for the good of the patient that is typical of the medical profession. Nothing could so well belie those vocal few who would have us believe that the specialist and the general practitioner are continually at each other's throat. The attendance and attention of the general practitioners earned them the respect of the speakers.

Members of the Minnesota Academy of General Practice have seen these meetings grow and have watched the enthusiasm mount each year. This annual meeting, along with the regional meetings that are held throughout the state, gives general practitioners an opportunity to get postgraduate instruction that will qualify them for continued membership in the Academy. It also provides nonmembers with an opportunity to "refresh" themselves.

The papers included in this symposium are not meant to be a basic contribution to the medical literature but do review some of the basic considerations in diagnosis and treatment of the conditions discussed. It is considered that their inclusion in Minnesota Medicine might be of value to physicians unable to attend the meeting.

H. A. Wente, M.D., *Secretary-Treasurer*
Minnesota Academy of General Practice

BMD

The last twenty-five years have flooded us with alphabetic agencies, and he is indeed a dolt who doesn't understand UN, WHO, UNESCO, et cetera. The Armed Forces gave us a goodly number of useful alphabetics; e.g., SOP for Standard Operating Procedure (quite reminiscent of the s.o.b. that appears on clinical histories to indicate short of breath—beware of capitalizing these letters, though), and SNAFU for Situation Normal, All Fouled Up. The tendency to reduce communications to a series of letters has even carried into domestic life. We know an efficient head-of-a-family who used to telephone home just as he left his office to deliver the command, "PMC," and the wheels would begin to turn at home in anticipation of his arrival. This worked fine as long as he drank only coffee, because even his youngest child could comprehend this cryptic command, "Pour My Coffee." As he rose in the world, his tastes became more expansive and expensive, and an alternate translation appeared, "Pour My Cocktail(s)." Finally, when he went on a reducing diet and "Peel My Carrots" was added to the reasonable interpretations, things became so chaotic that he had to revert to imperative sentences rather than initials—like ordinary inefficient folks. Thus ends one success story.

Now this BMD business, which insidiously entered our lives via an innocent conversation, has to do with success in another way. It is a criterion of success. You remember how in order to pick the man-most-likely-to-succeed in our class, we first strove for a definition of success. We settled for health, wealth and happiness, without thinking that we still had not arrived at an objective definition. In any event, many of us were sure that we were successful when we could go around with roseate cheeks, a smiling face, and a new Buick for house calls. This rotund, bourgeois concept of success was soon elbowed out by the unsettling information that a man was successful only when he had the prerogative of choosing the interior decorator who would do his office—at least junior partnership in the firm.

Now this BMD came to us as a subheading of the interior decorative criteria of success when a friend of ours declared he was working for his BMD. We were unable to equate this with any degrees we'd heard of and were wondering whether it had to do with a new telescoped type of Bachelor-Doctor of Medicine when he gave us the literal translation: Big Mahogany Desk.

H. G. M.

SPECIALTY TRAINING IN ALLERGY

Allergy must now be considered as a clinical entity with its various phases integrated by known factors or implications. It is a specialty which must be correlated with all other specialties of medicine. There is a growing appreciation that allergy is wholly or partially responsible for so many diseases or symptom-complexes that it is expected to be met in the practice of the internist, pediatrician, dermatologist, otorhinolaryngologist, and general practitioner. They cannot escape from the responsibility of at least its recognition so that the patient may be referred to a properly trained allergist or they must become trained in the specialty themselves.

The primary allergic diseases, asthma and hay fever, afflict approximately 10 per cent of the total population, making this category of chronic diseases third in prevalence in the United States. We must also recognize that the mechanism of hypersensitivity is responsible for plant dermatitis, urticaria, eczema, allergic headache, allergic evidence of infectious diseases, rheumatic fever, serum sickness and drug allergies, thus making the incidence of allergy much higher.

Allergy in industry is becoming a serious problem, and many manufactured products now cause important allergic diseases. Allergic reactions to our modern drugs, particularly to antibiotics, are mounting to alarming proportions and becoming a very serious problem. Allergy today is a social problem, extending from birth to old age.

Certification, in itself, in internal medicine and pediatrics does not indicate that a physician is qualified for the diagnosis and treatment of allergic disease. The extremely small number of physicians subcertified in allergy attests to this. There are only seventeen AMA-approved institutions in the United States which train residents and fellows in allergy. A well-organized American Foundation for Allergic Diseases is now carrying on these functions of teaching and research. The impact of allergy on medicine must be strengthened by the education of physicians and the public alike.

Fred W. Wittich, M.D.

FARM PRICES

The medical profession should be very familiar with the American concept of profitable production; namely, that the units of production multiplied by the price per unit produced, less the cost to produce, means the eventual profit.

Farmers, like doctors, live and progress on profits. They do not live off just price or production. Farm production is subject to the vagaries of the weather, varies from year to year and from locality to locality. Farm prices should be allowed to flex in line with needed production, in order to keep down price depressing surpluses on the one hand and to stimulate needed production on the other hand. This is the basic concept of our free enterprise system.

Since in the late 1930's, the government has been trying artificially to peg prices to farmers in order to insure a measure of farm stability. In times of full farm production and reasonable costs, artificially pegged high farm prices bring high prosperity to farmers. On the other hand, if artificially pegged high prices create surpluses which force production cuts and higher costs, the farmers actually receive less net income. This is what has happened in agriculture within the last several years.

Cotton producers and tobacco producers voted and accepted high government prices and took acreage cuts in return. These cut-out acres went into wheat, corn and feed which in some cases were also price-supported too high, and the results were surplus produce and acreage cuts.

Today, we are witnessing the confusion of government interference in agriculture. Some commodities are artificially high priced above market demand and are causing ceiling prices and restrictions. Other commodities are supported flexibily and are feeling the effects of the spill-over of acreage cuts of high supported crops. Feeders and dairymen are feeling the effects of governmentally determined high feed costs, while their products are allowed to drop. Butter must drop because it is too high for its competition or continue to loose its market. If the socializers and communizers wanted to create confusion in agriculture, they couldn't have done a better job. Or maybe the boys that wrote and policed the bills did know what they wanted to do.

I hope physicians won't buy such government paternalism. Perhaps we can all work together to get back to the real system.

J. Delbert Wells, *Secretary*
Minnesota Farm Bureau Federation

LOSS OF HEARING IN CHILDREN

Loss of hearing in children is of two main types: (1) middle-ear dysfunction, which is often preventable, and (2) nerve dysfunction, for which no prevention or cure exists at present.

The Committee on Conservation of Hearing of the Minnesota State Medical Association conducted a survey of school children in the state. As a result, 1,028 children were reported to have loss of hearing sufficient to be a handicap in school and later life. Only 175 of these were under treatment or in special schools.

The committee prepared a brochure that has been mailed to all physicians in Minnesota. This brochure presents a concise digest of present opinion in the fields of treatment for loss of hearing, training of the deaf child and selection of a hearing aid. It should be filed in an available location by all physicians.

The Minnesota Department of Health is co-operating in the project by mailing letters to the parents of children discovered by the survey. Parents are advised to seek medical advice for diagnosis and for therapy or rehabilitation of the handicapped child.

This program demonstrates the close co-operation between organized medicine and public health agencies. Further co-operation by individual physicians is required to carry the project to a successful conclusion. With the information provided, every physician can give valuable advice to the parents of children handicapped with respect to hearing.

K. M. Simonton, M.D.

DIABETES DETECTION PROGRAMS

It is axiomatic that the earlier the diagnosis of an ailment, the better the prognosis for greater comfort, for prolongation of life and for cures. It is unfortunate that in some diseases, such as cancer, a diagnosis is quite impossible until the condition is fairly well advanced. Diabetes is one disease in which a correct diagnosis can be made quite simply. It can be suspected without the patient's even visiting the doctor, by having the urine examined for sugar. If glycosuria is present, then more careful tests can be made to establish definitely the presence or absence of the disease. Because of this simplicity for mass detection, diabetes detection drives have been instituted all over the country. Through this procedure, a possible one million diabetic persons might be found and placed under proper supervision and control.

The more recent introduction of the St. Louis Dreypak strip has simplified the entire procedure. The strips, after having been immersed in the urine and dried, can be sent through the mail to the laboratory with practically no inconvenience to the patient. Every community should arrange for the simple facilities necessary to carry out this procedure. From this center, the results may be transmitted to the patients. Such an arrangement should prove very simple and inexpensive. The co-operation of the medical profession in the Diabetes Detection Week conducted by the American Diabetes Association will help establish excellent public relations between the medical profession and the community.

In the Diabetic Detection Drive in Minneapolis in November, 1952, 32,063 specimens were examined, of which 657 (about 2 per cent) were found to be positive for sugar. Of these persons, further tests proved that 40 per cent were truly diabetic, and they were advised to consult their doctors for management and control.

Moses Barron, M.D.
Governor, Minnesota District
American Diabetes Association

HOW LONG SHOULD ONE KEEP VALUABLE PAPERS?

These days all of you have to contend with the problem of deciding how long records and other valuable documents should be retained. You are concerned with keeping not only those records, documents and papers necessary for the protection of your interests in the ordinary administration of living but also those which are necessary as a result of your professional practice.

Quite apart from the maintenance of files with respect to the treatment of patients, as to which you may have some kind of moral obligation, there is the possibility that at a future date you may be called upon in a proceeding in court or elsewhere to present your records for the purpose of verifying and establishing what has or has not been done. It is impossible to enumerate all of the instances in which a physician's records may be required. There may be occasions where it will be of value for you to use records merely to refresh your recollection as to whether you were in or out of the city on a particular date. It is quite possible that at some time you will be called upon to explain and even justify the method of treatment employed in a particular instance. Almost surely, you will be called upon at some time to explain the extent and nature of your income and of your deductions for income tax purposes.

As a rule of thumb, it seems desirable that account books and all other records reflecting income and expenditures should be kept until January first of the eighth year after the year to which they pertain. If a creditor attempts within that time to assert that a bill has not been paid, you will have your records available to dispute him. Or if the tax collector has some question as to the proper tax liability for a given year, the correct data will be available for his perusal and for use in court, if necessary.

Deeds, mortgages, leases and other documents pertaining to real estate are seldom of burdensome volume and should be retained indefinitely. A minimum tax file should be kept for an indefinite period. This should contain not only the returns but also sufficient supporting evidence to establish your income and your deductions. Papers having no particular present interest may be destroyed when their usefulness has been served. In this class fall expired policies of insurance where no loss has occurred (other than liability insurance), cancelled savings account books, contracts which have been fully performed, and similar documents. As to checks and records pertaining to items which do not have tax consequences, the eight-year period seems to present only reasonable risks, if any.

Of course, in any unusual situation, you will want to consult your lawyer because of the obvious danger that the general rule may not cover the particular situation.

Roland J. Faricy, *Attorney*
Saint Paul, Minnesota

TONGUE CANCER TREATMENT

Initial treatment of the primary lesion in cancer of the tongue and floor of the mouth seems to be best accomplished by radiation therapy, Dr. C. L. Ash, of Toronto, Canada, has concluded.

Dr. Ash presented his conclusions, based on a five-year study of thirty-nine patients with cancer of the tongue and ninety-five patients with cancer of the mouth floor, in a recent (April, 1955) issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

The Canadian radiation specialist is associated with the Ontario Institute of Radiotherapy and the Department of Radiotherapy in the Toronto General Hospital.

"In general, the secondary lesions of the oral cavity developing in the lymph nodes of the neck are most adequately treated by radical surgery," Dr. Ash said.

He admitted, however, there are certain exceptions to this rule.

"The patients we treated represent all the various forms of radiation therapy—radium in the form of molds, needles, radon seeds, teleradium therapy, or roentgen rays of 200 to 400 kv., as well as intraoral roentgen rays generated at 88 to 200 kv."

His overall results showed a 49.9 per cent "control"—that is, a total of five years with the lesion healed—for cancer of the tongue and 38.7 per cent control in cancer of the mouth floor.

However, in the early cases of tongue cancer, there was 67.0 per cent control—as compared with 32.3 per cent control among the late cases.

In mouth-floor cancer, there was 49 per cent control in the early group and 26.2 per cent in the late.

"Better results appear to attend the use of radium, or a combination of interstitial radium and roentgen rays, than is the case when external therapy is used alone," Dr. Ash said. His conclusion:

Studies at present are being made as to the value of extending the field of surgery in the so-called radiation "failures"—particularly when initial treatment has failed and the lesions remain localized.

AIR CONDITIONING

The wide differential in temperature between summer climates and some air-conditioned interiors poses a possible health question. "The chief difficulty centers about the 'comings and goings' of occupants of this artificial climate." In winter, the human body becomes adjusted so that persons leaving interiors heated to about 80 degrees (Fahrenheit) can enter a near-zero environment with only the addition of an overcoat and hat. In summer, however, moving from air-conditioned environments with relatively low temperatures into exteriors of high temperatures may result in a condition of "thermal shock." The ideal solution of a three to four stage transition is unfortunately impractical. "Under the circumstances described, much may be accomplished by clothing adjustments. In many air-conditioned spaces, particularly if there is excessive air motion, added garments are necessary, especially among women. The divestment of these garments aids the physiological state on re-entering the hot outer air. Humidification of the air is less important to health than was earlier contended. As to comfort, the degree of humidity may be more important but still is not fundamental. Some investigators, with respect to air conditioning in summer, advise merely dehumidifying the air to a low point without effort to lower temperatures, but this may give rise to an unpleasant sense of chilling owing to increased evaporation. In any event, a humidity between 40 and 60 per cent is regarded with favor."—Queries and Minor Notes: Air conditioning, *Journal of the American Medical Association* (157:1265 (April 2) 1955.

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

AMA REVIEWS LEGISLATION STATUS

After three months of the 84th Congress, the American Medical Association has published a summary of the status of federal legislation of interest to the medical profession. More than 200 medical-interest bills are being followed by the Washington Office of the AMA, and also considered are pertinent testimony before committees, committee actions, and all available information on future plans and objectives of the Administration and Congress. An early April issue of an AMA Washington Office Special Report reviews current status of legislation under several categories:

Reinsurance

Legislation provides an initial $25 million to start a trust fund that would be maintained by percentage payments from premiums of participating health insurance plans. The fund would reimburse voluntary health insurance plans (commercial and nonprofit) for abnormal losses in extending coverage and expanding benefits. This is on the "must list" of the Administration's health and medical care program. No hearings had yet been scheduled in early April.

According to the Report:

> "While endorsing the stated purposes of the bills (to promote the best possible medical care on reasonable terms), the American Medical Association again opposes the proposal on the grounds that (1) extensive private funds are available within the insurance industry for such purposes, (2) reinsurance doesn't provide a means for making insurable what otherwise would be an uninsurable risk, (3) it will not fulfill its intended purpose and might even inhibit the satisfactory progress made to date by voluntary plans, and (4) it is a potential subsidy."

Mortgage Guarantee

For a fee, this legislation would guarantee up to 95% of private mortgages for construction of non-government health facilities, to be owned and operated for profit or on non-profit basis. The mortgage could not exceed 80 per cent of the estimated value of the project and equipment, and maturity could not exceed thirty years. The guarantee would apply to refinancing, as well as to new construction and modernization. Hearings are to be scheduled.

The AMA Report states:

> "The Association (AMA) is actively opposed to this legislation on grounds that (a) private financing is meeting the need, and (b) the proposal represents intrusion by the federal government into a field where government help is not required."

Mental Health

The Administration wants a new five-year program of increased grants to states on a need-population-per-capita-income formula to help maintain and improve mental health facilities, to stimulate special projects, and to train more personnel. Several bills for grants to finance a three-year survey have been introduced, and another to create a permanent federal commission on mental health, which would study the problem, make recommendations and attempt to co-ordinate all work in this field, private as well as government. These proposals have made the most progress of all health bills, and the former, both in the House and Senate, are expected to reach the floor.

AMA policy on this matter is: wholehearted support of the survey bill, also favoring the Administration bill, but only as a temporary measure until Congress has been able to redefine the federal and local areas of responsibility in public health matters. The Report states: "This interest in mental health problems dates back to the founding of the AMA more than a century ago."

Federal Aid to Medical Education

Plans for federal aid to medical education range from "one-shot" construction grants to outright operating aid for schools increasing enrollment. Most prominent in this Congress are identical bills in the House and Senate, providing a five-year $250 million program of construction, expansion, modernization, and maintenance grants to new and existing schools. Hearings should be scheduled shortly.

According to the Special Report:

> "The Association favors one-time construction, renovating and equipment grants to medical schools on a matching basis similar to the Hill-Burton formula, but opposes federal grants for instruction or continuing grants for operation or maintenance. It approves the Hill-Priest bill, providing some specific changes are made in line with the above policy. It opposes the Burnside bill, until he accepts changes that have been suggested to him."

Doctor Draft and Military Medical Scholarships

The Doctor Draft extension bill would continue the present act for another two years beyond July 1, 1955. Under the scholarship bill, the government would pay up to $133 a month, plus tuition and fees. Students would be obligated for three years' active duty if the scholarship was for a year or less, and four years if for more than one year.

The Doctor Draft provision is expected to come up this session, but hearings had not been scheduled in early April. The scholarship bill is an official Administration measure, and is planned for consideration before the Doctor Draft act.

The AMA policy on the above is described as follows:

> "When there was a proven need for it at the start of the Korean War in 1950, the Doctor Draft was supported by the AMA. The AMA also supported one extension of the law. Now, in the face of the Defense Department's request for another (and peacetime) extension, the AMA's policy is unchanged. If it can be demonstrated that there is a continuing need for the act, the AMA will support the extension. Evidence so far presented has not established the need. The Defense Department admits the regular draft obligation will supply it with the young physicians it needs, but says that the Doctor Draft is required to bring in more experienced men with special skills and administrative ability. Without these, the Department insists, the military medical services cannot be maintained during the next two years. The AMA believes the services could do a great deal more to build up their regular Medical Corps and to make more efficient use of the experienced men now in uniform."

Regarding the military scholarships, the AMA supports the scholarship idea, provided that (1) a student not be approached by the military until he is fully matriculated in the medical school, (2) no student so selected receive any preferential treatment, and (3) number of military scholarships in a school not exceed 5 per cent of any year's class or the total enrollment.

Military Dependents

The present program of medical care for military dependents is irregular. Legislation would substitute a plan that would (a) apply a uniform definition of dependents to all services, (b) make all dependents eligible for the same amount of treatment, and (c) offer relatively the same degree of care regardless of residence of the dependents. Uniformed physicians and military hospitals would take care of all dependent patients within limitation of resources. The Secretary of Defense could set a token fee schedule to be charged dependents in military facilities.

No hearings had been scheduled at the release of the AMA Special Report. Two identical bills have been introduced in the House and Senate, and it is felt that the Administration will support some kind of change in the present law.

The AMA has no argument with the military services on most provisions of the Defense Department bill, but is unalterably opposed to one part of it: "Whereas the Administration thinks the military medical departments should care for all the dependents they can handle, the AMA feels that dependents, like other civilians, should be treated by private physicians and in private hospitals unless these private facilities are inadequate. In the latter case it would be the military's responsibility to care for dependents." The AMA is working on a set of guiding principles to propose for amendment when the bill is brought up.

TAX DEFERMENT BILLS MAKE SLOW PROGRESS

There seems to be little evidence that Congress will act on the Jenkins-Keogh-Ray type of bills this session. The Treasury Department has studied some of the proposals, but the Administration is not supporting any.

The purpose of the Jenkins-Keogh and Ray bills is broadly the same: "namely," according to a recent issue of the AMA *Journal,* "to encourage but not compel saving for retirement by the self-employed and by employed persons whose employers have not established pension and/or profit-sharing plans for them . . . Both bills make special provisions—past service credits—for those older self-employed and pensionless employed persons who have not had the benefit of tax deferment during the early and middle parts of their working lifetimes."

The American Medical Association supports all these bills, including medical expense deduction bills, but among the tax deferment proposals it prefers the Jenkins-Keogh approach.

NATIONAL BOARD NOTES EXAM REQUIREMENTS

The National Board of Medical Examiners has released general information on taking national board examinations, which should be of value to many physicians. The National Board is a voluntary and unofficial examining agency, the purpose of which is to "prepare and to administer qualifying examinations of such high quality that legal agencies governing the practice of medicine within each state may at their discretion grant successful candidates a license without further examination," their publication states.

The National Board certificate is accepted as a suitable qualification for the practice of medicine by forty-one licensing authorities in the states, the District of Columbia, Hawaii and Puerto Rico. Minnesota is one of these states.

The examinations are divided into three separate parts, I, II and III which must be taken and completed in numerical sequence. Examinations are held in February and June at medical schools in the U. S. and Canada; an additional examination in Part II is held in April and an additional examination in Part I in September.

Who Is Eligible?

The National Board admits to its examinations any student in or graduate of approved medical schools in the United States and Canada. A candidate is eligible for Part I when he has completed the first two years of work in an approved medical school; eligibility for Part II calls for successful completion of a four-year medical course in an approved medical school; a candidate is eligible for Part III if he has passed Parts I and II, received a degree of doctor of medicine, and served at least one year in an approved hospital internship.

Advantages Named

In its recent publication, the National Board examination advantages are outlined as:

> "Because the answer sheets are machine scorable, the grading can be accomplished rapidly, accurately, and impartially. With this type of examination, it becomes possible to determine the level of difficulty of each test and to maintain comparability of examination scores from test to test and from year to year for any single subject. Moreover, of even greater long-range significance is the facility with which the total test and individual questions can be subjected to thorough and rapid statistical analyses, thus providing a sound basis for comparative studies and for continuing improvement in the quality of the test itself."

Further advantages result from the fact that these examinations are set up in three parts, the Board states, and may be taken during the course of training in the medical school when the student is fresh from his preparation in the various subjects included in the medical curriculum.

Preparation of Examinations Noted

The summary of examinations by the National Board publication states that approximately a year goes into the preparation of each examination. Each committee member thoughtfully and laboriously writes test questions selecting that type best suited to a particular point. Each question is then subjected to the critical review of the other members of the committee; any doubtful item is revised or discarded. It concludes: "Hence, each examination contains only material that has been thoroughly worked over and agreed upon as appropriate, free from ambiguity and representative not only of important aspects of a subject, but also of high standards of medical education."

In 1902, Charles Richet, a French physician, theorized that an inoculation of a given protein in animals would produce a hypersensitivity to that protein. He termed this process "anaphylaxis," derived from the Greek *ana,* meaning "backward," and *phylaxis,* meaning "protection." The term, then, refers to the reverse of "prophylaxis," or a state of increased susceptibility to a drug protein or toxin following administration of the drug.—Wain, H.: The story behind the word, *Ohio State Medical Journal* 51:362 (April) 1955.

Current Concepts

IATROGENIC HEART DISEASE

CHARLES N. HENSEL, M.D.
Saint Paul, Minnesota

The word "iatrogenic" comes from two Greek words meaning "physician and producing"—actually generated by the physician. It is a term applied to disorders induced in the patient by autosuggestion, resulting from the manner in which the physician conducts his examination, and what he tells the patient.

Today, when there appears to be such a great increase in the incidence of coronary arteriosclerosis with coronary occlusion and myocardial infarction, everyone is aware of this growing menace to life and health.

Consequently, more than ever before, people with any pain or abnormal feeling in the left chest seek medical advice to learn how serious this symptom may be. If the doctor, after a brief history and physical examination, finds a regular heart of normal size and a normal blood pressure. and tells the patient, "I find nothing wrong, but you'd better be careful at your age," or, "I find nothing wrong, but you'd better take a little digitalis and avoid doing anything strenuous," then he is is likely to generate "iatrogenic heart disease."

Actually, when a doctor gives such advice, he is "straddling." He is not entirely sure himself. and so, should this patient later develop actual organic heart disease, he wants to be able to say, "You see I was right. I warned you."

When any person first comes to a physician complaining of a pain or an ache in his left chest that physician is charged with a very grave responsibility for making the right diagnosis and for telling the patient, at once, he has or he does not have heart disease.

The first contact with such a patient is most important, because how the doctor conducts himself may determine success or failure in handling the case. He must sit quietly with the patient and take plenty of time to obtain a complete history of his chest pain or ache. When did it begin—day or night? What was its relation to exertion, to rest, to anxiety? Was there any antecedent exhausting illness? Was there a recent bereavement, or grief over the death of a loved one? If such a death were sudden and from a heart attack, does this patient fear that he, too, may be so stricken?

Has there been a series of strains and anxieties in the mother of a large family, with many illnesses and many nights of broken sleep? Has there been a recent "bout" of upper respiratory tract infection or a tonsillitis or a pleurisy?

Inquiry into all of these factors will point toward the diagnosis of either organic heart disease or a functional heart disturbance, such as a nervous heart.

The next step is a complete physical examination with clothes removed. This should include pupillary reactions, examination of the oral cavity, palpation of the thyroid gland, examination of the lungs, determination of the heart borders and auscultation of the heart, determination of blood pressure, examination of the abdomen, rectal examination, study of the reflexes, a check for edema of the shins, temperature of the body, and examination of urine and blood.

Next follows the examination of the heart after exercise, using such a simple test as twenty hops on one foot and twenty hops on the other foot. noting the height of the pulse and the blood pressure and the time of their return to normal.

Then, and not till then, comes the observation of the heart and lungs under the fluoroscope, study of the heart silhouette from a 6-foot heart film, and reading of the electrocardiogram.

If all of these examinations and tests have produced normal responses, then and only then, can the examining physician be justified in telling the patient that he does not have any heart disease at all, that the dull ache in his left chest and the palpitation of his heart when he gets into bed at night are "fear reactions," because fear tends to pour extra adrenalin into the blood stream, and it is this extra adrenalin that makes his heart slam and bang against his ribs.

Tell him that the symptoms of organic heart disease are relieved by the horizontal position. that the pain of organic heart disease is not a dull ache, but a sharp stabbing pain, which passes with rest or standing still.

Tell him that a dull ache in the left chest is frequent in people with a nervous heart.

Look for other associated nervous signs, such as light headedness, trembling, sweating and an abnormal tendency to sigh.

Tell him that exercise is good for people with nervous hearts and that they should walk erect and breath deeply.

Do not ever hedge—tell him yes or no. And if he has no organic heart disease, tell him to do normal things and to lead a normal life. But whatever you tell him, be definite.

Reports and Announcements

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, annual meeting, Minneapolis, May 23-25, 1955.

Minnesota Academy of Occupational Medicine and Surgery, annual meeting, Minneapolis Athletic Club, May 25, 1955.

National

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, June 2, 1955.

American Medical Association, annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

American Proctologic Society, annual meeting, Hotel Statler, New York, New York, June 1-4, 1955.

International

International College of Surgeons, twentieth anniversary congress, Palais Du Conseil General, Geneva, Switzerland, May 23-26, 1955.

International Hospital Congress, Lucerne, Switzerland, May 30-June 3, 1955.

International College of Surgeons, Cape Cod regional meeting, Chatham Bars Inn, Chatham, Massachusetts, July 1-4, 1955.

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

AMERICAN ACADEMY OF GENERAL PRACTICE

The American Academy of General Practice is allowing twenty hours of informal credit to its members who attend the medical sessions of the annual meeting of the National Tuberculosis Association and its medical section, the American Trudeau Society. The sessions will be held in Milwaukee, Wisconsin, May 22 through 27.

Further information may be obtained from the Minnesota Tuberculosis and Health Association, 614 Portland Avenue, St. Paul, Minnesota.

AMERICAN ASSOCIATION OF ANATOMISTS

At the sixty-eighth annual session of the American Association of Anatomists, held at the Jefferson Medical College, Philadelphia, April 6 through 8, Dr. Edward A. Boyden, University of Minnesota professor emeritus of anatomy, was named president-elect of the organization. He will take office in April, 1956.

Dr. Boyden, who retired as head of the University of Minnesota's anatomy department in 1954, is currently a visiting professor of anatomy at the University of Washington, Seattle.

MINNESOTA CHAPTER, ARTHRITIS AND RHEUMATISM FOUNDATION

The formation of the Minnesota Chapter of the Arthritis and Rheumatism Foundation was completed in Minneapolis on April 13 with acceptance of a charter from the national foundation. Elected as officers of the organization were Dr. Charles Slocumb, Rochester, president; Dr. Macnider Wetherby, Minneapolis, first vice president, and Dr. Cecil J. Watson, University of Minnesota Medical School, second vice president.

Executive secretary of the chapter is R. V. Stevenson, with offices at 89 South Tenth Street, Minneapolis.

Purpose of the foundation is to fight arthritis and rheumatism by working for establishment of free clinics for treatment and to provide for research through establishment of scholarship and laboratory grants.

ST. LOUIS COUNTY SOCIETY

The St. Louis County Medical Society held a meeting at St. Luke's Hospital, Duluth, on April 14. Principal speakers at the meeting were Dr. William B. Martin and Dr. Kenneth A. Storsteen, both of Duluth. They discussed the diagnosis and treatment of hyperthyroidism, using radioactive substances.

MINNESOTA OBSTETRICAL AND GYNECOLOGICAL SOCIETY

Recently elected as officers of the Minnesota Obstetrical and Gynecological Society were Dr. Leonard A. Lang, Minneapolis, president; Dr. Rodney F. Sturley, St. Paul, vice president, and Dr. Edward A. Banner, Rochester, secretary-treasurer.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The Minnesota Society of Neurology and Psychiatry will meet at the Mayo Clinic, Rochester, Minnesota, on May 21.

Surgical clinics will be held at St. Mary's Hospital at 8:00 a.m., followed by presentation of papers at 10:00 a.m. Papers to be presented are as follows: "Chloropromazine and Reserpine in the Treatment of Chronically Ill Mental Patients," by Dr. M. C. Petersen and Dr. C. W. Baars. "Hormones and Psychoses," by Dr. Howard P. Rome. "Investigation of Environmental Factors in Schizophrenia," by Dr. Peter Beckett and Dr. Jane Watson.

Luncheon will be served at the Foundation House at 12:30 p.m. Principal speaker at the luncheon will be Dr. J. H. Tillisch, who will discuss "The History of the First Minnesota Regiment in the Civil War."

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1) To promote the welfare of the medical School of the University of Minnesota
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4) To publish a weekly bulletin
5) To advance the interests of the University of Minnesota Medical School and its alumni

The Minnesota Medical Foundation is desirous of obtaining the support of all members of the medical profession in this area. The Weekly Bulletin is a major project of the Foundation. It is an expensive undertaking and a fund must be accumulated to insure continued publication. All members of the Foundation automatically receive the Weekly Bulletin of the University of Minnesota Hospitals and The Minnesota Medical Foundation.

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Minneapolis 14, Minnesota

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Woman's Auxiliary

INTERNATIONAL HEALTH ORGANIZATIONS—PART I

Mrs. H. F. Wahlquist

A needless cause of embarrassment to many wives of physicians is not knowing how to explain to many people just what World Medical Association and World Health Organization mean. Each of us has a vague idea of what these two organizations represent—we know they are organized for health purposes, but we lack knowledge of their basic principles, and their differences. Perhaps circumstance has really never made it imperative that we study and ferret out facts about their organizational structure and purposes.

Constantly, I am hearing some one refer to WMA when she means WHO and vice versa. As wives of physicians, we must know more about these two organizations. They are both related to medicine, and yet both are basically different. Perhaps one of the most basic differences is that WMA is made up of physicians; WHO of technical representatives of member states. As wives of physicians, we need to know more than this!

In these few brief paragraphs, I shall attempt to clarify the purpose of each organization and in later comments describe the organizational structure and functions of each.

The World Medical Association (WMA) and World Health Organization (WHO) are two international organizations. The WMA is non-governmental; it is an organization of national medical associations and its members are solely physicians. I say "solely," for in the United States a WMA U. S. Committee, Inc., of which other citizens may become a member, has been formed. The WHO is inter-governmental; it is a branch of the United Nations and represents the governments of the world in the field of health. It is a specialized technical agency of the United Nations system, but under a separate treaty with membership independent of membership in the United Nations and with its own governing assembly. The WHO is supported entirely by government funds. The WMA is supported by dues and voluntary contributions.

When the Constitution of WHO was written in 1946, specific functions were assigned in three general categories:

1. Functions relating to international sanitary regulations, causes of death, nomenclature of diseases, new drugs.

2. Coordination of health activities of governments for the eradication of epidemic and endemic diseases, the promotion of research in fields of public health and the promotion of improved standards in the medical and related professions.

3. Assistance to governments in strengthening national health services, furnishing technical assistance, promotion of improved housing, nutrition and sanitation and the promotion of maternal and child health.

Its real objective is to aid in the long term solution of a country's health problems through strengthening the national health service, rather than developing *specific programs.*

The World Medical Association aims to promote closer ties among the national medical organizations and thus among the physicians of the world. It seeks to serve as a forum for discussion of mutual problems. Its aim is to disseminate information and to raise the standards of health, medical education and medical care throughout the world. Through its officers, it can present the points of view of the medical profession before other international bodies; as WHO, UNESCO, giving physicians a voice in international affairs when matters of medicine and health are discussed. Perhaps the most significant of its aims is to work toward world peace.

(Parts II and III will appear in succeeding issues.)

CLARKFIELD HOLDS HEALTH DAY

Sponsored by the Clarkfield Community Hospital Auxiliary, a successful "Health Day" was held on Saturday, April 2, in the High School Auditorium in Clarkfield.

Mrs. M. I. Hauge, first chairman of the hospital auxiliary and state chairman of *Today's Health* for the Woman's Auxiliary to the Minnesota State Medical Association, served as chairman of a very active health day committee.

The afternoon and evening programs included a talk on "Child Accidents" by Dr. A. B. Rosenfield, Minnesota Department of Health, a talk on mental health by Rev. Fred Norstad, President, Citizens' State Mental Health Association, and panel discussions on child health, the aging population and the new wonder drugs. Music and numerous information booths helped round out an excellent program to a large crowd from Clarkfield and the vicinity.

RAMSEY AUXILIARY REPORTS ACTIVITIES

Mrs. L. T. Simons

Mrs. W. P. Gardner, president, called a board meeting on April 18 in the Medical Arts Library. Members made cancer dressings during and following the meeting. Annual reports of all committee chairmen were presented and plans for the May meeting were discussed.

Mrs. Duane Ausman and Mrs. E. R. Sterner, hospitality chairman and co-chairman, took reservations for the monthly luncheon meeting, held in the Commodore Hotel on April 25. Mrs. S. M. Loken, program chairman, presented Dr. Clarence Rowe, assistant teaching professor in psychiatry at the University of Minnesota and director of the Hamm Memorial Psychiatric Clinic, who was the main speaker on the program.

In Memoriam

RUDOLPH A. BEISE

Dr. Rudolph A. Beise, Brainerd physician, died April 4, 1955. He was seventy-eight years old.

Dr. Beise was born in Medo, Minnesota, in 1877. He attended school in Mankato before going to the University of Minnesota and Rush Medical School. He interned at Cook County Hospital in Chicago and started his practice in Brainerd soon afterward.

During his active life, Dr. Beise participated in many civic affairs. He was mayor of Brainerd from 1915 to 1919. He was a member and secretary of the Brainerd Park Board. In 1953, Governor C. Elmer Anderson selected him to represent Minnesota at the First Western Hemisphere Conference of the World Medical Association.

Dr. Beise was a vestryman of St. Paul's Episcopal Church. He was a charter member of the Brainerd Rotary Club and served as its president from 1923 to 1924. In 1951, the club held a testimonial dinner honoring him for his fifty years as a practicing physician.

Dr. Beise was chief of staff at St. Joseph's Hospital in Brainerd. He was a member of the Upper Mississippi Medical Society, the Minnesota State Medical Association and the American Medical Association. He was a Life Member of the State Association and a member of the "Fifty Club" for doctors who have been in practice for fifty years.

He is survived by his wife, the former Minnie Keller; a daughter, Mrs. Ruth Louise Quanstrom, Brainerd; and a sister, Ida Beise, of Ada.

HUBERT TOWNSEND SHERMAN

Dr. Hubert T. Sherman, long in practice in southern Minnesota, died March 23, 1955, in Cambridge. He had been a medical staff member at the Cambridge state school and hospital since 1944.

Dr. Sherman was born at Wykoff, Minnesota, in 1879. He attended high school at Minneapolis Central and continued his education at Hamline University and the Minneapolis College of Physicians and Surgeons, graduating in 1904.

Dr. Sherman practiced in several Minnesota locations, spending brief periods of time at Becker, Minneapolis, Grand Meadow, Bellingham and Echo. He spent many years in practice at Big Lake, Franklin, and Plainview.

While at Plainview, Dr. Sherman served as President of the Wabasha County Medical Society. He was a Life Member of the Minnesota State Medical Association and was a member of county societies where he was practicing.

He is survived by his wife, Rachel; three daughters, Mrs. Will McCoy, Rock Falls, Ill., Mrs. Clifford Kast, Aitkin, Minn., and Mrs. Roy Peterson, Mahtomedi; three sons, John and Hubert, both of Minneapolis, and Roy, of Albuquerque, New Mexico; a sister, Mrs. H. E. Hart, Long Prairie, Minn.; eleven grandchildren, and three great-grandchildren.

JAMES CORY FERGUSON

Dr. James C. Ferguson died April 17, 1955, at the age of seventy-nine. He had practiced medicine for over fifty years.

Dr. Ferguson was born at Fort Totten in what was then the Dakota territory. He attended school in Cooperstown, New York, the home town of his mother. He came west to attend the University of Minnesota, graduating in 1901.

He combined his internship with work as a pathologist for St. Joseph's Hospital in Saint Paul for several years and became a member of its staff.

Dr. Ferguson served with the army during World War I, both in this country and overseas. He was a Life Member of the Ramsey County Medical Society, the Minnesota State Medical association, and a member of the American Medical Association. He was a charter member of the Minnesota Pathological Society and a member of the Association of Military Surgeons. In 1951, Dr. Ferguson became a member of the "Fifty Club" of the Minnesota State Medical Association.

He was active in Masonic affairs and was a life member of the Minnesota Historical Society.

He is survived by his wife, Emily K.; three sons, James Cory of Lake Minnetonka and Robert Bruce and William Frederick of Saint Paul, and six grandchildren.

A sample survey of some 12,000 industrial firms, including manufacturers of chemicals and professional and scientific equipment, has been started by the National Science Foundation. Director Alan T. Waterman said the findings should be valuable to companies in planning and appraising their own research programs, "besides contributing greatly to the formulation of national policies for research." He estimated industry's contribution to the total $3.5 billion national research and development effort at around $1 billion a year. The federal government, in addition, spends about $1 billion annually to support research and development in industrial laboratories. Survey plans were worked out in consultation with National Association of Manufacturers, Industrial Research Institute and other industry groups.

Of General Interest

Dr. Karl Lundeberg, Minneapolis health commissioner, has announced that Minneapolis ranks lowest in cities of 400,000 or more for reported cases of venereal disease in 1954. Minneapolis had a rate of 18 cases of syphilis per 100,000 population. The highest rate in the nation, reported in Washington, D. C., was 517 cases per 100,000. Similarly, Minneapolis had only 84 cases of gonorrhea per 100,000 population, compared to 1,400 per 100,000 in Washington.

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Among the speakers at a meeting of the fifth district of the Minnesota Society of X-Ray Technicians at Mankato on March 13 was **Dr. J. W. Burnett** of New Ulm. Dr. Burnett spoke on "Urinary Tract Examination."

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Dr. Henry W. Meyerding, Rochester, who retired in November, 1949, as professor of orthopedic surgery in the Mayo Foundation, has been elected an honorary member of the Turkish Surgical Association and an honorary fellow of the Philippine College of Surgeons.

Dr. Meyerding recently completed an around-the-world tour by airplane. Countries he visited included Hawaii, the Philippines, Japan, China, India, Thailand, Pakistan, Lebanon, Turkey, Israel, Greece, Italy, Iceland and France. Chapters of the International College of Surgeons were installed and lectures were given at various universities and surgical meetings.

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Dr. Henry E. Michelson, Minneapolis, has been invited to address the 1955 annual session of the Michigan State Medical Society in Grand Rapids, Michigan, on the subject, "Dermatological Diagnosis and Treatment."

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Dr. Wendell L. Downing, Le Mars, Iowa, a graduate of the University of Minnesota Medical School in 1919, has been named president-elect of the Iowa State Medical Society. Dr. Downing has been a member of the organization's board of trustees and was chairman of it last year. In February he was elected president of the Sioux Valley Medical Society.

* * *

Principal speaker at a clinical conference at the Community Memorial Hospital, New Prague, on March 15 was **Dr. Stuart W. Arhelger,** assistant professor in the department of surgery at the Mayo Memorial Hospital, University of Minnesota. He discussed the diagnosis and treatment of cancer. The clinical conference was one of a series sponsored by the Minnesota Department of Health, Minnesota State Medical Association, University of Minnesota, Minnesota Heart Association and Minnesota Cancer Society.

Guest consultant at a clinical conference at the Kanabec Hospital, Mora, late in March was **Dr. Paul Winchell,** staff member of the Variety Club Heart Hospital at the University of Minnesota. He discussed diseases of the heart.

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Dr. H. H. Russ, Blue Earth, was guest speaker at a meeting of the Kiester Parent-Teacher Association at Kiester late in March. He spoke on cerebral palsy and showed a motion picture on the same subject.

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Dr. Tray G. Rollins, formerly of Elmore and now located at Cass Lake, has been awarded a fellowship in dermatology at the Mayo Clinic, Rochester, effective January 1, 1956.

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Dr. E. L. Penk, Springfield, has been appointed to a three-year term as Springfield's health officer. He replaces **Dr. W. G. Nuessle,** whose term of office has expired. Dr. Penk also heads the Springfield board of health.

* * *

Fathers and expectant fathers heard **Dr. Milton L. Kaiser,** New Ulm, discuss the anatomy and physiology of reproduction and the responsibilities of fathers at a parent education meeting in New Ulm late in March. Two motion pictures on the subject were also shown. The session was the last in a series of parenthood classes sponsored by the Union Hospital at New Ulm.

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Dr. Dale Cameron, medical director of the Minnesota Department of Public Welfare, was the principal speaker at a meeting of the Minneapolis and St. Paul sections of the National Council of Jewish Women in St. Paul on April 4. Dr. Cameron spoke on mental health problems in Minnesota and the work of volunteers in mental health programs.

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Dr. S. J. Raetz, Maple Lake, attended a meeting of the International Academy of Proctology in New York City during the last week of March. While at the meeting he also attended lectures and surgical clinics at the Jersey City Medical Center.

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Childhood accidents were discussed by **Dr. Thomas Murn,** Thief River Falls, at a meeting of the Oklee Parent-Teacher Association in Oklee late in March.

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Dr. E. H. Rynearson, Rochester, has been named by the board of managers of the Gamehaven Foundation as general chairman of the Camp Building Fund Campaign, a drive to raise funds for improvement of scout camps near Rochester and Lake City.

Dr. G. W. Clifford, Alexandria, was guest speaker at a meeting of the Woman's Club in Alexandria on April 4. Dr. Clifford spoke on the subject, "Understanding Old Folks."

* * *

Dr. and Mrs. Robert N. Bowers, Lake City, spent an eleven-day vacation trip in Haiti, Jamaica and Cuba during the middle of March. They made the journey by air. While in Cuba they were entertained by friends, Dr. and Mrs. Carlos Menendez of Havana.

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An auxiliary of Doctors Memorial Hospital, Minneapolis, has been organized by **Dr. Olga S. Hansen** to assist the hospital and its patients.

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The engagement of Suzanne Elizabeth Scott, St. Paul, and **Dr. Thomas H. Kirschbaum**, Minneapolis, has been announced. A graduate of the University of Minnesota Medical School, Dr. Kirschbaum interned at University Hospitals. At present he is on active duty with the Navy at San Diego.

* * *

Principal speaker at a meeting of the Kiwanis Club at Alexandria late in March was **Dr. L. F. Wasson** of Alexandria, who spoke on the topic, "Does Cigarette Smoking Cause Cancer?" He discussed the opinions of groups on both sides of the somewhat controversial question.

* * *

Fifty-four members of the Chicago Surgical Society visited Rochester on March 25 and 26 for scientific sessions, observations of operations and tours of the Medical Sciences Building. **Dr. John M. Waugh** of the Mayo Clinic was in charge of the two-day affair. During the scientific sessions papers were presented by fellows in the Mayo Foundation and by members of the clinic staff.

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Dr. A. O. Swenson, Duluth, president of the Minnesota State Medical Association, has been named a member of the advisory board of the Minnesota Poll, a public opinion survey conducted by the Minneapolis *Tribune* as a public service.

* * *

Dr. and Mrs. S. W. Harrington, Rochester, returned home in mid-March after spending almost two months in South America. During their tour Dr. Harrington, a retired staff member of the Mayo Clinic, presented lectures at several colleges and universities. He received an honorary award from the University of Buenos Aires.

* * *

June 26 has been designated as Dr. McKaig Day for the residents of Pine Island and surrounding communities. On that date they will celebrate the birthday of **Dr. Carle B. McKaig** and honor him for his half-century of medical service. Committees have been formed, and the citizens of Pine Island are busily preparing for the event.

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Dr. C. E. Anderson, who became resident physician at Stillwater Prison on February 15, has resigned from the post because of illness.

* * *

Dr. and Mrs. O. K. Behr and their two sons have returned to Crookston after a six months' absence, during which time Dr. Behr had a senior residency in urology at the San Diego County Hospital in California. While Dr. Behr was in California his practice at Crookston was conducted by Dr. Norman R. Haugan, who is now in practice at Park Rapids.

* * *

Among the speakers at the Upper Midwest Business and Industrial Forum on Problem Drinking, held in Minneapolis on April 12, was **Dr. Charles W. Mayo**, Rochester. Dr. Mayo spoke on "People Who Overdrink." The meeting was conducted by the Upper Midwest Foundation on Problem Drinking, assisted by the Minneapolis Chamber of Commerce and the Salvation Army.

* * *

Dr. Donald E. Dille, Litchfield, left for Fort Sam Houston, San Antonio, Texas, on April 3 to report for a two-year tour of duty in the Army. Dr. Dille has been associated with the Litchfield Clinic since 1946. While he is in service his duties as Meeker County coroner will be carried out by **Dr. Harold Wilmot**, previously deputy coroner.

* * *

Two staff members of the University of Minnesota were the principal speakers at a one-day institute for private duty nurses held at the Midway YMCA, St. Paul, by the Minnesota Nurses Association on April 21. Speaking on the cross-circulation technique for intra-cardiac surgery was **Dr. Raymond Reed**, while **Dr. P. H. Soucheray** discussed the psychosomatic aspect of illness in older persons.

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A biographical sketch of **Dr. Samuel G. Balkin**, Minneapolis, appeared in the Town Toppers column in the Minneapolis *Star* on April 6. The article described Dr. Balkin's civic and professional activities. Town Toppers features brief biographies of leading citizens of Minneapolis each day.

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The Minnesota department of the Veterans of Foreign Wars recently presented the University of Minnesota with a certificate announcing that the organization had raised over $100,000 in its campaign to get $450,000 for a cancer research institute to be built at the University. Accepting the certificate for the University were J. L. Morrill, president, and **Dr. Owen H. Wangensteen**, chairman of the department of surgery. The VFW group voted at its annual meeting in 1953 to raise the money needed to build an eleven-bed nine-laboratory center without public solicitations for donations. It passed the $100,000 mark in less than eighteen months.

* * *

Dr. Kenneth H. Peterson, Hutchinson, left for Fort Sam Houston, Texas, on April 3 to report for a two-year tour of duty in the Army. He holds the rank of captain in the reserve. Dr. Peterson has practiced at Hutchinson for the past ten years.

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Dr. E. V. Allen, Rochester, was chairman of a symposium on hypertension at a postgraduate course conducted by the American College of Physicians in New York City late in March.

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The prevention and treatment of ski injuries was the topic discussed by **Dr. Vernon D. E. Smith**, St. Paul, at a meeting of the American College of Surgeons at Sun Valley, Idaho, April 18 to 20.

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Dr. R. B. J. Schoch, St. Paul health officer, was guest on "The Doctors' Round Table," a television show on WCCO-TV, on the last Sunday in March. Dr. Schoch discussed a case study made in St. Paul on the spread of tuberculosis in a family.

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Dr. Donald R. McFarlane, associated with the Oliver Clinic in Graceville, reported at Fort Sam Houston, Texas, on May 8 to put in a two-year tour of duty in the Army.

* * *

Dr. Thaddeus J. Litzow, Rochester, has been appointed to the staff of the Mayo Clinic as a consultant in plastic surgery and laryngology. Dr. Litzow entered the Mayo Foundation in 1951 and was an assistant to the staff from 1953 to April, 1954.

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Among Minnesota physicians attending the seventh annual scientific assembly of the American Academy of General Practice at Los Angeles, California, during the last week of March were the following: **Dr. J. A. Cosgriff, Sr.**, Olivia; **Dr. H. L. Huffington**, Waterville; **Dr. O. B. Fesenmaier**, New Ulm; **Dr. C. A. Wilmot**, Litchfield; **Dr. William E. Johnson**, Morgan; **Dr. H. E. Drill**, Hopkins; **Dr. B. F. Pearson**, Shakopee; **Dr. E. J. Tanquist**, Alexandria, and **Dr. E. W. Lippmann**, Hutchinson.

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Winner of second place by popular ballot at the 1955 Rural Art Show in the Agricultural Library at the University campus, St. Paul, was a painting by **Dr. Paul F. Brabec**, Hastings. Dr. Brabec's painting, entitled "Captain Bill," was entered in competition among painters from towns of under 10,000 population. Over 400 entries were received for the show, which was attended by 2,000 visitors.

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Dr. T. M. Seery, Austin, attended a series of lectures on "Pathology and Pathologic Physiology in Internal Medicine" at the Frank E. Bunts Educational Institute in Cleveland late in March. The lectures were presented by the Cleveland Clinic Foundation.

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Dr. and Mrs. Frank E. Mork, Anoka, returned in mid-March from a trip through the South. During the journey Dr. Mork attended the annual meeting of the Graduate Medical Assembly in New Orleans, Louisiana.

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Several section changes at the Mayo Clinic, Rochester, became effective April 1. **Dr. R. K. Ghormley** became senior consultant in orthopedic surgery, with **Dr. H. H. Young** as chairman of section. **Dr. F. A. Figi** became senior consultant in the section of plastic surgery, with **Dr. J. B. Erich** as head of section. **Dr. W. McK. Craig** became senior consultant in the section of neurosurgery, with **Dr. J. Grafton Love** as head of section.

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Dr. R. H. Puumala attended a three-day course in radiology at the University of Minnesota Continuation Center on April 11, 12 and 13.

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Dr. Douglas L. Johnson, Little Falls, began a fellowship in ophthalmology at the University of Minnesota on March 28. On completion of the graduate study he plans to return to Little Falls and limit his practice to ophthalmology.

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Principal speaker at a meeting of the Lions Club in Mabel late in March was **Dr. Joseph F. Schaefer**, Owatonna. The meeting was held in honor of the local Boy Scouts.

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Three Minnesota surgeons participated in a sectional meeting of the American College of Surgeons at Winnipeg, Canada, April 25 and 26. They were **Dr. Morley Cohen** and **Dr. W. Robert Schmidt**, Minneapolis, and **Dr. John C. Ivins**, Rochester.

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Guest consultant at a clinical conference at the Municipal Hospital at Wells on March 28 was **Dr. Alan P. Rusterholz** of the department of medicine at the University of Minnesota. He discussed heart disease at the conference, which was sponsored by several Minnesota medical organizations.

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Dr. and Mrs. S. T. Kucera, Northfield, returned in late March after a three-week tour of the southern states. While on the trip Dr. Kucera attended the eighteenth annual meeting of the New Orleans Graduate Assembly.

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Dr. Thomas L. Pool, Rochester, associate professor of urology in the Mayo Foundation, was a consultant in medicine at a Career Conference at Knox College, Galesburg, Illinois, recently. Experts from twenty-six vocational fields attended the conference for a day of counseling and meeting with students.

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A biographical sketch of **Dr. Ivar Sivertsen,** Minneapolis, in the Town Toppers column of the Minneapolis *Star* on April 14 emphasized the physician's early affiliation with Fairview Hospital. Dr. Sivertsen admitted the first patient to the hospital in 1916 and performed the first operation in the hospital's old operating suite.

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Dr. Jack A. Guy, New London, now with the U. S. Public Health Service, expects to return to his practice at New London about June 1. He recently completed a two-week course in fractures and traumatic surgery at the Cook County Postgraduate School of Medicine in Chicago.

* * *

Dr. and Mrs. C. J. Olson, Belle Plaine, returned on April 7 from a vacation trip to California and Texas. While in San Diego, Dr. Olson attended an institute in heart disease at the naval hospital.

NEW LOCATIONS

Dr. R. G. Ziegler has announced that he will discontinue his practice at Welcome about May 22 and will move to Brush, Colorado. Beginning July 1, he will be affiliated there with the Brush Clinic. He has practiced at Welcome since 1951.

* * *

Dr. John W. Schut opened offices for the practice of medicine in Anoka on April 1. A graduate of Northwestern University Medical School, he was a resident at the University of Minnesota for two years and served as a psychiatrist at the Anoka State Hospital for another two years. Until recently he was doing research at the Galesburg State Research Hospital, Galesburg, Illinois.

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Dr. Wallace E. Anderson has announced that he will leave Lakeville in July to become associated in practice with **Dr. Stanley Stone,** Minneapolis. Dr. Anderson has practiced at Lakeville since July, 1952.

* * *

Dr. Norman Haugan, formerly of Crookston, opened offices for the practice of medicine in Park Rapids on April 1. After graduating from the University of Minnesota Medical School in 1953, Dr. Haugan practiced in Watertown before moving to Crookston where he was associated with the Crookston Clinic.

MINNESOTA BLUE SHIELD-BLUE CROSS PLANS

Blue Shield

At a Blue Shield professional relations meeting held in Chicago on February 24 and 25, 1955, over 100 representatives of Blue Shield Plans throughout the country were present to review and evaluate methods used in professional relations programs. Arthur J. Doherty, of Minnesota Blue Shield, found that the whole program was devoted to consideration of means of providing greater service to the doctor, bringing information about Blue Shield directly to the doctor, and creating a closer relationship between the doctors and the Blue Shield Plans.

At the conclusion of these sessions, it was the opinion of those present that developing closer liaison between the doctor and Blue Shield through personal visits is the most effective feature of any such program. This method of improving professional relations is being used by many of the Blue Shield Plans in other states and has proved successful in securing the necessary close relationship and understanding between the doctor and Blue Shield.

Minnesota's Blue Shield program of professional relations is strikingly similar to that carried on in other states. In October, 1954, the Minnesota program became fully active. Since then, three Blue Shield field representatives have made over 1800 personal calls to doctors' offices. In more than 25 per cent of these calls, the doctor has been personally visited. When the doctor has not been available, various features of Blue Shield have been discussed with the members of his office staff. In each instance, the Blue Shield contract and procedures are explained to the doctor and his staff for clarification of Blue Shield objectives of subscriber benefits, claims reporting, and payment of claims.

During the first two months of 1955, maternity cases ranked first in frequency of occurrence for hospitalization of Blue Cross subscribers, respiratory illnesses ranked second and accidents third.

Blue Cross

The number of maternity cases paid during the first two months of this year totaled 4,302, a slight increase over the 4,277 cases paid during the same period of 1954. Maternity benefits of $463,106.08 were provided by Blue Cross during the first two months of 1955.

Although respiratory illnesses ranked second in frequency of occurrence during the first two months there was a slight decrease in the number of cases over the previous year. There were 3,857 respiratory cases for the first two months of 1955 with Blue Cross respiratory benefits totaling $309,102.19.

Accident cases numbered 3,599 for the first two months of 1955 and the Blue Cross accident benefits allowance amounted to $190,888.17.

Since 1933, $113,979,811.15 has been provided by Minnesota Blue Cross for hospital care of subscribers.

During the month of February, 1955, 4,056 contracts representing 10,171 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of February 28, 1955 to 1,003,104 participant members.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

CLINICAL DISORDERS OF HYDRATION AND ACID-BASE EQUILIBRIUM. Louis G. Welt, M.D. Professor of Medicine, Department of Medicine, University of North Carolina. 262 pages. Illus. Price $6.00, cloth. Boston: Little, Brown & Co., 1955.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY. 12th Edition. Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.R.S.E., Emeritus Surgeon, Royal Northern Hospital, London; General Surgeon, Metropolitan Ear, Nose and Throat Hospital; Consulting Surgeon, Italian Hospital; Hunterian Professor, Royal College of Surgeons; formerly External Examiner in Surgery, University of Bristol. 456 pages. Illus. Price $8.00, cloth. Baltimore: William & Wilkins, 1954.

BRUCELLOSIS. M. Ruiz Casteñeda. Director del Departmento de Investigaciones Médicas, Hospital General, México, D. F. Miembro del Comité de Expertos en Brucelosis de la Organizatión Mundial de la Salud. 302 pages. Illus. Mexico, D. F.: La Prensa Medica Mexicana, 1954.

THE TEMPOROMANDIBULAR JOINT. Edited by Bernard G. Sarnat, M.S., M.D., D.D.S., F.A.C.S., Professor and Head of the Department of Oral and and Maxillofacial Surgery, College of Dentistry; Clinical Assistant Professor, Department of Surgery (Plastic Surgery), College of Medicine, University of Illinois, Chicago, Ill.; Diplomate of the American Board of Plastic Surgery. 148 Pages. Illus. Price $4.75. Springfield, Ill.: Charles C Thomas, 1951.

This small volume by five faculty members of the dental schools of three universities in Chicago discusses the anatomy, physiology, and pathology of the temporomandibular joint with emphasis on the dental aspects. The Costen syndrome, attributing numerous auditory and vestibular symptoms to dysfunction of the temporomandibular joints, is thoroughly evaluated with the conclusion that it is not supported by facts and should be abandoned. The book is strictly limited in its objectives and makes no pretense at covering all the clinical features of the temporomandibular joints.

Elmer C. Paulson, M.D.

BONE TUMORS. By Louis Lichtenstein, M.D., Senior Pathologist, General Medical and Surgical Hospital, Veterans Administration Center, Los Angeles. 315 pages. Illus. Price $10.50. St. Louis: The C. V. Mosby Company, 1952.

The introductory chapter stresses the importance of biopsy in conjunction with roentgenographic examination of suspected bone tumors before treatment is attempted. Since many new skeletal lesions, both neoplastic and non-neoplastic, have recently been described, the necessity for open-mindedness is emphasized.

A classification of bone tumors is presented for consideration as a helpful working hypothesis, along with pertinent comment in support of proposed categories.

The chapters on bone tumors describe the clinical features, pathology, radiograms and therapy of various bone tumors. An appendix discusses some lesions of bone often mistaken for tumors.

The text is adequate and readable. The illustrations include radiograms, gross photographs, and photomicrographs of good quality. There is an adequate bibliography with each chapter. This book will prove itself useful to pathologists, surgeons, and radiologists alike.

J. W. James, M.D.

PRIMER OF ALLERGY; A GUIDEBOOK FOR THOSE WHO MUST FIND THEIR WAY THROUGH THE MAZES OF THIS STRANGE AND TANTALIZING STATE. By Warren T. Vaughan, M.S., M.D., Richmond, Va. Illustrations by John P. Tillery, 4th edition, revised by J. Harvey Black, M.D., Dallas, Texas. 191 pages. Price $4.25. St. Louis: C. V. Mosby Co., 1954.

This book has fulfilled its primary purpose. It is an interesting book on allergy which will be read by the intelligent patient and enable him to co-operate with his doctor. It is written in a style which stimulates the patient to do his best in treating his allergies. Every practicing physician will do the patient and himself a favor by introducing the patient with an allergic disease to this short book. The language of the doctor will no longer be just a lot of words after the patient has read this book.

The secondary purpose of this book, that of giving concise information to the physician doing a minimum of allergy work, has also been fulfilled. However, this reading should be expanded by reading a larger book in the field of allergy as soon as the physician's time permits.

Donald W. Koza, M.D.

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PUBLIC POLICY COMMITTEE

L. L. Sogge, M.D.	Windom
G. I. Badeaux, M.D.	Brainerd
J. A. Bargen, M.D.	Rochester
James A. Blake, Jr., M.D.	Hopkins
V. M. Johnson, M.D.	Dawson
E. J. Kaufman, M.D.	Appleton
M. E. L. Lenander, M.D.	St. Peter
J. N. Libert, M.D.	St. Cloud
J. A. Malmstrom, M.D.	Virginia
A. E. Ritt, M.D.	St. Paul
George Sather, M.D.	Fosston

RURAL MEDICAL SERVICE

First District

Paul C. Leck, M.D., *Chairman*	Austin

Second District

V. W. Doman, M.D.	Lakefield

Third District

Magnus Westby, M.D.	Madison

Fourth District

Lawrence Sjostrom, M.D.	St. Peter

Fifth District

A. K. Stratte, M.D.	Pine City

Sixth District

W. W. Rieke, M.D.	Wayzata

Seventh District

D. L. Johnson, M.D.	Little Falls

Eighth District

C. W. Jacobson, M.D.	Breckenridge

Ninth District

J. K. Butler, M.D.	Cloquet

RURAL MEDICAL SCHOLARSHIP COMMITTEE

F. J. Elias, M.D.	Duluth
C. G. Sheppard, M.D.	Hutchinson
E. J. Simons, M.D.	Saint Paul

UNIVERSITY RELATIONS

Justus Ohage, M.D.	Saint Paul
O. J. Campbell, M.D.	Minneapolis
Chas. C. Cooper, M.D.	Saint Paul
F. J. Elias, M.D.	Duluth
R. L. J. Kennedy, M.D.	Rochester

VETERANS AFFAIRS

Ralph H. Creighton, M.D.	Minneapolis
Alvin Erickson, M.D.	Long Prairie
J. J. Eustermann, M.D.	Mankato
J. E. Murphy, M.D.	Marshall
E. I. Parson, M.D.	Duluth
Alvan Sach-Rowitz, M.D.	Moose Lake
P. H. Soucheray, M.D.	St. Paul
C. H. Watkins, M.D.	Rochester

Councilor Districts

FIRST DISTRICT

J. M. Stickney, Jr., M.D.……Rochester
Counties—Dodge, Fillmore, Goodhue, Houston, Mower, Olmsted, Rice, Steele, Wabasha, Winona

SECOND DISTRICT

R. C. Hunt, Sr., M.D.……Fairmont
Counties—Cottonwood, Faribault, Freeborn, Jackson, Martin, Murray, Nobles, Pipestone, Rock, Watonwan

THIRD DISTRICT

P. E. Hermanson, M.D.……Hendricks
Counties—Big Stone, Chippewa, Kandiyohi, Lac Qui Parle, Lincoln, Lyon, Meeker, Pope, Redwood, Renville, Stevens, Swift, Traverse, Yellow Medicine

FOURTH DISTRICT

H. J. Nilson, M.D.……North Mankato
Counties—Blue Earth, Brown, Carver, Le Sueur, McLeod, Nicollet, Scott, Sibley, Waseca

FIFTH DISTRICT

L. R. Critchfield, M.D.……Saint Paul
Counties—Anoka, Chisago, Dakota, Isanti, Kanabec, Mille Lacs, Pine, Ramsey, Sherburne, Washington

SIXTH DISTRICT

H. B. Sweetser, M.D.……Minneapolis
Counties—Hennepin, Wright

SEVENTH DISTRICT

W. W. Will, M.D.……Bertha
Counties—Aitkin, Beltrami, Benton, Cass, Clearwater, Crow Wing, Hubbard, Koochiching, Morrison, Stearns, Todd, Wadena

EIGHTH DISTRICT

C. L. Oppegaard, M.D.……Crookston
Counties—Becker, Clay, Douglas, Grant, Kittson, Lake of the Woods, Mahnomen, Marshall, Norman, Otter Tail, Pennington, Polk, Red Lake, Roseau, Wilkin

NINTH DISTRICT

Clarence Jacobson, M.D.……Chisholm
Counties—Carlton, Cook, Itasca, Lake, St. Louis

County Medical Advisory Committees

STATE-WIDE ADVISORY COMMITTEE TO DEPARTMENT OF SOCIAL WELFARE

Rolland H. Wilson, M.D., *Chairman*	Winona
Haddon Carryer, M.D	Rochester
O. M. Heiberg, M.D	Worthington
Douglas L. Johnson, M.D	Little Falls
Karl E. Johnson, M.D	Duluth
J. P. Medelman, M.D	Saint Paul
E. W. Lippmann, M.D	Hutchinson
C. W. Moberg, M.D	Detroit Lakes
I. L. Oliver, M.D	Graceville
F. R. Hirshfield, M.D	Minneapolis
Leo G. Rigler, M.D	Minneapolis

AITKIN COUNTY

F. C. Closuit	Aitkin
H. T. Petraborg	Aitkin

ANOKA COUNTY

R. J. Spurzem	Anoka
B. W. Bunker	Anoka
Melvin Vik	Anoka

BECKER COUNTY

H. C. Otto	Frazee
W. C. Dodds	Detroit Lakes
Arnold Larson	Detroit Lakes

BELTRAMI COUNTY

T. P. Groschupf	Bemidji
D. E. Hoganson	Bemidji
H. A. Palmer	Blackduck

BIG STONE COUNTY

I. L. Oliver	Graceville
Otto Bergan	Clinton
H. H. Hedemark	Ortonville

BLUE EARTH COUNTY

George E. Penn	Mankato
Hugh Morgan	Amboy
P. G. Hoeper	Mankato

BROWN COUNTY

C. A. Saffert	New Ulm
Albert Fritsche	New Ulm
E. J. Wohlrabe	Springfield
O. B. Fesenmaier	New Ulm
A. P. Goblirsch	Sleepy Eye

CARLTON COUNTY

J. K. Butler	Cloquet
R. M. Eppard	Cloquet
E. O. Hanson	Carlton

CARVER COUNTY

M. B. Hebeisen	Chaska
B. H. Simons	Chaska
R. E. Pogue	Watertown

CASS COUNTY

O. F. Ringle	Walker
C. H. Coombs	Cass Lake
A. T. Rozycki	Pine River

CHIPPEWA COUNTY

H. A. Roust	Montevideo
M. A. Burns	Milan
L. A. Krystosek	Clara City

CHISAGO COUNTY

J. E. Halpin	Rush City
A. L. Holmes	Rush City
R. G. Swenson	North Branch

CLAY COUNTY

V. D. Thysell	Hawley
Carl Simison	Barnesville
H. G. Rice	Moorhead

CLEARWATER COUNTY

L. J. Larson	Bagley
W. E. Anderson	Clearbrook

COOK COUNTY

W. R. Smith	Grand Marais

COTTONWOOD COUNTY

H. C. Stratte	Windom
E. S. Schutz	Mountain Lake
J. V. Carlson	Westbrook

CROW WING COUNTY

V. E. Quanstrom	Brainerd
G. E. Badeaux	Brainerd
J. B. Nixon	Crosby

DAKOTA COUNTY

Bernice Thoreson	South St. Paul
H. J. Just	Hastings
A. H. Field	Farmington

DODGE COUNTY

C. E. Bigelow	Dodge Center
D. E. Affeldt	Kasson
G. E. Olson	West Concord

DOUGLAS COUNTY

E. E. Emerson	Osakis
G. W. Clifford	Alexandria
E. R. Sather	Alexandria

FARIBAULT COUNTY

W. C. Chambers	Blue Earth
M. D. Cooper	Winnebago
Richard Virnig	Wells

FILLMORE COUNTY

H. M. Skaug	Chatfield
L. W. Clark	Spring Valley
J. P. Nehring	Preston

FREEBORN COUNTY

J. P. Person	Albert Lea
C. E. J. Nelson	Albert Lea
A. G. Sherman	Albert Lea
L. C. Barr	Albert Lea

GOODHUE COUNTY

W. W. Liffrig	Red Wing
R. B. Graves	Red Wing
G. F. Hartnagel	Red Wing

GRANT COUNTY

L. R. Parson	Elbow Lake
J. H. Cain	Hoffman

HENNEPIN COUNTY (RURAL)

W. W. Rieke	Wayzata
H. E. Drill	Hopkins
E. J. Lillehei	Robbinsdale

HOUSTON COUNTY

N. T. Norris	Caledonia
L. K. Onsgard	Houston
L. A. Knutson	Spring Grove

HUBBARD COUNTY

Donald Houston	Park Rapids
W. W. Higgs	Park Rapids
John Eiler	Park Rapids

ISANTI COUNTY

W. T. Nygren	Braham
R. J. Gully	Cambridge
G. E. Larson	Cambridge

ITASCA COUNTY

E. K. Rowles	Coleraine
G. M. Erskine	Grand Rapids
M. J. McKenna	Grand Rapids

JACKSON COUNTY

J. T. Rose	Lakefield
H. A. Christensen	Jackson
W. H. Halloran	Jackson

KANABEC COUNTY

C. S. Bossert	Mora
W. F. Nordman	Mora
Harry L. Berge	Mora

KANDIYOHI COUNTY

H. G. Bosland	Willmar
R. V. Hodapp	Willmar
J. C. Jacobs	Willmar

KITTSON COUNTY

A. S. Berlin	Hallock
R. B. Skogerboe	Karlstad

KOOCHICHING COUNTY

R. A. MacDonald	Littlefork
R. D. Hanover	International Falls
C. C. Craig	International Falls

LAC QUI PARLE COUNTY

V. T. Fallon	Dawson
Chester Anderson	Madison

LAKE COUNTY

Ralph Papermaster	Two Harbors

LAKE-OF-THE-WOODS

A. A. Brink	Baudette
A. G. Janecky	Baudette

LINCOLN COUNTY

P. E. Hermanson	Hendricks
A. L. Vadheim	Tyler
M. H. Larson	Lake Benton

LYON COUNTY

B. C. Ford	Marshall
W. W. Yaeger	Marshall
W. G. Workman	Tracy

MAHNOMEN COUNTY

K. W. Covey	Mahnomen
J. M. Gacusana	Mahnomen

MARSHALL COUNTY

Carle Holmstrom	Warren
E. E. Pumula	Warren

MARTIN COUNTY

R. C. Hunt	Fairmont
O. E. Wandke	Fairmont
C. W. Krause	Fairmont
H. A. Williamson	Fairmont

MCLEOD COUNTY

A. M. Jensen	Brownton
E. W. Lippman	Hutchinson
Joseph Selmo	Norwood

MEEKER COUNTY

Harold Wilmot	Litchfield
Lennox Danielson	Litchfield
David D. Allison	Litchfield

MILLE LACS COUNTY

W. R. Blomberg	Princeton
A. T. Kapsner	Princeton
N. B. Metcalf	Princeton

MORRISON COUNTY

D. L. Johnson	Little Falls
A. M. Watson	Royalton
E. G. Knight	Swanville

MOWER COUNTY

L. F. Twiggs	Austin
J. G. Havens	Austin

MURRAY COUNTY

B. M. Stevenson	Fulda
R. F. Pierson	Slayton
H. D. Patterson	Slayton

NICOLLET-LE SUEUR COUNTY

E. G. Olmanson	St. Peter
D. C. Olson	Gaylord
B. P. Grimes	St. Peter

NOBLES COUNTY

E. W. Arnold	Adrian
E. A. Kilbride	Worthington
C. R. Stanley	Worthington

NORMAN COUNTY

Eskil Erickson	Halstad
Byron Kinkade	Ada
Theodore Loken	Ada

OLMSTED COUNTY

H. F. Polley	Rochester
C. B. McKaig	Pine Island
T. O. Wellner	Rochester

OTTERTAIL COUNTY

Charles Lewis	Henning
Frank Naegeli	Fergus Falls
H. K. Helseth	Pelican Rapids

PENNINGTON COUNTY

M. D. Starekow	Thief River Falls
George T. Van Rooy	Thief River Falls

PINE COUNTY

A. D. Stratte	Pine City
E. G. Hubin	Sandstone
H. P. Dredge	Sandstone

PIPESTONE COUNTY

W. G. Benjamin	Pipestone
J. G. Lohmann	Pipestone
G. Beckering	Edgerton

POLK COUNTY

George Sather	Fosston
C. G. Uhley	Crookston
C. L. Roholt	McIntosh

POPE COUNTY

Paul Swedenberg	Glenwood
A. F. Giesen	Starbuck

RAMSEY COUNTY

D. M. Craig	St. Paul
M. L. Straus	St. Paul
A. E. Muller	No. St. Paul

RED LAKE COUNTY

Lester Dale	Red Lake Falls
Alan McKaig	Red Lake Falls

REDWOOD COUNTY

W. J. Alcorn	Wabasso
A. W. Diessner	Redwood Falls
W. E. Johnson	Morgan

RENVILLE COUNTY

J. A. Cosgriff, Sr.	Olivia
John Dordal	Sacred Heart
A. M. Fawcett	Renville

RICE COUNTY

A. W. Nuetzman	Faribault
Heinz H. Bruhl	Faribault
Bernard Street	Northfield

ROCK COUNTY

C. L. Sherman	Luverne
A. C. Martin	Luverne
F. W. Bofenkamp	Luverne

ROSEAU COUNTY

John L. Delmore, Jr.	Roseau
David O. Berge	Roseau
Kenneth Nelson	Warroad
Loyd Klefstad	Greenbush

ST. LOUIS COUNTY

C. M. Bagley	Duluth
Sam Boyer, Jr.	Duluth
P. F. Eckman	Duluth

SCOTT COUNTY

H. M. Juergens	Belle Plaine
B. F. Pearson	Shakopee

SHERBURNE COUNTY

A. B. Roehlke	Elk River
G. H. Tesch	Elk River

SIBLEY COUNTY

Rolf Hovde	Winthrop
R. H. Kath	Arlington
D. C. Olson	Gaylord

STEARNS-BENTON COUNTY

C. F. Brigham, Jr.	St. Cloud
J. B. Beuning	St. Cloud
R. N. Jones	St. Cloud
N. F. Musachio	Foley
K. A. Walfred	St. Cloud

STEELE COUNTY

A. J. Olson	Owatonna
J. A. McIntyre	Owatonna

STEVENS COUNTY

M. L. Ransom	Hancock
R. A. Rossberg	Morris
A. I. Arneson	Morris

SWIFT COUNTY

E. J. Kaufman	Appleton
B. C. Ostling	Kerkhoven
R. P. Griffin	Benson

TODD COUNTY

M. E. Mosby	Long Prairie
J. M. Cook	Staples
C. B. Will	Bertha

TRAVERSE COUNTY

A. L. Lindberg	Wheaton
A. E. Magnuson	Wheaton
W. F. Muir	Browns Valley

WABASHA COUNTY

C. G. Ochsner	Wabasha
E. C. Bayley	Lake City
D. G. Mahle	Plainview

WADENA COUNTY

L. T. Davis	Wadena
C. H. Pierce	Wadena
W. E. Parker	Sebeka

WASECA COUNTY

G. H. Olds	New Richland
B. J. Gallagher	Waseca

WASHINGTON COUNTY

M. Juergens	Stillwater
C. H. Sherman	Bayport
R. E. Carlson	Stillwater

WATONWAN COUNTY

O. B. Bergman	Saint James
Herbert Boysen	Madelia

WILKIN COUNTY

C. W. Jacobson	Breckenridge

WINONA COUNTY

R. H. Wilson	Winona

WRIGHT COUNTY

S. J. Raetz	Maple Lake
R. M. Sandeen	Buffalo

YELLOW MEDICINE COUNTY

Carl Lundell	Granite Falls
O. M. Odland	Granite Falls
E. R. Hudec	Echo

WOMAN'S AUXILIARY to the MINNESOTA STATE MEDICAL ASSOCIATION

Officers

Mrs. Peter S. Rudie	*President*	Duluth
Mrs. Henry W. Quist, Sr.	*Past President*	Minneapolis
Mrs. H. H. Fesler	*President-elect*	St. Paul
Mr.. John Dordal	*First Vice President*	Sacred Heart
Mrs. V. J. Schwartz	*Second Vice President*	Minneapolis
Mrs. L. P. Howell	*Third Vice President*	Rochester
Mrs. Justus Ohage	*Fourth Vice President*	St. Paul
Mrs. David Halpern	*Recording Secretary*	Brewster
Mrs. Charles W. Froats	*Treasurer*	St. Paul
Mrs. G. H. Goehrs	*Auditor*	St. Cloud
Mrs. S. N. Litman	*Corresponding Secretary*	Duluth
Mrs. A. J. Bianco	*Historian*	Duluth
Mrs. Leo Fink	*Parliamentarian*	Minneapolis

Regional Advisors

Mrs. W. E. Wellman	*First District*	Rochester
Mrs. O. M. Heiberg	*Second District*	Worthington
Mrs. E. R. Hudec	*Third District*	Echo
Mrs. T. R. Fritsche	*Fourth District*	New Ulm
Mrs. O. I. Sohlberg	*Fifth District*	Saint Paul
Mrs. Ralph Creighton	*Sixth District*	Minneapolis
Mrs. J. C. Buscher	*Seventh District*	Saint Cloud
Mrs. C. W. Moberg	*Eighth District*	Detroit Lakes
Mrs. J. L. McLeod	*Ninth District*	Grand Rapids

Chairmen of Committees

Advisory	Mrs. Henry W. Quist, Sr.	Minneapolis
Allied Medical Careers	Mrs. Stanley Peterson	Austin
American Medical Education Foundation Fund	Mrs. J. A. Cosgriff	Olivia
Archives	Mrs. J. L. Benepe	Saint Paul
Bulletin	Mrs. C. E. Carlson	Alexandria
Cancer	Mrs. W. G. Benjamin	Pipestone
Civilian Defense	Mrs. D. G. Mahle	Plainview
Editor (Minnesota Medicine)	Mrs. L. Raymond Scherer	Minneapolis
Finance	Mrs. Mark Ryan	Saint Paul
Health Days	Mrs. Harold F. Wahlquist	Minneapolis
In Memoriam	Mrs. John J. Ryan	Saint Paul
Legislation	Mrs. Philip K. Arzt	Saint Paul
Medical and Surgical Relief	Mrs. W. T. Greenfield	Cokato
Mental Health	Mrs. A. E. Lindblom	Mankato
News Letter	Mrs. M. F. Fellows	Duluth
Organization	Mrs. H. H. Fesler	Saint Paul
Press and Publicity	Mrs. A. B. Rosenfield	Minneapolis
Printing	Mrs. William Gjerde	Lake City
Program and Health Education	Mrs. C. E. Merkert	Minneapolis
Public Relations	Mrs. G. A. Hedberg	Nopeming
Resolutions	Mrs. P. J. Pankratz	Mountain Lake
Revisions	Mrs. D. V. Boardman	Winona
Roster	Mrs. R. F. Erickson	Minneapolis
School of Instruction	Mrs. Leonard Arling	Minneapolis
Today's Health	Mrs. M. I. Hauge	Clarkfield

County Society Roster

Key to Symbols: *Deceased, †Affiliate, Associate or Life Member; ‡In Service; §Wife is Member of Woman's Auxiliary.

BLUE EARTH COUNTY MEDICAL SOCIETY

Regular meetings, last Monday of each month.
Annual meeting, last Monday of December
Number of Members—57

President
EUSTERMANN, JOHN J. .... Mankato
Secretary
MCNEAR, GEORGE R., JR. .... Mankato

§ Anderson, James J. .... Mankato
Anderson, Margaret C. .... Mankato
†§Andrews, Roy N. .... Mankato
§ Baird, Raymond L. .... Lake Crystal
§ Batdorf, B. Niles .... Good Thunder
§ Butzer, John A. .... Mankato
§ Butzer, John F. .... Mankato
§ Chalgren, William S. .... Mankato
§ Conley, Robert H. .... Mankato
Dobson, M. W. .... Mankato
§ Engstrom, Robert .... Mankato
§ Eustermann, John J. .... Mankato
†§Franchere, Fredk. Wm. .... Lake Crystal
§ Fugina, George R. .... Mankato
§ Geurs, Benjamin R. .... Mankato
§ Haes, Julius E. .... Mankato
§ Hammar, Lawrence M. .... Mankato
Hankerson, Robert G. .... Minnesota Lake
† Hassett, Roger G. .... Mankato
§ Heller, Edgar E. .... Mankato
§ Hoeper, Philip G. .... Mankato
§ Howard, Marshall I. .... Mankato
† Huffington, Herbert L., Sr. .... Mankato
§ Huffington, Herbert L., Jr. .... Waterville
§ Jones, Orville H. .... Mankato
† Juliar, Richard O. .... Los Angeles, Calif.
§ Kaufman, Walter B. .... Mankato
§ Kearney, Rochfort Wynn .... Mankato
§ Kemp, Alphonse F. .... Mankato
§ Koenigsberger, Charles .... Mankato
§ Langhoff, Arthur H. .... Mankato
† Liedloff, Adolph G. .... Mankato
§ Lindblom, Alton E. .... No. Mankato
† Luck, Hilda .... Mankato
§ McNear, George R., Jr. .... Mankato
§ Mickelson, John C. .... Mankato
†§Miller, Victor I. .... Mankato
§ Morgan, Hugh O. .... Amboy
§ Olive, John T., Jr. .... Mankato
§ Penn, George E. .... Mankato
§ Roth, Frederick .... Mankato
§ Samuelson, L. Gordon .... Mankato
§ Sanford, Raymond A. .... Mankato
† Schmidt, Paul A. .... Aurora, Ill.
§ Schmitz, Anthony A. .... Mankato
§ Sjoding, J. Donald .... Mankato
§ Smith, Harry J. .... Lake Crystal
§ Smith, Paul M. .... Lake Crystal
† Sohmer, Alphonse E. .... Mankato
§ Stillwell, Walter C .... Mankato
§ Thiem, Chester E. .... Mankato
§ Troost, Henry B. .... Mankato
§ Vezina, John C. .... Mapleton
‡§Von Drasek, Joseph .... Mankato
†§Wentworth, Albert J. .... Mankato
† Williams, Hugh O. .... Lake Crystal
§ Wohlrabe, John C. .... St. Clair

BLUE EARTH VALLEY MEDICAL SOCIETY

(Martin—Watonwan—Faribault Counties)
Regular meetings, third Thursday of each month.
Annual meeting in November
Number of Members—44

President
ARMSTRONG, R. S. .... Winnebago
Secretary
BOYSEN, HERBERT .... Madelia

§ Armstrong, Ralph S. .... Winnebago
Barr, James S. .... Elmore
§ Bergman, Oscar B. .... St. James
§ Blumberg, Henry B. .... French Cp., Calif.
Boysen, Herbert .... Madelia
Bratrude, Earl J. .... St. James
†§Chambers, Winslow C. .... Blue Earth
† Cooper, Maurice D. .... Winnebago
§ Coulter, Harold E. .... Madelia
§ Drexler, George W. .... Blue Earth
§ Gamble, Elbert J. .... Bricelyn
§ Gardner, Victor H. .... Fairmont
§ Hanson, Lewis .... Frost
§ Heimark, Julius J. .... Fairmont
§ Hruza, William J. .... Madelia
†§Hunt, Roscoe C. .... Fairmont
Kraemer, George N. .... Fairmont
§ Krause, C. W. .... Fairmont
§ Lester, Malcolm J., Jr. .... Truman
§ Lindahl, Merlyn J. .... Sherburn
Louisell, Charles .... Fairmont
† McGroarty, John J. .... Easton
† Mills, John L. .... Winnebago
‡ Moulton, K. B. .... St. James
§ Nickerson, John R. .... Fairmont
§ Nickerson, Neil D. .... Fairmont
§ Ourada, Anthony L. .... Ceylon
§ Parsons, R. A. .... St. James
Parsons, Ralph L. .... Monterey
‡ Rollins, Troy G. .... Cass Lake
§ Rovelstad, Roger Andrew .... Fairmont
*†Rowe, William H. .... Fairmont
§ Russ, Homer H. .... Blue Earth
§ Smith, Don V. .... Blue Earth
§ Snyder, Clifford D. .... Kiester
§ Thayer, Ellsworth A. .... Fairmont
§ Vaughan, Victor M. .... Truman
§ Virnig, Mark P. .... Wells
§ Virnig, Richard P. .... Wells
§ Wandke, Otto E. .... Fairmont
§ Watkins, John A. .... Wells
§ Williamson, Harold A. .... Fairmont
† Wilson, Clyde E. .... Blue Earth
§ Zemke, Erhart E. .... Fairmont
Ziegler, Robert G. .... Welcome

BROWN COUNTY MEDICAL SOCIETY

Regular meetings, quarterly. Annual meeting, in January
Number of Members—34

President
SEIFERT, OTTO J. .... New Ulm
Secretary
KAISER, MILTON .... New Ulm

§ Black, William A. .... New Ulm
Burnett, Joseph W. .... New Ulm
§ Cairns, Robert J. .... Redwood Falls
Carthey, Frank J. .... New Ulm
§ Dubbe, Fredk. H. .... New Ulm
§ Dysterheft, Adolf F. .... Gaylord
§ Fesenmaier, Otto B. .... New Ulm
Flinn, James B. .... Redwood Falls
§ Fritsche, Albert .... New Ulm
§ Fritsche, Carl J. .... New Ulm
§ Fritsche, Theodore R. .... New Ulm
Goblirsch, Andrew P. .... Sleepy Eye
Hedlund, Charles J. .... Owatonna
Hovde, Rolf .... Winthrop
Inglis, William .... Redwood Falls
§ Kaiser, Milton L. .... New Ulm
§ Keithahn, Elmer E. .... Sleepy Eye
Kitzberger, Peter J. .... New Ulm
§ Kruzick, S. J. .... Sleepy Eye
†§Kusske, Arthur L. .... New Ulm
Mattson, Albert D. .... St. James
Muesing, William J. .... New Ulm
Nuessle, Walter G. .... Springfield
† O'Leary, John B. .... Minneapolis
§ Penk, Engward L. .... Springfield
Peterson, Roy A. .... Vesta
Rayner, Ralph R. .... Glencoe
†§Reineke, George F. .... New Ulm
§ Saffert, Cornelius A. .... New Ulm
Schroeppel, John E. .... Winthrop
§ Seifert, Otto J. .... New Ulm
§ Vogel, Howard A. L. .... New Ulm
Wisness, Osmund A. .... Comfrey
§ Wohlrabe, Edwin J. .... Springfield

CAMP RELEASE DISTRICT MEDICAL SOCIETY

(Chippewa, Lac qui Parle and Yellow Medicine Counties)
Regular meeting, first and third Thursday of April, May, September and October
Annual meeting, third Thursday in October
Number of Members—33

President
BARR, RONALD W. .... Montevideo
Secretary
ALLEN, JOHN H. .... Montevideo

§ Allen, John H. .... Montevideo
Anderson, Chester A. .... Madison
§ Barr, Ronald W. .... Montevideo
Boody, George, Jr. .... Sandstone
§ Burns, Floyd M. .... Milan
§ Burns, M. Alpheus .... Milan
§ Camp, Ray Junior .... Madison
Fallon, Virgil .... Dawson

‡ Flom, Robert S..........Columbus, Ga.
Guilbert, G. D..........Minneapolis
§ Hartfiel, Herbert A..........Montevideo
§ Hauge, Malvin I..........Clarkfield
Holmberg, LeRoy J..........Canby
§ Hudec, Elwyn R..........Echo
Hustad, Edward G..........Montevideo
‡ Johnson, Curtis M..........Dawson
§ Johnson, Vilhelm M..........Dawson

† Jordan, Kathleen Smith....Granite Falls
Jordan, Lewis S..........Granite Falls
§ Kaufman, William C..........Appleton
§ Krystosek, Lee A..........Clara City
† Lee, Walter N..........Claremont, Calif.
§ Lima, Ludvig R., Jr..........Montevideo
§ Lundell. Carl L..........Granite Falls
‡ Maus, Philip..........New Orleans, La.

Nelson, Melvin S..........Granite Falls
§ Odland, Olin M..........Granite Falls
§ Owens, William A..........Montevideo
Pertl, Albert L..........Canby
§ Roust, Henry A..........Montevideo
§ Schmidt, Paul G., Jr..........Granite Falls
* Smith, Leon G..........Montevideo
§ Westby, Magnus..........Madison
§ Westby, Norval M..........Madison

CLAY-BECKER COUNTY MEDICAL SOCIETY

Regular meeting, spring, fall and winter. Annual meeting, November 30

Number of Members—26

President
ODLAND, MARK..........Detroit Lakes
Secretary
DODDS, WILLIAM C..........Detroit Lakes
§ Berg, Arnold..........Hawley
§ Bigler, Earl E..........Perham
§ Bigler, Ivan E..........Perham
Bottolfson, Bottolf T..........Moorhead
§ Carlson, Vernon J..........Moorhead
§ Dodds, William C..........Detroit Lakes

§ Duncan, James W..........Moorhead
Gacusana, Jose M..........Mahnomen
§ Geib, Marvin J..........Moorhead
† Hagen, Olaf J..........Moorhead
§ Houglum, Arvid J..........Lake Park
§ Humphrey, Edward W..........Moorhead
Johnson, Olga H..........Moorhead
§ Larson, Arnold..........Detroit Lakes
§ Lorentzen, Ernest S..........Detroit Lakes
§ Midthune, A. S..........Lake Park

§ Moberg, Clarence W..........Detroit Lakes
§ Odland, Mark E..........Detroit Lakes
§ Oliver, James..........Moorhead
§ Otto, Henry C..........Frazee
§ Rice, Hagbart G..........Moorhead
§ Rutledge, Lloyd H..........Detroit Lakes
Saxman, Gertrude..........Georgetown
§ Simison, Carl..........Barnesville
† Thysell, Fred A..........Moorhead
§ Thysell, Vernon D..........Hawley

EAST CENTRAL MINNESOTA MEDICAL SOCIETY

(Anoka, Chisago, Isanti, Kanabec, Mille Lacs, Pine and Sherburne Counties)

Regular meetings, first Tuesday of every other month of the year
Annual meeting, first Tuesday in December

Number of Members—40

President
LARSON, GERALD E..........Cambridge
Secretary
TESCH, GORDON H..........Elk River
Adkins, Galen H..........Anoka
§ Albrecht, H. H..........Chisago City
‡ Baars, Conrad W.....Camp Cook, Calif.
§ Berge, Harry L..........Mora
‡ Beyer, Eugene F.....San Francisco, Calif.
§ Blomberg, Wm. R..........Princeton
† Bossert, Clarence S..........Mora
§ Bunker, Bevan W..........Anoka
§ Burseth, Edgar C..........Mora
† Dredge, Homer P..........Sandstone
§ Gully, Raymond J..........Cambridge

Halpin, Joseph E..........Rush City
Hedenstrom, Paul H..........Cambridge
‡ Henry, Harold W..........Benton, Ill.
Holmes, Alva E..........Rush City
§ Hubin, Edwin G..........Sandstone
Johnson, Aldridge F..........Isle
§ Kapsner, Alfred T..........Princeton
† Kelsey, Carleton G..........St. Paul
Kragh, Lyle O..........Braham
§ Larson, Gerald E..........Cambridge
‡ Lorton, Wm. L..........Rochester
§ McManus, William F..........Princeton
‡ Magnuson, R. C..........Cambridge
§ March, Kenneth A..........Cambridge
Metcalf, Norman B..........Princeton

§ Nordman, Willard F..........Mora
Nygren, William T..........Braham
Pasek, Edward A..........Minneapolis
Pone, John..........Cambridge
§ Roehlke, Arthur B..........Elk River
Rudolph, Frank A..........Sandstone
*†§Sherman, Hubert T..........Cambridge
Spurzem, Raymond J..........Anoka
‡ Stahn, Louis H..........El Paso, Texas
§ Stratte, Alf K..........Pine City
§ Swensen, Roy G...........North Branch
§ Tesch, Gordon H..........Elk River
Vik, Melvin..........Anoka
† Waller, Joseph D..........Pine City
Woyda, William C..........Elk River

FREEBORN COUNTY MEDICAL SOCIETY

Regular meetings, third Thursday of even months
Annual meeting, December

Number of Members—30

President
EGGE, S. G..........Albert Lea
Secretary
ELLERTSON, L. M..........Albert Lea
§ Barr, Lowell C..........Albert Lea
Burns, Catherine..........Albert Lea
§ Butturff, Carl R..........Freeborn
†§Calhoun, F. W..........Albert Lea
§ Demo, Robert A..........Albert Lea
§ Donovan, Daniel L..........Albert Lea
§ Egge, Sanford G..........Albert Lea
§ Ellertson, Leonard M..........Albert Lea

§ Erdal, Ove A..........Albert Lea
§ Folken, Frank G..........Albert Lea
† Freeman, John P..........Glenville
† Gullickson, Andrew.....Longmont, Colo.
§ Hansen, Theodore M..........Albert Lea
§ Holian, Darwin K..........Albert Lea
* Kaasa, Lawrence J..........Albert Lea
*†Kamp, Byron A..........Albert Lea
§ Keil, Marcus A..........Albert Lea
† Leopard, Brand A.....Brownsville, Texas
§ Neel, Harry B..........Albert Lea

§ Nelson, Clayton E..........Albert Lea
§ Nesheim, Martin O..........Emmons
§ Palmer, Clinton F..........Albert Lea
§ Person, John P..........Albert Lea
§ Prins, Leo R..........Albert Lea
Ryding, Vincent..........Albert Lea
§ Schmidt, Ruben F..........Alden
†§Schultz, J. Albert..........Albert Lea
§ Sherman, Alfred G..........Albert Lea
†§Steiner, Leon E..........Philadelphia, Pa.
§ Whitson, Sidney A..........Albert Lea
§ Wilcox, G. Charles..........Albert Lea

GOODHUE COUNTY MEDICAL SOCIETY

Regular meeting, none
Annual meeting, December

Number of Members—29

President
SHERMAN, ROYAL V..........Red Wing
Secretary
HAWLEY, GEORGE M. B..........Red Wing
§ Akins, Willard M..........Red Wing
§ Allen, George S..........Cannon Falls
‡ Bagby, George W...Ft. Len. Wood, Mo.
‡ Boswell, J. Thornton....Portsmouth, Va.
§ Bridge, Ezra V..........Cannon Falls
§ Brusegard, James F..........Red Wing
Claydon, Howard F..........Red Wing

§ DeGeest, James H..........Goodhue
‡ Dovenmuehle, Robt. H...Durham, N. C.
§ Falls, John L..........Red Wing
Flom, Martin G..........Moorhead
§ Graves, Richard B..........Red Wing
§ Halvorson, James W..........Zumbrota
§ Hartnagel, G. F..........Red Wing
§ Hawley, George M. B..........Red Wing
§ Hedin, Raymond F..........Red Wing
† Jones, Alvah W..........Red Wing
§ Juers, Edward H..........Red Wing

§ Kimmel, George C..........Red Wing
§ Liffrig, William W..........Red Wing
§ Miller, Winston R..........Red Wing
§ Molenaar, Robert E..........Cannon Falls
§ Sherman, Royal V..........Red Wing
† Smith, Myron W..........Red Wing
§ Steffens, Leon A..........Red Wing
§ Walter, William E..........Wanamingo
§ Wasmund, Clarence W..........Red Wing
Weiss, Carl A..........Hastings
§ Williams, Marland R..........Cannon Falls

HENNEPIN COUNTY MEDICAL SOCIETY

Regular meetings, first Monday of each month October through May
Election in February
Annual meeting, first Monday in October
Number of Members—960

President
ALING, CHARLES A. Minneapolis
Secretary
HOLMBERG, CONRAD J. Minneapolis
Executive Secretary
COOK, THOMAS P. Minneapolis

Abramson, Milton	Minneapolis
Adkins, Charles D.	Minneapolis
§ Agustsson, Hreidar	Minneapolis
§ Ahern, Eugene E.	Minneapolis
§ Alexander, Harlan A.	Minneapolis
§ Aling, Charles A.	Minneapolis
Althausen, Theo. L., Jr.	Minneapolis
† Altnow, Hugo O.	Coral Gables, Fla.
Amatuzio, Donald S.	Minneapolis
§ Andersen, Silas C.	Minneapolis
Anderson, Arnold S.	St. Louis Park
§ Anderson, David M.	Minneapolis
† Anderson, Edward D.	Minneapolis
§ Anderson, Ernest R.	Minneapolis
§ Anderson, Frank J.	Minneapolis
§ Anderson, John T.	Minneapolis
§ Anderson, Karl W.	Minneapolis
Anderson, Richard W.	Minneapolis
§ Anderson, Roger L.	Minneapolis
§ Anderson, U. Schuyler	Minneapolis
Anderson, Wallace E.	Lakeville
§ Anderson, William H.	Minneapolis
Anderson, William T.	Minneapolis
Andreassen, Einar C.	St. Paul
§ Andreassen, Rolf L.	Minneapolis
§ Andresen, Karl D'A.	Minneapolis
§ Andrews, Robert S.	Minneapolis
§ Ankner, Frank J.	Minneapolis
* Archer, Willard E.	Anoka
Arends, Archabald L.	Minneapolis
§ Arey, S. Lane	Minneapolis
Arhelger, Stuart W.	Minneapolis
§ Arlander, Clarence E.	Minneapolis
§ Arling, Leonard S.	Minneapolis
§ Arms, James J.	Minneapolis
Arnold, Anna W.	Minneapolis
Arvidson, Carl G.	Minneapolis
†§Aune, Martin	Minneapolis
† Aurand, William H.	Minneapolis
§ Baggenstoss, Osmond J.	Minneapolis
Bagley, Russell W.	Minneapolis
§ Baird, Joseph W.	Minneapolis
§ Baken, Melvin P.	Minneapolis
Baker, A. B.	Minneapolis
Baker, Alfred T.	Minneapolis
*†Baker, Looe	Minneapolis
§ Baker, Milton E.	Minneapolis
§ Baleisis, Peter	Minneapolis
§ Balkin, Samuel G.	Minneapolis
Balogh, Charles J.	Minneapolis
Bank, Harry E.	San Francisco, Calif.
§ Barno, Alex	St. Louis Park
§ Barr, Maxwell	Minneapolis
Barr, Robert N.	Minneapolis
Barron, Jesse J.	Minneapolis
†§Barron, Moses	Minneapolis
‡ Barron, S. Steven	Minneapolis
†§Baxter, Stephen H.	Minneapolis
§ Beach, Northrop	Minneapolis
† Becker, Arnetta	Minneapolis
§ Bedford, Edgar W.	Minneapolis
§ Beirstein, Samuel	Minneapolis
§ Beiswanger, Richard H.	Minneapolis
† Bell, E. T.	Minneapolis
§ Bellville, Titus P.	Minneapolis
§ Belzer, Meyer S.	Minneapolis
Benesh, Louis A.	Minneapolis
§ Benjamin, Edwin G.	Minneapolis
§ Benjamin, Harold G.	Minneapolis
Benson, Ellis S.	Minneapolis
Berg, Clinton C.	Excelsior
Berger, Alex G.	Minneapolis
§ Bergh, George S.	Minneapolis
Bergh, Solveig M.	Minneapolis
Berglund, Eldon B.	Minneapolis
Bergquist, James R.	Minneapolis
§ Berkwitz, Nathaniel J.	Minneapolis
Berman, Reuben	Minneapolis
Bernstein, Irving C.	Minneapolis
§ Bessesen, Alfred N., Jr.	Minneapolis
*†Bessesen, William A.	Minneapolis
Bieter, Raymond N.	Minneapolis
§ Bilka, Paul J.	Minneapolis
§ Binder, Manuel R.	Minneapolis
Bjornson, Robert G. B.	Minneapolis
§ Blake, Allen J.	Hopkins
§ Blake, James A.	Hopkins
§ Blake, Paul S.	Hopkins
§ Bloedel, T. J. G.	Osseo
Blomberg, Robert D.	Minneapolis
§ Bloom, Norman B.	Minneapolis
§ Blumenthal, Jacob S.	Minneapolis
*†Bockman, Michael W. H.	Hot Springs Nat'l Park, Ark.
‡ Bodelson, A. H.	Little Rock, Ark.
§ Boehrer, John J.	Minneapolis
§ Bofenkamp, Benjamin	Minneapolis
‡§Bohn, Donald G.	Minneapolis
§ Boies, Lawrence R.	Minneapolis
† Booth, Albert E.	Minneapolis
† Boreen, Clifton A.	Minneapolis
†§Borgeson, Egbert J.	St. Paul
§ Borman, Chauncey N.	Minneapolis
§ Borowicz, Leonard A.	Minneapolis
§ Bowers, Gordon G.	Minneapolis
Boynton, Ruth E.	Minneapolis
§ Bratrud, Arthur F.	Minneapolis
Bratrud, Theodore E.	Minneapolis
‡ Breitenbucher, R. B.	Columbia, S. C.
§ Brekke, Harvey J.	Minneapolis
§ Bridge, Allyn G.	Minneapolis
Brill, Alice K.	Minneapolis
† Brooks, Charles N.	Minneapolis
† Brown, Edgar D.	Paynesville
Brown, Ian A.	Minneapolis
Brown, William D.	Minneapolis
§ Buchstein, Harold F.	Minneapolis
§ Buirge, Raymond E.	Minneapolis
Bulkley, Kenneth	Minneapolis
§ Burnham, Wesley H.	Minneapolis
§ Bushard, Wilford J.	Minneapolis
§ Buzzelle, Leonard K.	Minneapolis
§ Cable, Morris L.	Minneapolis
§ Cabot, Clyde M.	Minneapolis
Cady, Laurence H.	Minneapolis
Cameron, Isabell L.	Minneapolis
Campbell, Lowell M.	Minneapolis
§ Campbell, Orwood J.	Minneapolis
§ Caplan, Leslie	Minneapolis
§ Card, William H.	Minneapolis
§ Carey, James B.	Minneapolis
§ Carlander, Lester W.	Minneapolis
§ Carlson, Lawrence	Minneapolis
§ Carlson, Leonard T.	Minneapolis
§ Caron, Robert P.	Minneapolis
Carr, William J.	Minneapolis
§ Caspers, Carl G.	Minneapolis
†§Cavanor, Frank T.	Minneapolis
§ Ceder, Elmer T.	Minneapolis
Challman, Samuel Alan	Minneapolis
§ Chavez, Demetrio A.	Minneapolis
§ Chesler, Merrill D.	Minneapolis
† Chesley, Albert J.	Minneapolis
§ Chisholm, Tague C.	Minneapolis
§ Christensen, Llewellyn E.	Minneapolis
Clark, Malcolm D.	Minneapolis
Clarke, Eric K.	Minneapolis
§ Clay, Lyman B.	Minneapolis
§ Cochrane, Ray F.	Minneapolis
Coe, John I.	Minneapolis
§ Cohen, Bernard A.	Minneapolis
Cohen, Ephraim B.	Minneapolis
Cohen, Maynard M.	St. Paul
§ Cohen, Sumner S.	Oak Terrace
Cole, James S.	Minneapolis
Colp, Edward Adams	Minneapolis
Condit, William H.	Minneapolis
§ Cooper, John P.	Excelsior
§ Cooper, Robert R.	Minneapolis
Corniea, Albert D.	Minneapolis
§ Correa, Dale H.	Minneapolis
§ Corrigan, Cyril J.	Minneapolis
Cowan, Donald W.	Minneapolis
Craig, M. Elizabeth	Minneapolis
§ Cranmer, Richard R.	Minneapolis
Cranston, Robert W.	Minneapolis
§ Creevy, Charles D.	Minneapolis
§ Creighton, Ralph H.	Minneapolis
§ Culligan, Leo C.	Minneapolis
§ Cundy, Donald T.	Minneapolis
† Cutts, George	Minneapolis
Dady, Elmer E.	Minneapolis
§ Daggett, Donald R.	Minneapolis
§ Dahl, Elmer O.	Minneapolis
‡ Dahl, James C.	Minneapolis
§ Dahl, John A.	Minneapolis
†§Daniel, Donald H.	Minneapolis
§ Danyluk, Michael	Minneapolis
§ David, Reuben	Minneapolis
§ Davis, Jay C.	Minneapolis
§ Davis, William I.	Mound
§ del Plaine, Carlos W.	Minneapolis
§ Devereaux, Thomas J.	Wayzata
DeWall, Richard	Minneapolis
Dickman, Roy W.	Minneapolis
§ Diefenbach, Eugene J., Jr.	Minneapolis
§ Diehl, Harold S.	Minneapolis
§ Dierker, Heinrich	Minneapolis
Diessner, Henry D.	Minneapolis
§ Donatelle, Edward P.	Minneapolis
§ Dorge, Richard I.	Minneapolis
§ Dornbach, Robert A.	Excelsior
† Dornblaser, Harry B.	Los Gatos, Calif.
§ Dorsey, George C.	Minneapolis
† Doxey, Gilbert L.	Minneapolis
§ Doyle, Lawrence O.	Minneapolis
†§Drake, Charles R.	Minneapolis
§ Drill, Herman E.	Hopkins
§ Duff, Edwin R.	Minneapolis
Dummer, Donald J.	Minneapolis
§ Dunlap, Earl H.	Minneapolis
§ Dupont, Joseph A.	Excelsior
§ Duryea, Willis M.	Minneapolis
† Dutton, C. E.	Minneapolis
§ Dvorak, Benjamin A.	Minneapolis
§ Dwan, Paul F.	Minneapolis
§ Dworsky, Samuel D.	Minneapolis
§ Eder, W. P.	Minneapolis
§ Ehrenberg, Claude J.	Minneapolis
§ Ehrlich, S. Paul	Minneapolis
§ Eich, Matthew A.	Minneapolis
§ Eichhorn, Edmund P.	Minneapolis
Eisenstadt, David H.	Minneapolis
§ Eisenstadt, William S.	Minneapolis
§ Eitel, George D.	Minneapolis
§ Ellison, David E.	Minneapolis
§ Ellison, Ellis	Minneapolis
§ Emond, Albert J.	Farmington
Emond, Joseph S.	Farmington
Engel, Joseph P.	Minneapolis
§ Engelhart, Peter C.	Minneapolis
Englund, Elvin F.	Minneapolis
§ Engstrand, Oscar J.	Minneapolis
§ Engstrom, Denton P.	Minneapolis
§ Erickson, Clifford O.	Minneapolis
§ Erickson, Laurence F.	Minneapolis
§ Erickson, Myron E.	Minneapolis
§ Erickson, Reuben F.	Minneapolis
§ Ericson, Reinhold M.	Wayzata
§ Esensten, Sidney	Minneapolis
§ Evans, Edward T.	Minneapolis
Evans, Robert D.	Minneapolis
† Fahr, George	Minneapolis
§ Fansler, Walter A.	Minneapolis
§ Feeney, John M.	Minneapolis
‡§Feigal, David W.	Air Base, Wyoming
Feinberg, Philip	Minneapolis
§ Feinstein, Julius Y.	Minneapolis
§ Fenger, Ejvind P. K.	Oak Terrace
§ Field, Charles W.	Minneapolis
§ Fingerman, David L.	Minneapolis
§ Fink, Leo Wm.	Minneapolis
§ Fink, Walter H.	Minneapolis
§ Fisher, Isadore I.	Minneapolis
† Fitzgerald, D. F.	Wayzata
†§Fjeldstad, Christian A.	Minneapolis
§ Fleeson, William H.	Minneapolis
*†Fleming, Aloysius S.	Madison, N. J.
Fleming, Dean S.	Hopkins
§ Fliehr, Richard R.	Minneapolis
Flink, Edmund B.	Minneapolis
§ Foker, Leslie W.	Minneapolis
§ Folsom, Louis B.	Minneapolis
§ Ford, William H.	Minneapolis
§ Foster, Orley W.	Minneapolis
§ Fowler, Lucius Haynes	Minneapolis
Fox, Donald P.	Minneapolis
§ Fox, James Rogers	Minneapolis
§ Frane, Donald B.	Minneapolis
† Frear, Rosemary R.	Minneapolis
§ Fredericks, George M.	Minneapolis
§ Freeman, Craig W.	Minneapolis
Freeman, Donald W.	Minneapolis
§ French, Lyle A.	Minneapolis
§ Frey, Richard J.	Minneapolis
Friberg, Joseph B.	Minneapolis
§ Fried, Louis A.	Minneapolis
§ Friedell, Aaron	Minneapolis
Friedell, George	St. Louis Park
§ Friedman, Harry S.	Minneapolis
§ Friedman, Jack	Minneapolis
Friend, Charles A.	Minneapolis
§ Frost, John B.	Minneapolis
§ Frost, Russell H.	Minneapolis

§ Frykman, Howard M.	Minneapolis
Fuller, Alice H.	Minneapolis
§ Funk, Victor K.	Oak Terrace
Furman, Lucie Christine	Minneapolis
§ Gaard, Richard C.	Minneapolis
§ Gallett, Lester E.	Minneapolis
† Galligan, Margaret M.	Minneapolis
*§Galloway, John B.	Minneapolis
§ Gammell, John H.	Minneapolis
§ Garske, George L.	Minneapolis
Garten, Joseph L.	Minneapolis
Garvey, James T.	Minneapolis
§ Gaviser, David	Minneapolis
§ Gibbs, Robert W.	Minneapolis
§ Giebenhain, John N.	Minneapolis
§ Giere, Joseph C.	Minneapolis
§ Giere, Richard W.	Minneapolis
Gilbert, Maurice G.	Minneapolis
Gingold, Benjamin A.	Minneapolis
† Girvin, Richard B.	Minneapolis
§ Glaeser, John H.	Minneapolis
§ Goldberg, Isadore M.	Minneapolis
§ Goldman, Theodore I.	Minneapolis
§ Goldner, Meyer Z.	Minneapolis
§ Goltz, Robert W.	Minneapolis
§ Good, Hoff D.	Minneapolis
Goodchild, William R.	Minneapolis
Gordon, Philip E.	Minneapolis
Gordon, Sewell S.	St. Louis Park
‡§Grais, Melvin L.	Colo. Springs, Colo.
Grant, Suzanne	Minneapolis
§ Gratzek, Frank R. E.	Minneapolis
Gray, Royal C.	Minneapolis
Green, Robert A.	St. Louis Park
§ Greenberg, Albert J.	Minneapolis
§ Greenfield, Irving	Minneapolis
† Greishiemer, Esther M.	Phila., Pa.
Grimes, Marian	Minneapolis
Grimmell, Francis J.	Minneapolis
§ Gronvall, Paul R.	Minneapolis
§ Grotting, John K.	Minneapolis
Gullickson, Glenn, Jr.	Minneapolis
§ Gunlaugson, Frederick G.	Minneapolis
§ Gushurst, Edward G.	Minneapolis
§ Gustason, Harold T.	Minneapolis
Haberer, Helen R.	Minneapolis
§ Hagen, Kristofer	Richfield
§ Hagen, Wayne S.	Minneapolis
† Haggard, G. D.	Minneapolis
§ Hall, Harry B.	Minneapolis
§ Hall, Wendell H.	Minneapolis
* Hallberg, Charles A.	Minneapolis
§ Hannah, Hewitt B.	Minneapolis
§ Hansen, Cyrus O.	Minneapolis
§ Hansen, Erling W.	Minneapolis
Hansen, Olga S.	Minneapolis
‡§Hansen, Rollin M.	Falls Church, Va.
§ Hanson, Harlow J.	Minneapolis
Hanson, Henry V.	Minneapolis
§ Hanson, Malcolm B.	Minneapolis
§ Hanson, Mark C. L.	Minneapolis
§ Hanson, William A. H.	Minneapolis
§ Happe, Lawrence J.	Minneapolis
Harris, Leon D.	Minneapolis
Hartman, Evelyn E.	Minneapolis
§ Hass, Frederick M.	Minneapolis
§ Hastings, DeForest R.	Minneapolis
§ Hastings, Donald W.	Minneapolis
§ Hauge, Erling T.	Minneapolis
§ Haugen, George W.	Minneapolis
Haugen, John A.	Minneapolis
Hauser, Donald C.	Minneapolis
Hauser, George W.	Minneapolis
Havel, Robert J.	Minneapolis
§ Haven, Walter K.	Minneapolis
§ Hawkinson, Raymond P.	Minneapolis
§ Hay, Lyle J.	Minneapolis
† Hayes, James M.	Minneapolis
§ Hays, Albert T.	Minneapolis
§ Head, Douglas P.	Minneapolis
§ Hebbel, Robert	Minneapolis
‡§Heisler, John J.	Minneapolis
Hempel, Dean J.	Minneapolis
† Hendrickson, John F.	Minneapolis
§ Henrikson, Earl C.	Minneapolis
† Henry, Clifford E.	Kirksville, Mo.
Herbert, Willis L.	Minneapolis
§ Hermann, Harold W.	Minneapolis
†§Higgins, John H.	Minneapolis
Hill, Earl	Minneapolis
§ Hill, Elmer M.	Minneapolis
†§Hillis, Samuel J.	East Bradenton, Fla.
Hinckley, Robert G.	Minneapolis
§ Hirshfield, Frank R.	Minneapolis
§ Hitchcock, Claude R.	Minneapolis
Hoffbauer, Frederick W.	Minneapolis
§ Hoffert, Henry E.	Minneapolis
§ Hoffman, Roy A.	Minneapolis
§ Hoffman, Walter L.	Minneapolis
§ Holmberg, Conrad J.	Minneapolis
§ Holzapfel, Fred C.	Minneapolis
‡ Horns, Howard L.	Minneapolis
§ Horns, Richard C.	Minneapolis
† Houkom, Bjarne	T. T. East Africa
§ Houle, Rollin J.	New Brighton
Hovland, Melvin L.	Minneapolis
Howard, Robert B.	Minneapolis
Howard, Solomon E.	Minneapolis
§ Howell, Carter W.	Minneapolis
Hudson, George E.	Minneapolis
§ Huenekens, Edgar J.	Minneapolis
§ Hultkrans, Rudolph E.	Minneapolis
† Hurd, Annah	Minneapolis
Hutchinson, Dorothy W.	Oak Terrace
§ Hymes, Charles	Minneapolis
† Hynes, John E.	Minneapolis
Ide, Arthur W., Jr.	Minneapolis
§ Idstrom, Linneus G.	Minneapolis
§ Ingalls, Edgar G.	Minneapolis
† Irvine, Harry G.	Minneapolis
§ Iverson, Rolf M.	Minneapolis
§ Jacobson, Loren J.	Minneapolis
§ Jacobson, Wyman E.	St. Louis Park
§ Jay, Alan R.	Minneapolis
Jefferies, William L.	Minneapolis
‡ Jensen, Adrian R.	Roslindale, Mass.
§ Jensen, Harry C.	Minneapolis
†§Jensen, Marius J.	Minneapolis
§ Jensen, Nathan K.	Minneapolis
Jensen, Reynold A.	Minneapolis
Jerome, Bourne	Minneapolis
Jerome, Elizabeth	Minneapolis
‡ Jeub, Robert P.	San Antonio, Texas
§ Johnson, Arthur B.	Minneapolis
§ Johnson, August E.	Minneapolis
§ Johnson, Emil W.	Minneapolis
Johnson, Frank E.	Minneapolis
§ Johnson, Harry A.	Minneapolis
†§Johnson, James A.	Minneapolis
§ Johnson, John W.	Minneapolis
†§Johnson, Julius	Minneapolis
§ Johnson, Malcolm R.	Minneapolis
Johnson, Norman P.	Minneapolis
§ Johnson, Norton T.	Minneapolis
§ Johnson, Reinald G.	Minneapolis
§ Johnson, Reuben A.	Minneapolis
§ Johnson, Robert E.	Minneapolis
Johnson, Roger S.	Wayzata
Johnson, Youbert T.	Minneapolis
§ Jones, David G.	Minneapolis
†§Jones, Herbert W., Jr.	Brookline, Mass.
§ Jones, Richard H.	Minneapolis
§ Jones, William R.	Minneapolis
Josewich, Alexander	Minneapolis
§ Judd, Allen S.	Minneapolis
§ Judd, Walter H.	Washington, D. C.
Juhl, John H.	Minneapolis
§ Jurdy, Mitchell J.	Minneapolis
Kadesky, Harold B.	Minneapolis
§ Kalin, Oscar T.	Minneapolis
§ Kallestad, Leonard L.	Wayzata
Kantar, Bruce L.	Minneapolis
§ Kaplan, Harold A.	Minneapolis
§ Kaplan, John J.	Minneapolis
§ Karleen, Conrad I.	Minneapolis
§ Karlen, Markle	Minneapolis
† Katz, Yale J.	Minneapolis
Kaufman, Herschel J.	Minneapolis
§ Kelby, Giert M.	Minneapolis
§ Kelly, Charles F.	Minneapolis
§ Kelly, John P.	Minneapolis
†§Kennedy, Claude C.	Minneapolis
† Kennedy, Jane F.	Minneapolis
§ Kerkhof, Arthur C.	Minneapolis
Kiesler, Frank, Jr.	Minneapolis
† King, Edgar A.	Minneapolis
King, Frances W.	Oak Terrace
§ Kinsella, Thomas J.	Minneapolis
Kistler, Alvin J.	Minneapolis
§ Kline, Richard F.	Wayzata
§ Knapp, Miland E.	Minneapolis
§ Knight, Ralph T.	Minneapolis
Knight, Ray R.	Minneapolis
Knudsen, Helen L.	Minneapolis
§ Koepcke, Gerald M.	Minneapolis
Koller, Hermann M.	Minneapolis
Koller, Louis R.	Minneapolis
Koller, Robert L.	Minneapolis
Korchik, John P.	Minneapolis
Kosiak, John, Jr.	Minneapolis
Kottke, Frederic J.	Minneapolis
§ Koucky, Rudolph W.	Minneapolis
§ Kovack, Freeman D.	Minneapolis
Kremen, Arnold J.	New York, N. Y.
Krieser, Albert E.	Anoka
§ Kucera, Frank J.	Hopkins
§ Kucera, William J., Sr.	Minneapolis
§ Kucera, William J., Jr.	Minneapolis
§ Kusz, Clarence	Minneapolis
§ LaBree, John W.	St. Louis Park
†§Lagaard, Sheldon M.	Minneapolis
† Lajoie, John M.	Minneapolis
§ Lamb, H. Douglas	Minneapolis
§ Lang, Leonard A.	Minneapolis
‡ Lange, Robert D.	Washington, D. C.
§ Lapierre, Arthur P.	Minneapolis
Lapierre, Jean T.	Minneapolis
§ Larsen, Frank W.	Minneapolis
§ Larson, Clarence M.	Minneapolis
§ Larson, Donald M.	Minneapolis
§ Larson, Lawrence M.	Minneapolis
§ Larson, Leonard M.	Oak Terrace
§ Larson, Loren J.	Minneapolis
§ Larson, Paul N.	Minneapolis
Larson, Ralph H.	Anoka
† Larson, Roger C.	Minneapolis
† LaVake, Rae T.	Minneapolis
§ Laymon, Carl W.	Minneapolis
*†Lazar, Henry L.	Excelsior
† Leavitt, H. H.	Minneapolis
† Lebowske, Joseph A.	Minneapolis
§ Leemhuis, Andrew J.	Minneapolis
Leiferman, Robert J.	Minneapolis
§ Leland, Harold R.	Minneapolis
§ Lenz, Otto A.	Minneapolis
§ Leonard, Lawrence J.	Minneapolis
§ Leonard, Samuel	Minneapolis
Lerner, A. Ross	Minneapolis
§ Lewis, F. John	Minneapolis
Lillehei, C. Walton	Minneapolis
Lillehei, Elmer J.	Robbinsdale
‡ Lind, C. J., Jr.	Ft. Sam Houston, Tex.
† Lindberg, Arvid C.	Minneapolis
§ Lindberg, Vernon L.	Minneapolis
Lindberg, Winston R.	Minneapolis
Lindblom, Maurice L.	Minneapolis
§ Lindemann, Charles E.	Minneapolis
§ Lindgren, Russell C.	Minneapolis
§ Lindner, Janus C.	Minneapolis
§ Lindquist, Richard H.	Minneapolis
§ Linner, Gunnar	Minneapolis
†§Linner, Henry P.	Minneapolis
§ Linner, John H.	Minneapolis
§ Linner, Paul W.	Minneapolis
Lippman, Emanuel S.	Minneapolis
§ Lipschultz, Oscar	Minneapolis
†§Litchfield, John T.	Minneapolis
Litman, Abraham B.	Minneapolis
Lober, Paul H.	Minneapolis
§ Lofsness, Stanley V.	St. Paul
†§Logefeil, Rudolph C.	Minneapolis
† Loomis, Earl A.	Minneapolis
§ Lott, Frederick H.	Minneapolis
Lovett, Beatrice R.	Oak Terrace
Lowry, Elizabeth C.	Minneapolis
§ Lowry, Paul T.	Minneapolis
Lowry, Thomas	Minneapolis
§ Lueck, Wallace W.	Minneapolis
§ Lufkin, Nathaniel H.	Minneapolis
Lund, George W.	Minneapolis
Lundberg, Ruth I.	Minneapolis
Lundblad, Roy A.	Minneapolis
Lundblad, Stanley W.	Minneapolis
Lundeberg, Karl R.	Minneapolis
§ Lundquist, Virgil J. P.	Minneapolis
† Lynch, Matthew J.	Minneapolis
Lyons, James H.	Minneapolis
†§Lysne, Henry	Minneapolis
§ Lysne, Myron	Minneapolis
Lysyj, Anatol	Minneapolis
Lyzenga, Anton G.	Minneapolis
§ McCaffrey, F. John	Minneapolis
§ McCann, Eugene J.	Minneapolis
§ McCannel, Malcolm A.	Minneapolis
McCarthy, Donald	St. Paul
McCartney, James S.	Minneapolis
§ McCormick, Donald P.	Minneapolis
† McDaniel, Orianna	Minneapolis
§ McFarland, Arthur H.	Minneapolis
§ McGandy, Robert F.	Minneapolis
§ McGeary, George E.	Minneapolis
§ McInerny, Maurice W.	Minneapolis
McKelvey, John L.	Minneapolis
§ McKenzie, Charles H.	Minneapolis
§ McKinlay, Chauncey A.	Minneapolis
§ McKinney, Frank S.	Minneapolis
§ McLaughlin, Byron H.	Minneapolis
§ McMurtrie, William B.	Minneapolis
McNeil, John J.	Minneapolis
§ McPheeters, Herman O.	Minneapolis
† McQuarrie, Irvine	Minneapolis
§ MacDonald, Daniel A.	Minneapolis
§ MacDonald, John W.	Minneapolis
§ MacKinnon, Donald C.	Minneapolis
§ Mach, Frank B.	Minneapolis
§ Maeder, Edward C.	Minneapolis
†§Maland, Clarence O.	Minneapolis
Mandel, Sheldon L.	Minneapolis
§ Mankey, James C.	Minneapolis
§ Marking, George H.	Minneapolis
§ Martin, George R.	Minneapolis
Martinson, Carl J.	Wayzata
Martinson, Elmer J.	Wayzata
§ Mattill, Peter M.	Oak Terrace
§ Mattson, Hamlin A. N.	Minneapolis
§ Maxeiner, Stanley R.	Minneapolis
Maxeiner, Stanley R., Jr.	Minneapolis
§ Meller, Robert L.	Minneapolis
§ Merkert, Charles E.	Minneapolis

§ Merkert, George L	Minneapolis
† Merrick, Charlotte T	St. Paul
§ Meyer, Alvin J	Minneapolis
†§Meyer, Ette L	Minneapolis
Meyer, Robert J	Minneapolis
Michael, Joseph C	Minneapolis
§ Michel, Henry H	Minneapolis
§ Michelson, Henry E	Minneapolis
† Mickelsen, Emma F	Minneapolis
§ Miller, Arden L	Minneapolis
§ Miller, Harold E	Minneapolis
§ Miller, Hugo E	Minneapolis
§ Miller, John Carleton	Minneapolis
§ Millett, D. Keith	Minneapolis
§ Milton, John S	Minneapolis
Minsky, Armen A	Minneapolis
§ Mitby, Irving	Minneapolis
§ Mitchell, Berton D	Minneapolis
Mitchell, Edward C	Minneapolis
§ Mitchell, Mancel T	Minneapolis
‡§Mixer, Harry W	Ft. Hood, Texas
Moberg, Thomas D	Minneapolis
§ Moe, John H	Minneapolis
§ Moehn, John T	Minneapolis
§ Moen, Johannes K., Jr	Minneapolis
† Monahan, Elizabeth S	Minneapolis
§ Monson, Einer M	Minneapolis
§ Moore, Irvin H	Minneapolis
Moorhead, Marie	Minneapolis
§ Moos, Daniel J	Minneapolis
§ Mork, Arthur H	Anoka
§ Mork, Frank E	Anoka
Morrison, Charlotte J	Minneapolis
§ Mosser, Donn G	Minneapolis
§ Moyer, Leonard B	Minneapolis
§ Mulholland, William M	Minneapolis
Murphy, Edmund P	Minneapolis
† Murray, Elisabeth Merrill	Minneapolis
Muske, Marvin M	Minneapolis
§ Musty, Nicholas J	Minneapolis
§ Myers, Jay A	Minneapolis
§ Myhre, James A	Minneapolis
§ Naslund, Ames W	Minneapolis
Nauth, Bernard S	Minneapolis
§ Neal, Joe M	St. Paul
§ Neary, Richard P	Minneapolis
Nelson, Bernette G	Minneapolis
Nelson, Bernice A	Minneapolis
Nelson, C. Barton	Minneapolis
Nelson, Carleton A	Minneapolis
§ Nelson, Edward N	Minneapolis
§ Nelson, Harvey	Minneapolis
§ Nelson, Lloyd S	Minneapolis
Nelson, Maxine O	Minneapolis
§ Nelson, Maynard C	Minneapolis
§ Nelson, O. L. Norman	Minneapolis
§ Nelson, Wallace I	Minneapolis
§ Nerenberg, Samuel T	Minneapolis
§ Nesbitt, Samuel	Minneapolis
§ Nesset, Lawren B	Minneapolis
§ Nesset, William D	Minneapolis
§ Neumaier, Arthur	Minneapolis
§ Neumeister, Charles A	Minneapolis
§ Nice, Charles M	Minneapolis
* Noonan, William J	Minneapolis
Noran, Axel S. N	Minneapolis
§ Noran, Harold H	Minneapolis
§ Nord, Robert E	Edina
§ Nordin, Gustaf T	Minneapolis
§ Nordland, Martin, Sr	Minneapolis
§ Nordland, Martin, Jr	Minneapolis
† Noth, Henry W	Minneapolis
Nudell, Gerald	Minneapolis
§ Nydahl, Malvin J	Minneapolis
§ Nylander, Emil G	Minneapolis
O'Donnell, James E	Minneapolis
O'Hanlon, John A	Minneapolis
§ O'Phelan, E. Harvey	Minneapolis
† Oberg, Carl M	Minneapolis
Olavs, Olga	Minneapolis
§ Olsen, Edward George	Minneapolis
§ Olson, Alton C	Minneapolis
§ Olson, Carl J	Minneapolis
Olson, C. Kent	Minneapolis
Olson, Detlof M	Minneapolis
† Olson, Olof A	Minneapolis
Olson, Rolland A	Minneapolis
§ Oppen, E. Gerhard	Minneapolis
§ Oppen, Melvin G	Wayzata
§ Opstad, Earl T	Oak Terrace
§ Palen, Benjamin J	Minneapolis
Papermaster, Theodore C	Minneapolis
Park, Wilford E	Minneapolis
Pattee, James J	Minneapolis
§ Peluso, Charles R	Minneapolis
§ Peppard, Thomas A	Minneapolis
§ Perlman, Everett C	Minneapolis
§ Perlman, Herschel L	Minneapolis
§ Peteler, Jennings C. L	Minneapolis
§ Petersen, Deane A	Minneapolis
§ Petersen, Glenn L	Minneapolis
Petersen, Peter C	Minneapolis
§ Petersen, William E	Minneapolis
Peterson, Alice H	Minneapolis
Peterson, Henry	Minneapolis
§ Peterson, Herbert W	Minneapolis
§ Peterson, Nordahl P	Minneapolis
Peterson, Oliver H	Minneapolis
† Peterson, O. H., Jr	Minneapolis
§ Peterson, Palmer A	Minneapolis
§ Peterson, Peter E	Minneapolis
§ Peterson, Willard C	Minneapolis
§ Petit, Julien V	Minneapolis
§ Petit, Leon J	Minneapolis
§ Pewters, John T	Minneapolis
§ Peyton, William T	Minneapolis
§ Phelps, Kenneth A	Minneapolis
Plasha, Matthew K	Anoka
§ Plass, Herbert F. R	Minneapolis
†§Platou, Erling S	Minneapolis
Pleissner, Karl W	Minneapolis
§ Plimpton, Nathan C., Jr	Minneapolis
Pohl, John F. M	Minneapolis
†§Pollock, David K	Minneapolis
§ Polzak, Jacob A	Minneapolis
† Poppe, Frederick H	Minneapolis
Potek, David	Minneapolis
§ Potter, Robert B	Hopkins
† Pratt, Fred J., Sr	Minneapolis
§ Pratt, Fred J., Jr	Minneapolis
§ Preston, Paul J	Minneapolis
§ Price, William E	Minneapolis
§ Priest, Robert E	Minneapolis
†§Prim, Joseph A	Minneapolis
§ Proffitt, William E	Minneapolis
§ Proshek, Charles E	Minneapolis
§ Quello, Robert O	Minneapolis
§ Quiggle, Arthur B	Minneapolis
†§Quist, Henry W., Sr	Minneapolis
§ Quist, Henry W., Jr	Minneapolis
§ Ransom, H. Robert	Osseo
§ Reader, Donald R	Minneapolis
§ Regnier, Edward A	Minneapolis
§ Reif, Harold A	Minneapolis
§ Reiley, Richard E	Minneapolis
Reitmann, John H	Anoka
§ Remole, William D	Minneapolis
§ Resch, Joseph A	Minneapolis
§ Rice, Carl O	Minneapolis
‡ Rice, Frank B	Minneapolis
§ Rice, Fred A	Minneapolis
§ Richdorf, Lawrence F	Minneapolis
§ Rieke, Wellington W	Wayzata
Rigler, Leo G	Minneapolis
Riordan, Elsie M	Minneapolis
§ Risch, Ronald E	Minneapolis
§ Rizer, Dean K	Minneapolis
† Rizer, Robert I	Minneapolis
Robb, Edwin F	Minneapolis
§ Robbins, Owen F	Minneapolis
† Roberts, Byron H	Minneapolis
§ Roberts, Lewis J	Minneapolis
§ Roberts, Stanley W	Minneapolis
†§Roberts, William B	Minneapolis
Robinson, Cartland O	Minneapolis
§ Rocknem, Robert E	Minneapolis
§ Rockwell, Curtiss V	Minneapolis
† Rodda, Fredk. C	Minneapolis
§ Rodgers, Richard S	Minneapolis
§ Romness, Kenneth B	Mound
† Rosander, Phyllis	Minneapolis
§ Rosenbaum, David L	Minneapolis
§ Rosendahl, Frederick G	Minneapolis
§ Rosenfield, Abraham B	Minneapolis
§ Rosenow, John H	Minneapolis
† Rosenwald, Reuben M	Anoka
§ Ross, Alexander J	Minneapolis
§ Rossen, Ralph X	Minneapolis
Rotenberg, Robert J	Minneapolis
§ Rothnem, Morris S	Minneapolis
§ Rucker, William H	Minneapolis
Rud, Norman E	Minneapolis
† Rudell, Gustaf L	Minneapolis
§ Russeth, Arthur N	Minneapolis
§ Rusten, Elmer M	Minneapolis
Rydburg, Wayne C	Minneapolis
§ St. Cyr, Harry M., Jr	Robbinsdale
§ St. Cyr, Kenneth J	Robbinsdale
§ Sadler, Wm. P., Jr	Minneapolis
Safirescu, Sorin R	Minneapolis
§ Saliterman, Bernard I	Minneapolis
§ Samuelson, Samuel	Minneapolis
§ Sandt, Karl E	Minneapolis
§ Sawatzky, William A	Minneapolis
§ Sborov, Abe M	Minneapolis
§ Schaaf, Frederick H. K	Minneapolis
Schaar, Frances E	Minneapolis
§ Schaefer, Kenneth F	Minneapolis
Schaefer, Wesley G	Minneapolis
† Scheldrup, N. H	Minneapolis
§ Scherer, Leslie Raymond	Minneapolis
Scherling, Sidney S	Minneapolis
§ Schiele, Bertrum C	Minneapolis
§ Schissel, Gregory A	Minneapolis
§ Schmidt, W. Robert	Minneapolis
† Schmitt, S. C	Los Angeles, Calif.
Schneck, Jack I	Waukesha, Wis.
§ Schottler, Max E	Minneapolis
§ Schroeder, Albert J	Minneapolis
Schultz, Alvin L	Minneapolis
§ Schultz, J. Harold	Minneapolis
§ Schultz, Peter J	Minneapolis
§ Schulze, William M	Minneapolis
§ Schumacher, John W	Robbinsdale
§ Schwartz, E. Robert	Minneapolis
§ Schwartz, Virgil J	Minneapolis
§ Scott, Horace G	Minneapolis
§ Seaberg, John A	Minneapolis
Seham, Max	Minneapolis
§ Seifert, Milton H	Minneapolis
§ Seljeskog, Sigsbee R	Minneapolis
§ Semsch, Robert D	Minneapolis
§ Shandorf, James F	Minneapolis
Shaperman, Eva P	Minneapolis
§ Shapiro, Sidney K	Minneapolis
§ Sharp, David	Minneapolis
§ Shaw, Howard A	Minneapolis
§ Shea, Andrew W	Minneapolis
Sheldon, Warren N	Minneapolis
§ Sher, Lewis	Minneapolis
§ Sherman, Lloyd F	Minneapolis
Shillington, Maurice A	Minneapolis
Shronts, John F	Minneapolis
Sidell, Franklin D	Butte, Mont.
§ Siegmann, William C	Minneapolis
Silas, Ralph M	Minneapolis
Silver, John D	Minneapolis
†§Simons, Jalmar	Minneapolis
§ Simonson, Donald B	Minneapolis
† Simpson, Ellery De	Phoenix, Ariz.
§ Sinykin, Melvin B	Minneapolis
Siperstein, David M	Minneapolis
§ Sisterman, Thomas J	Minneapolis
† Sivertsen, Andrew	Minneapolis
†§Sivertsen, Ivar	Minneapolis
§ Skjold, Arthur C	Minneapolis
Smiley, John T	Minneapolis
§ Smisek, Frank M. E	Minneapolis
Smith, Adam M	Minneapolis
§ Smith, Archie M	Minneapolis
§ Smith, Baxter A., Jr	Minneapolis
§ Smith, Graham G	Minneapolis
† Smith, Homer R	Minneapolis
† Smith, Margaret I	Gardena, Calif.
§ Smith, Norvin R	Willmar
§ Smith, Theodore S	Minneapolis
§ Smith, William T	Minneapolis
§ Soderlind, Ragnar T	Minneapolis
§ Solhaug, Samuel B	Minneapolis
§ Solvason, Harold M	Minneapolis
Spain, W. Thomas	Princeton, N. J.
§ Spano, Joseph P	Minneapolis
§ Spencer, Bernard J	Minneapolis
§ Spink, Wesley W	Minneapolis
§ Sponsel, Kenath H	Minneapolis
† Spratt, Charles N	Minneapolis
§ Stahr, Aubrey C	Hopkins
§ Stanford, Charles E	Minneapolis
Staub, Henry P	Minneapolis
§ Stelter, Lloyd A	Minneapolis
§ Stennes, John L	Minneapolis
Stenstrom, Annette E. T	Minneapolis
§ Sterrie, Norman A	Minneapolis
Stewart, Marvin J	Minneapolis
§ Stewart, Rolla I	Minneapolis
†§Stiegler, Farrell S	St. Paul
§ Stoesser, Albert V	Minneapolis
§ Stoltz, Robert C	Minneapolis
† Stomel, Joseph	Blackfoot, Idaho
Stone, Stanley P	Minneapolis
† Strachauer, Arthur C	Minneapolis
§ Strickler, Jacob H	Minneapolis
§ Strom, Gordon W	Minneapolis
Stromgren, Delph T	Minneapolis
§ Stromme, William B	Minneapolis
Strunk, Clarence A	Minneapolis
§ Sturges, Robert L	Minneapolis
†§Subby, Walter	Minneapolis
Sukov, Marvin	Minneapolis
Sullivan, Raymond M	Minneapolis
Sullivan, Robert E	Minneapolis
§ Swain, Francis M	Minneapolis
Swanson, Roy E	Minneapolis
§ Sweetser, Horatio B	Minneapolis
§ Sweetser, Theodore H., Sr	Minneapolis
Sweetser, Theodore H., Jr	Minneapolis
† Sweitzer, Samuel E	Minneapolis
†§Swendseen, Carl G	Starbuck
§ Syverton, Jerome T	Minneapolis
Tam, Ernest C	Minneapolis
§ Tangen, George M	Minneapolis
Taylor, Joseph H	Minneapolis
§ Tenner, Robert J	Minneapolis
† Thomas, George E	Minneapolis
Thomes, A. Boyd	Minneapolis
Thompson, Arthur	Minneapolis

§ Thompson, Willis H. ... Minneapolis
§ Thorsen, David S. ... Minneapolis
§ Thorson, Stuart V. ... Minneapolis
§ Thysell, Desmond M. ... Minneapolis
Tichy, Fae Y. ... Minneapolis
† Tingdale, August C. ... Minneapolis
§ Tinkham, Robert G. ... Minneapolis
§ Titrud, Leonard A. ... Minneapolis
§ Tobin, John D. ... Minneapolis
§ Trach, Benedict B. ... Minneapolis
§ Trow, James E. ... Minneapolis
§ Trow, William H. ... Minneapolis
Trueman, Harold S. ... Minneapolis
Tsai, Shih Hao ... Oak Terrace
§ Tucker, Richard C. ... Minneapolis
§ Tudor, Richard B. ... Minneapolis
†§Turnacliff, Dale D. ... St. Paul
Twomey, John E. ... Minneapolis
§ Ude, Walter H. ... Minneapolis
†§Ulrich, Henry L. ... Minneapolis
§ Ulvestad, Harold S. ... Minneapolis
§ Undine, Clyde A. ... Minneapolis
Vermund, Halvor ... Minneapolis
Vik, A. Elliott ... Minneapolis
§ Vitols, T. M. ... Minneapolis
§ Wahlquist, Harold F. ... Minneapolis
Waldron, Carl W. ... Hopkins
§ Wall, Carl R. ... Minneapolis
§ Walonick, Albert L. ... St. Louis Park
§ Walsh, Francis M. ... Minneapolis
§ Walsh, William T. ... Minneapolis
Wangensteen, Owen H. ... Minneapolis
Ward, Percy A. ... Minneapolis
§ Watson, C. Gordon ... Minneapolis
Watson, Cecil J. ... Minneapolis
† Weaver, Myron Mc ... Vancouver, B. C., Canada
§ Webb, Edgar A. ... Minneapolis
Webb, Roscoe C. ... Minneapolis
§ Webber, Richard J. ... St. Louis Park
§ Weber, Lowell W. ... Minneapolis
§ Weisberg, Rapheal J. ... Minneapolis
§ Wendland, John P. ... Minneapolis
† Werner, George ... Minneapolis
§ Wesolowski, Stanley P. ... Minneapolis
† West, Catherine C. ... Minneapolis
Wetherby, Macnider ... Minneapolis
Wheeler, Robert W. ... Minneapolis
§ White, Asher A. ... Minneapolis
† White, S. Marx ... Minneapolis
§ White, Willard D. ... Minneapolis
§ Whitesell, Lloyd A. ... Minneapolis
§ Widen, Wilford F. ... Minneapolis
† Wilder, Kenneth W. ... Minneapolis
§ Wilder, Robert L. ... Minneapolis
†§Wilder, R. M., Jr. ... Minneapolis
§ Wilken, Paul A. ... Minneapolis
† Willcutt, Clarence E. ... Phoenix, Ariz.
† Williams, Robert ... New York, N. Y.
Winchell, Paul ... Minneapolis
Winther, Nora M. C. ... Minneapolis
§ Wippermann, Frederic F. ... Minneapolis
†§Witham, Carl A. ... Minneapolis
§ Wittich, Frederick W. ... Minneapolis
Wohlrabe, Arthur A. ... Minneapolis
§ Wohlrabe, A. Cabot ... Minneapolis
§ Wolf, Alfred H. ... Minneapolis
Wolter, Frederick H. ... Minneapolis
§ Wright, Thomas D. ... Minneapolis
§ Wright, Wale S. ... Minneapolis
§ Wright, William S. ... Minneapolis
Wyatt, Oswald S. ... Minneapolis
Wynne, Herbert M. N. ... Minneapolis
§ Ylvisaker, Ragnvald S. ... Minneapolis
§ Yoerg, Otto W. ... Minneapolis
Yue, Wen Y. ... Oak Terrace
§ Zahrendt, O. Lewis ... Minneapolis
§ Zarling, V. Richard ... Minneapolis
§ Zaworski, Leo A. ... Minneapolis
§ Zierold, Arthur A. ... Minneapolis
§ Zinter, Ferdinand A. ... Minneapolis
§ Ziskin, Thomas ... Minneapolis

KANDIYOHI-SWIFT-MEEKER COUNTY MEDICAL SOCIETY

Regular meetings, third Thursday
Annual meeting, third Thursday in November
Number of Members—51

President
Jacobs, Douglas L. ... Willmar
Secretary
Opsahl, Lawrence J. ... Willmar

Allison, David D. ... Litchfield
Anderson, Richard E. ... Willmar
Austrian, Sol ... Cosmos
§ Bosland, Howard G. ... Willmar
† Chadbourne, Wayne A. ... Minneapolis
Chunn, Stanley S. ... Willmar
† Daignault, Oscar ... Benson
† Danielson, Karl A. ... Litchfield
Danielson, Lennox ... Litchfield
Dille, Donald E. ... Litchfield
Douglas, Kenneth W. ... Sandstone
Eberley, Tobe S. ... Benson
§ Ellinger, Albert J. ... Willmar
Frederickson, Alice C. ... Willmar
† Frost, Edward H. ... Willmar
§ Gaebe, Milton B. ... Clara City
Giere, Silas W. ... Benson
§ Gilman, Lloyd C. ... Willmar
Griffin, R. P. ... Benson
‡ Guy, Jack A. ... New London
§ Hinz, Walter E. ... Willmar
§ Hodapp, Robert V. ... Willmar
§ Holm, Donald F. ... Benson
§ Houts, Joseph C. ... Dassel
Jacobs, Douglas L. ... Willmar
†§Jacobs, Johannes C. ... Willmar
Kaufman, Edward J. ... Appleton
Lundblad, Robert M. ... Minneapolis
McCarthy, Austin M. ... Willmar
Macklin, William E., Jr. ... Willmar
§ Michels, Roger P. ... Willmar
Nash, Eldore B. ... Eden Valley
†§O'Connor, Daniel C. ... Eden Valley
Olson, Gregory M. ... Litchfield
§ Opsahl, Lawrence J. ... Willmar
Ostling, Burton C. ... Kerkhoven
† Penhall, Fletcher W. ... Willmar
§ Peterson, Willard E. ... Willmar
Porter, Oliver M. ... Atwater
§ Proeschel, Ray K. ... Willmar
§ Rorem, Joseph A. ... Appleton
§ Ruchie, Warren H. ... Willmar
Rygh, Harold N. ... Atwater
Schnell, Frederick S. ... Litchfield
†§Solsem, Fredk. N. S. ... Spicer
Sorum, F. T. ... Willmar
Sutherland, W. H. ... New London
Thompson, A. Henry ... Litchfield
Wagner, Norman W. ... Benson
Wilmot, Cecil A. ... Litchfield
Wilmot, Harold E. ... Litchfield

LYON-LINCOLN COUNTY MEDICAL SOCIETY

Regular meetings, last Tuesday of each month
Annual meeting, last Tuesday of October
Number of Members—26

President
Bodaski, Albert A. ... Tyler
Secretary
Purves, George H. ... Hendricks

Bodaski, Albert A. ... Tyler
Eckdale, John E. ... Marshall
Ferguson, William C. ... Walnut Grove
Ford, Burton C. ... Marshall
† Gray, Frank D. ... Marshall
Hedenstrom, Philip C. ... Marshall
Helferty, John K. ... Boise, Idaho
Hermanson, Peter E. ... Hendricks
† Hoidale, Andrew D. ... Tracy
Johnson, C. Percy ... Tyler
Kreuzer, Titus ... Marshall
Larson, Milo H. ... Lake Benton
Lee, Norman J. ... Tracy
Monson, Leonard J. ... Canby
Murphy, Joseph E. ... Marshall
Peterson, Kenneth A. ... Marshall
Purves, G. Harland ... Hendricks
Remsberg, R. R. ... Tracy
Smith, Lloyd A. ... Willmar
Thill, Leonard J. ... Balaton
Thompson, Carl O. ... Hendricks
†§Vadheim, Alfred L. ... Tyler
†§Valentine, Walter H. ... Tracy
Wolstan, Simon D. ... Minneota
Workman, Warner G. ... Tracy
Yaeger, Wilbert W. ... Marshall

McLEOD COUNTY MEDICAL SOCIETY

Regular meeting, third Thursday of each month
Annual meeting, third Thursday in December
Number of Members—20

President
Smyth, John J. ... Lester Prairie
Secretary
Huebert, Dan W. ... Hutchinson

Bretzke, Carl O. ... Hutchinson
§ Brink, Donald M. ... Hutchinson
Carroll, John J. ... Winsted
† Clement, John B. ... Lester Prairie
§ Floersch, Adrian J. ... Glencoe
§ Griebie, Grant L. ... Brownton
§ Howell, Milton ... Glencoe
§ Huebert, Dan W. ... Hutchinson
§ Jensen, Alvin M. ... Brownton
§ Klima, William W. ... Stewart
§ Lippmann, Elmer W. ... Hutchinson
§ Peterson, Kenneth H. ... Hutchinson
Sahr, Walter G. ... Hutchinson
† Scholpp, Otto W. ... Hutchinson
§ Selmo, Joseph D. ... Norwood
§ Sheppard, Charles G. ... Hutchinson
§ Smith, George R. ... Hutchinson
§ Smyth, John J. ... Lester Prairie
§ Struxness, David F. ... Glencoe
§ Truesdale, Clark W. ... Glencoe

MOWER COUNTY MEDICAL SOCIETY

Regular meeting, last Thursday of every month
Annual meeting, December
New officers elected in November
Number of Members—36

President
PETERSON, STANLEY C..........Austin
Secretary
ROSENTHAL, F. HAROLD..........Austin

§ Anderson, David P., Jr..........Austin
§ Anderson, Harold J..........Austin
§ Anderson, Wallace R..........Austin
§ Barber, Tracy ....Austin
§ Cronwell, Bernhard J..........Austin
Elliot, Harold J..........Hayfield
§ Fisch, Herbert M..........Austin
§ Flanagan, Leonard G..........Austin
§ Grise, William B..........Austin
† Havens, John G. W..........Austin
† Hegge, Olav H..........Austin
†§Hegge, Rolv S..........Austin
† Hertel, Garfield E..........Austin
§ Hesla, Inman A..........Austin
§ Leck, Paul C..........Austin
§ Lommen, Peter A..........Austin
McKenna, Elizabeth M..........Austin
§ McKenna, Jay K..........Austin
† Melzer, George R..........Lyle
§ Miller, Herman..........Grand Meadow
Morse, Morton P..........LeRoy
§ Nesse, J. A..........Austin
§ Osborn, Donald O..........Austin
§ Peterson, Stanley C..........Austin
§ Robertson, Paul A..........Austin
§ Rosenthal, F. Harold..........Austin
§ Sargent, E. C., Jr..........Austin
Schneider, Paul J..........Adams
§ Seery, Thomas M..........Austin
§ Sheedy, Chester L..........Austin
§ Stahl, George W..........Austin
§ Thomson, James M..........Austin
§ Twiggs, Leo F..........Austin
§ Van Cleve, Horatio P., Jr..........Austin
‡ Wilson, Franklin C..........Ft. Belvoir, Va.
§ Wright, Robert R..........Austin

NICOLLET-LE SUEUR COUNTY MEDICAL SOCIETY

Regular meeting, not scheduled
Annual meeting, December
Number of Members—27

President
OLSON, DUANE O..........Gaylord
Secretary
GRIDLEY, JOHN W..........Arlington

†§Aitkens, Herbert B..........LeCenter
§ Canfield, Wayne W..........St. Peter
§ Covell, Walter W..........St. Peter
§ Curtis, Rauen A..........LeCenter
† Ericson, Swan..........Le Sueur
Gridley, John W..........Arlington
§ Grimes, Burton P..........St. Peter
Henry, Martin R..........St. Peter
§ Hiniker, Peter J..........Le Sueur
Johnson, Hobart C..........North Mankato
Kabrick, Ola A..........St. Peter
Kath, Reinhard H..........Arlington
§ Lenander, Mellvin E..........St. Peter
Limbeck, Donald A..........Le Sueur
Maertz, William F..........St. Peter
§ Nilson, Helmer J..........North Mankato
§ Olmanson, Edmund G..........St. Peter
§ Olson, Duane O..........Gaylord
‡ Orwoll, Harold S..........Japan
§ Rudie, Clifford N..........St. Peter
Schulberg, Verne A..........Gaylord
§ Sjostrom, Lawrence E..........St. Peter
§ Sonnesyn, Nels N..........Le Sueur
§ Strathern, Carleton S..........St. Peter
†§Strathern, Fred P..........St. Peter
Traxler, J. Felix..........Henderson
§ Wohlrabe, Clarence F..........North Mankato

OLMSTED-HOUSTON-FILLMORE-DODGE COUNTY MEDICAL SOCIETY

Annual meeting, first Wednesday in November
Regular meeting, first Wednesday every second month starting with January
Number of Members—517

President
JACKMAN, RAYMOND J..........Rochester
Secretary
WELLMAN, WILLIAM E..........Rochester

Abbott, Albert R..........Rochester
§ Achor, Richard W. P..........Rochester
§ Adams, R. Charles..........Rochester
§ Affeldt, Daniel E..........Kasson
Aga, John H..........Rochester
§ Ahlfs, Jacob J..........Caledonia
§ Allen, Edgar V. N..........Rochester
† Amberg, Samuel ....Rochester
§ Andersen, Howard A..........Rochester
§ Anderson, Mark J..........Rochester
Anderson, Markham J., Jr..........Rochester
Anderson, Milton W..........Rochester
Ardan, Nicholas I., Jr..........Rochester
§ Atmore, William G..........Rochester
§ Baggenstoss, Archie H..........Rochester
Bain, Robert C..........Rochester
Bair, Hugo L..........Rochester
§ Baker, George S..........Rochester
†§Baker, Harry R..........Hayfield
‡ Baker, Hillier L., Jr..........West Point, N. Y.
Baker, Russell L..........Hayfield
†§Balfour, Donald C..........Rochester
§ Balfour, William M..........Rochester
§ Banner, Edward Arthur..........Rochester
§ Bargen, J. Arnold..........Rochester
§ Barker, Nelson W..........Rochester
Barlow, Loren C..........Rochester
Barnes, Arlie R..........Rochester
Barry, Maurice J., Jr..........Rochester
Bartholomew, Lloyd G..........Rochester
§ Bastron, James A..........Rochester
‡§Bayard, Edwin D..........
Scott Air Force Base, Illinois
§ Beahrs, Oliver H..........Rochester
Beddow, Ralph M..........Rochester
† Benedict, William L..........Rochester
Berge, Kenneth G..........Rochester
† Berkman, David M..........Oronoco
§ Berkman, John M..........Rochester
§ Bernatz, Philip E..........Rochester
Betts, Charles S..........Rochester
§ Bickel, William Harold..........Rochester
† Bigelow, Charles E..........Dodge Center
§ Black, B. Marden..........Rochester
‡ Bonnet, John D..........Clovia, New Mexico
Bossard, John Wesley..........Rochester
† Botham, Richard James..........Rochester
† Bowers, Dorrance ....Rochester
Bowes, Donald Earl..........Rochester
§ Boyd, David A., Jr..........Rochester
§ Braasch, John W..........Rochester
†§Braasch, William F..........Rochester
Brackett, Ralph D..........Rochester
§ Brandenburg, Robert O..........Rochester
Braun, Robert A..........Rochester
Broadbent, James C..........Rochester
Broders, C. W..........Rochester
§ Brown, Alex E..........Rochester
§ Brown, Henry A..........Rochester
§ Brown, Joe R..........Rochester
Brown, Philip W..........Rochester
Brown, Roland G..........Rochester
§ Brunsting, Louis A..........Rochester
§ Buie, Louis A..........Rochester
Bumpus, Frank Thatcher..........Rochester
§ Burchell, Howard B..........Rochester
§ Burke, Edmund C..........Rochester
§ Butt, Hugh R..........Rochester
§ Cain, James Clarence..........Rochester
† Campagna, Mario Joseph..........Rochester
Campbell, Donald C..........Rochester
§ Carr, David T..........Rochester
§ Carryer, Haddon McC..........Rochester
Chapin, Lemuel E..........Rochester
§ Childs, Donald S., Jr..........Rochester
§ Christensen, Norman A..........Rochester
§ Clagett, Oscar T..........Rochester
Clark, Edward C..........Rochester
§ Clark, Leslie William..........Spring Valley
Clifton, Theodore A..........Chatfield
Colby, M. Y., Jr..........Rochester
Collins, Loren E..........Rochester
§ Comfort, Mandred W..........Rochester
Compton, Russell F..........Rochester
Cook, Edward N..........Rochester
§ Cooley, Jack Crain..........Rochester
Cooper, Milton ....Rochester
Corbin, Kendall B..........Rochester
Costello, Addis C..........Rochester
Counseller, Virgil S..........Rochester
Counts, Robert N..........Rochester
§ Coventry, Markham B..........Rochester
‡ Craig, Richard M..........Rochester
§ Craig, Winchell McK..........Rochester
Crumbley, James J., Jr..........Rochester
Culp, Ormond S..........Rochester
§ Dahlin, David C..........Rochester
‡ Daly, David ....Rochester
§ Daugherty, Guy Wilson..........Rochester
§ Davis, Austin C..........Rochester
Davis, Charles Truman..........Rochester
§ Davis, George D..........Rochester
Davis, Harry L..........Rochester
Davis, Robert E..........Rochester
Dawe, Clyde J..........Rochester
Dearing, William H..........Rochester
Decker, David G..........Rochester
§ Devine, Kenneth D..........Rochester
§ DeWeerd, James H..........Rochester
§ Diessner, Grant Roy..........Rochester
† Dines, David E..........Rochester
Dixon, Claude F..........Rochester
Doane, Joseph C..........Rochester
§ Dockerty, Malcolm B..........Rochester
Dodge, Henry W., Jr..........Rochester
Doehlert, Charles A., Jr..........Rochester
Donin, J. F..........Winnipeg, Man., Canada
§ Donoghue, Francis Edmund..........Rochester
§ Douglass, Bruce E..........Rochester
Doyle, James R..........Rochester
† Drips, Della G..........Oronoco
§ Dry, Thomas J..........Rochester
Dunn, Jack, Jr..........Rochester
† Dyer, John Allen..........Rochester
‡ Dykstra, Peter Calvin..........West Point, N. Y.
§ Eaton, Lealdes M..........Rochester
Edelmann, Robert B..........Rochester
Edwards, Jesse E..........Rochester
§ Elkins, Earl C..........Rochester
§ Ellis, F. Henry..........Rochester
Elstner, Howard L..........Rochester
Emmett, John L..........Rochester
† Ensrud, Earl Richard..........Rochester
§ Erich, John B..........Rochester
Erickson, Donald J..........Rochester
Erickson, John G..........Rochester
§ Estes, J. Earle..........Rochester
†§Eusterman, George B..........Rochester
† Evarts, Arrah B..........Rochester
Ewen, Edgar F..........Rochester
§ Faber, John E..........Rochester
† Fabi, Mario Nestor..........Rochester
Faucett, Robert L..........Rochester
Faulconer, Albert, Jr..........Rochester
Feldmann, Floyd M..........New York, N. Y.
Fergeson, James O..........Rochester
§ Ferris, DeWard O..........Rochester

Fieldman, E. Jay	Rochester
Figi, Fredk. A	Rochester
Fly, Orceneth A., Jr	Rochester
Foss, Edward Lehmann	Rochester
§ Fricke, Robert E	Rochester
†§Gambill, Carl M	Rochester
Gambill, Earl E	Rochester
Garrett, Charles M., Jr	Rochester
‡§Gastineau, Clifford F	New York, N. Y.
† Gatchell, Frank G	Rochester
Gaunt, William D	Rochester
Gee, Vernon Ray	Rochester
Geraci, Joseph Emil	Rochester
§ Ghormley, Ralph K	Rochester
†§Giffin, Herbert Z	Rochester
Giffin, Mary E	Rochester
§ Gifford, R. W., Jr	Silver Soring, Maryland
‡§Ginsberg, Robert L	Rochester
† Glew, William Bainbridge	Rochester
Goff, John R	Rochester
‡§Goldstein, Norman P	Bethesda, Md.
§ Good, C. Allen	Rochester
† Grace, Joseph B	Rochester
Grattan, Robert T	Rochester
§ Gray, Howard K	Rochester
Green, Paul A	Rochester
Greene, Laurence F	Rochester
§ Griffin, George D. J., Jr	Rochester
§ Grindlay, John H	Rochester
Groch, Sigmund N	Rochester
Grosh, John Livingston	Rochester
§ Gross, John B	Rochester
§ Habein, Harold C., Sr	Rochester
‡ Habein, Harold C., Jr	Wiesbaden, Germany
‡§Hagedorn, Albert Berner	New York, N. Y.
§ Haines, Samuel F	Rochester
§ Hallberg, Olav Erik	Rochester
†§Hallenbeck, Dorr F	Rochester
§ Hallenbeck, George A	Rochester
Hanlon, David G	Rochester
§ Hanson, Norbert Orrin	Rochester
† Hardy, William M	Rochester
Hare, Robert Lewis	Rochester
§ Harrington, Stuart W	Rochester
§ Harris, Lloyd E	Rochester
Hartman, Howard R	Rochester
§ Havens, Fred Z	Riverside, California
Hayles, Alvin Beasley	Rochester
Healy, John R	Rochester
Heck, Frank J	Rochester
† Heilman, Dorothy M. H	San Diego, California
§ Heilman, Fordyce R	Rochester
Heimark, John J	Rochester
*†Helland, Gustav M	Spring Grove
† Helland, John W	Spring Grove
†§Helmholz, Henry F	Rochester
†§Hempstead, Bert E	Rochester
Hench, Philip S	Rochester
§ Henderson, Edward D	Rochester
§ Henderson, John W	Rochester
Henderson, Lowell	Rochester
*†§Henderson, Melvin S	Rochester
† Henderson, Robert Earl	Rochester
Hepper, Norman G	Rochester
† Hewitt, Edith S	Rochester
†§Hewitt, Richard M	Rochester
†§Heyerdale, O. C	Rochester
§ Hill, John Roger	Rochester
† Hill, Richard Woolsey	Rochester
Hines, Edgar A., Jr	Rochester
Hodgson, Corrin H	Rochester
§ Hodgson, John R	Rochester
Holland, C. R	Rochester
Hollenhorst, Robert W	Rochester
§ Holman, Colin B	Rochester
§ Holt, Allen Howard	Rochester
Horton, Bayard T	Rochester
§ Howell, Llewelyn P	Rochester
§ Huizenga, Kenneth A	Rochester
§ Hunt, Arthur B	Rochester
§ Hunter, James S., Jr	Rochester
§ Ivins, John C	Rochester
§ Jackman, Raymond J	Rochester
Jacks, Quentin D	Rochester
Jackson, Richard L	Rochester
§ Janes, Joseph M	Rochester
Johnson, Adelaide McF	Rochester
§ Johnson, Carl Eric	Rochester
Johnson, Einer W., Jr	Rochester
Johnson, Ralph B	Lanesboro
Jordan, Stanley	Rochester
Joyce, George L	Rochester
† Judd. Edward Starr, Jr	Rochester
§ Juergens, John L	Rochester
Kearns, Thomas P	Rochester
‡ Keating, Francis Raymond Jr	Rochester
§ Keith, Haddow M	Rochester
†§Keith, Norman M	Rochester
§ Kennedy, Roger L. J	Rochester
‡ Kent, George B	Phoenix, Ariz.
§ Kernohan, James W	Rochester
Keys, John R	Rochester
§ Kiely, James Patrick	Rochester
‡ Kiely, Joseph M	Joliet, Illinois
§ Kierland, Robert R	Rochester
Kilby, Ralph A	Rochester
Kirby, Thomas J., Jr	Rochester
§ Kirklin, B. R	Rochester
§ Kirklin, John W	Rochester
Klakeg, Clayton H	Rochester
‡ Klass, Donald W	Rochester
† Knight, James Harry	Rochester
Knutson, Lewis A	Spring Grove
§ Koelsche, Giles A	Rochester
§ Kroboth, Frank J., Jr	Rochester
‡ Kroll, Harry G	Rochester
§ Krout, Robert M	Rochester
§ Krusen, Frank H	Rochester
† Kuhn, Arthur J	Rochester
Kunkel, Melvin G	Rochester
§ Kunkel, William M., Jr	Harrisburg, Pa.
§ Kvale, Walter F	Rochester
Lacy, Paul E	Rochester
§ Lake, Clifford F	Rochester
Law, William M	Rochester
Lazarte, Jorge A	Rochester
§ Leddy, Eugene T	Rochester
Lee, Philip R	Rochester
*†§Lemon, Willis S	Clifton Forge, Va.
Leuallen, Edmund C	Rochester
Lewis, Barton L	Rochester
§ Lick, Louis C	Rochester
†§Lillie, Harold I	Rochester
Lillie, John C	Rochester
§ Lipscomb, Paul R	Rochester
§ Litin, Edward M	Rochester
Lofgren, Karl A	Rochester
†§Logan, Archibald H	Rochester
§ Logan, George B	Rochester
† Lommel, Jerome G	Rochester
§ Love, J. Grafton	Rochester
Lundsten, Lloyd R	Rochester
§ Lundy, John S	Rochester
† Lynn, Thomas E	Rochester
McBean, J. B	Rochester
‡ McClure, Rensselaer W. Jr.,	Lawrence, Kansas
McConahey, William M., Jr.,	Rochester
McDonald, John R	Rochester
McFarlane, John A	Rochester
McKaig, Carle B	Pine Island
‡ McKee, Robert E	Bryan, Texas
McKibben, Helena	Rochester
§ MacCarty, Collin S	Rochester
†§MacCarty, William C	Rochester
† Madalin, Herbert E	Rochester
§ Madison, Mitchell S	Austin, Texas
§ Magath, Thomas B	Rochester
Manger, William M	Rochester
Mankin, Harold T	Rochester
Mankin, Haven W	Rochester
Mann, Frank D	Rochester
§ Martens, Theodore G	Rochester
§ Martin, George M	Rochester
§ Martin, William J	Rochester
Masson, Duncan M	Rochester
†§Masson, James C	Rochester
Masson, James K	Rochester
Mathieson, Don R	Rochester
Mayne, John G	Rochester
§ Mayo, Charles William	Rochester
Menges, Charles G. H	Rochester
§ Merritt, Wallace A	Rochester
† Meyerding, Henry W	Rochester
§ Miller, Roland D	Rochester
Miller, William J	Rochester
§ Mills, Steohen D	Rochester
Moersch, Frederick P	Rochester
§ Moersch, Herman J	Rochester
§ Montgomery, Hamilton	Rochester
Moore, Chris H	Rochester
Moores, Kenneth D	Rochester
§ Morlock, Carl G	Rochester
Mortensen. J. D	Rochester
Mulder, Donald W	Rochester
Mussey, Mary E	Rochester
† Mussey, Robert D	Rochester
Myers, Cortland III	Rochester
‡ Myers, Richard L	Rochester
Myers, Thomas T	Rochester
Myre, Theodore T	Rochester
Nehring, Jesse P	Preston
Neuman, Harold W	Rochester
Nichols, Donald R	Rochester
Norris, Neil T	Caledonia
‡ O'Keefe, Mathew E	Rochester
O'Leary, Paul A	Rochester
O'Neil, Richard L	Rochester
‡§O'Shaughnessy, Edward J	Camp Kilmer, N. J.
§ Odel, Howard M	Rochester
§ Olsen, Arthur M	Rochester
Olson, Ernest A	Pine Island
Olson, Grant E	West Concord
† Onifer, Theodore M	Rochester
Onsgard, L. Kenneth	Houston
Opfell, Richard W	Rochester
§ Osborn, John E	Miamisburg, Ohio
Owen, Charles A. J	Rochester
‡ Paris, Jaime	Rochester
Parker, Harry L	Rochester
§ Parker, Robert L	Rochester
Parkhill, Edith M	Rochester
Parkin, Thomas W	Rochester
‡§Parsons, William B., Jr	Coronado, Calif.
Patrick, Robert T	Rochester
§ Paulson, John A	Rochester
§ Paynter, Camen R	Great Falls, Montana
Peake, Charles O	Rochester
Pearce, Francis M., Jr	Rochester
Pease, Gertrude L	Rochester
Pemberton, Albert H	Rochester
† Pemberton, John	Rochester
§ Perry, Harold	Rochester
§ Peters, Gustavus A	Rochester
Petersen, Arthur B	Rochester
§ Petersen, Magnus C	Rochester
Peterson, Willard H	Spring Valley
Pettersen, George R	Mabel
Pettet, John R	Rochester
† Piper, Monte C	Rochester
Place, Virgil Alan	Rochester
Polley, Howard F	Rochester
Pool, Thomas L	Rochester
‡ Powelson, Myron H	Great Lakes, Illinois
* Powers, Fred H	Rochester
* Prangen, Avery DeH	Rochester
‡ Pratt, George F	San Francisco, California
§ Pratt, Joseph Hyde, Jr	Rochester
Price, Mary L	Rochester
§ Prickman, Louis E	Rochester
§ Priestley, James T	Rochester
§ Pruitt, Raymond D	Rochester
§ Pugh, David G	Rochester
Purnell, Don C	Rochester
§ Ralston, Donald E	Rochester
§ Randall, Lawrence M	Rochester
Randall, Raymond V	Rochester
§ Rasmussen, Waldemar C	Rochester
† Rawls, Thompson T	Rochester
† Reifsnyder, William Henry III	Rochester
Reitemeier, Richard J	Rochester
ReMine, William H., Jr	Rochester
Rice, Roberta G	Grand Island, Nebr.
Rigler, Robert G	Rochester
‡ Ringer, Merritt G., Jr	Rochester
‡ Ripepi, James D	New York, N. Y.
§ Risser, Alden F	Stewartville
† Rodenbaugh, Fredrich Hase,	Rochester
Rogne, William Gustav	Spring Grove
§ Rome, Howard P	Rochester
§ Rooke, E. D	Rochester
Rothwell, Walter S	Rochester
* Rotnem, Orville M	Iowa City, Iowa
Rovelstad, Randolph A	Rochester
§ Rucker, Charles W	Rochester
Ruggieri, Bartholomew A	Rochester
Rushton, Joseph G	Rochester
Ryan, Robert Frank	Rochester
§ Rynearson, Edward H	Rochester
Sabanas, Alvina O	Rochester
† Sabin, Frederick Chapman	Rochester
§ Salassa, Robert M	Rochester
†§Sanford, Arthur H	Rochester
Sauer, William G	Rochester
Sayre, George P	Rochester
§ Scheidel, Alois McK	Rochester
Scheifley, Charles H	Rochester
† Schirger, Alexander	Rochester
§ Schmidt, Herbert William	Rochester
Schwartz, Carl A	Rochester
Schwarz, Bert E	Rochester
Schweinfurth, J. D	Rochester
§ Scudamore, Harold H	Rochester
‡ Seay, James Elbert III	Rochester
† Selby, John B	Rochester
§ Seldon, Thomas H	Rochester
† Seybold, Herbert M	Rochester
Shick, Richard M	Rochester
Shields, Jack W	Rochester
Shocket, Everett	Rochester
Sibley, John Adams	Rochester
Siekert, R. G	Rochester
‡ Silver, Arthur W	Rochester

§ Simonton, Kinsey M. .... Rochester
Skaug, Harold M. .... Chatfield
† Skrdla, Willard B. .... Rochester
§ Slocumb, Charles H. .... Rochester
Smid, Arthur C. .... Rochester
†§Smith, Fredk. L. .... Rochester
§ Smith, Harry L. .... Rochester
§ Smith, Lucian A. .... Rochester
Smith, Meredith P. .... Rochester
Smith, Reginald A. .... Rochester
† Smith, Ross H., Jr. .... Rochester
Smith, William George .... Rochester
§ Soule, Edward H. .... Rochester
Spear, Harold C. ..... New Haven, Conn.
Spencer, Jean A. .... Rochester
Sperl, Michael P., Jr. .... Rochester
Spittel, John A., Jr. .... Rochester
Sprague, Randall G. .... Rochester
Starr, Grier F. .... Rochester
Stauffer, Maurice H. .... Rochester
† Stein, Harold A. .... Rochester
Steinhilber, Richard M. .... Rochester
† Stevens, Grant M. .... Rochester
§ Stickney, J. M. .... Rochester
§ Stilwell, George G. .... Rochester
Stillwell, George K. .... Rochester
§ Stroebel, Charles F., Jr. .... Rochester
Struthers, A. Morgan .... Rochester
Stuhler, Louis G. .... Rochester
Svien, Hendrik J. .... Rochester
Symmonds, Richard E. .... Rochester
Tani, George T. .... Rochester
Taub, Robert G. .... Rochester
Thompson, Gershom J. .... Rochester
Tihen, Edward N. .... Rochester
§ Tillisch, Jan H. .... Rochester
§ Uihlein, Alfred .... Rochester
Underdahl, Laurentius O. .... Rochester
Utne, John R. .... Rochester
‡§Utz, David C. .... Rochester
§ Van Herik, Martin .... Rochester
Van Patter, Ward .... Seattle, Wash.
§ Vaughn, Louis D. .... Rochester
Verby, John E. .... Rochester
Virnig, Hildegard J. .... Rochester
§ Wagener, Henry P. .... Rochester
Wagoner, James M. .... Harmony
Wakefield, Elmer G. .... Rochester
†§Wakim, Khalil G. .... Rochester
§ Walters, Waltman .... Rochester
‡ Wang, Jun-Chuan .... Honolulu, Hawaii
Ward, Bert B. .... Rochester
§ Ward, Louis E. .... Rochester
§ Watkins, Charles H. .... Rochester
Watson, Eleanor J. .... Rochester
Watson, John R. .... Rochester
Waud, Robert E. .... Rochester
§ Waugh, John M. .... Rochester
§ Weber, Harry M. .... Rochester
§ Weed, Lyle A. .... Rochester
† Weeks, Richard E. .... Rochester
Wehr, M. B. .... Rochester
Weiner, Alan D. .... Rochester
§ Weir, James F. .... Rochester
Welch, John S. .... Rochester
§ Wellman, William E. .... Rochester
Wellner, Theodore O. .... Rochester
§ Wente, Harold A. .... Rochester
§ Westrup, John E. .... Lanesboro
† Wilder, Russell M. .... Bethesda, Md.
‡ Wilkinson, George R., Jr. .... Rochester
Williams, George E. .... Rochester
Williams, Henry L. .... Rochester
*†Williams, Rasmus V. .... Rushford
§ Wilson, Robert B. .... Rochester
Wilson, Theodore W. .... Rochester
§ Wilson, V. O. .... Rochester
Winter, Malcolm D., Jr. .... Rochester
Wollaeger, Eric E. .... Rochester
§ Woltman, Henry Wm. F. .... Rochester
†§Wood, Harry G. .... Rochester
‡ Woodington, George F. .... Rochester
§ Woolner, Lewis B. .... Rochester
‡ Wuest, John H. .... Los Angeles, California
§ Young, Henry H. .... Rochester
Zimmer, J. F. .... Rochester

PARK REGION DISTRICT AND COUNTY MEDICAL SOCIETY

(Douglas, Grant, Otter Tail and Wilkin Counties)

Regular meeting, last Wednesday even numbered months
Annual meeting, December

Number of Members—64

President
Estrem, Robert D. .... Fergus Falls
Secretary
Shaver, Ward .... Fergus Falls

Arndt, Harry W. .... Detroit Lakes
Baker, Jeannette L. .... Fergus Falls
Baker, Norman H. .... Fergus Falls
† Blakey, Adam R. .... Osakis
§ Boline, Clifford A. .... Battle Lake
† Boysen, Peter .... Bemidji
§ Cain, James H. .... Hoffman
§ Carlson, Carl E. .... Alexandria
Clifford, George W. .... Alexandria
§ Combacker, Leon C. .... Fergus Falls
‡§Daehlin, Rolf .... Fergus Falls
§ DeKruif, Hendrik .... Fergus Falls
§ Doms, Vernon A. .... Elbow Lake
Emerson, Edwin E. .... Osakis
§ Estrem, Ralph L. .... Fergus Falls
§ Estrem, Robert D. .... Fergus Falls
§ Geiser, Peter M. .... Alexandria
§ Hamlon, John S. .... Fergus Falls
Hanson, Everett C. .... New York Mills
§ Hanson, LeRoy W. .... Pelican Rapids
† Harris, Evelyn S. .... Seattle, Wash.
Heegaard, William G. .... Alexandria
§ Heiberg, Emmett A. .... Fergus Falls
§ Helseth, Hovald K. .... Pelican Rapids
‡ Hom, Leong, Y. W. .... North Chicago, Illinois
§ Hunt, William .... Fergus Falls
§ Jacobson, Clifford W. .... Breckenridge
§ Kevern, Jay L. .... Henning
Kippen, Neil .... Breckenridge
§ Korda, Henry A. .... Pelican Rapids
Leibold, Herbert H. .... Parkers Prairie
§ Lewis, Arthur J. .... Henning
Lewis, Charles W. .... Henning
Love, Fredk. A. .... Carlos
§ Lund, Carl J. T. .... Fergus Falls
McDaniel, S. P. .... Fergus Falls
§ Mahowald, Aloys .... Fergus Falls
† Miller, William Anthony .... New York Mills
Mortensen, Nels G. .... Minneapolis
§ Mouritsen, Glenn J. .... Fergus Falls
† Naegeli, Frank .... Fergus Falls
§ Nelson, Roy A. .... Fergus Falls
§ Nelson, Wilburn O. B. .... Fergus Falls
§ O'Brien, Louis T. .... Breckenridge
Ostergaard, Erling .... Evansville
Palmerton, Ernest S. .... Fergus Falls
Parson, Lillian B. .... Elbow Lake
Parson, Lester R. .... Elbow Lake
† Patterson, William L. .... Fergus Falls
† Paulson, Theodore S. .... Fergus Falls
Rockwood, Philo H. .... Fergus Falls
§ Sanderson, David J. .... Fergus Falls
† Satersmoen, Theodore .... Pelican Rapids
§ Sather, Edgar R. .... Alexandria
§ Schamber, Walter F. .... Parkers Prairie
Schoeneberger, P. B. .... Perham
§ Shaver, Ward .... Fergus Falls
§ Sommerness, M. Duane .... Fergus Falls
§ Stemsrud, Harold L. .... Alexandria
Sutton, Harris R. .... Hoffman
§ Tanquist, Edwin J. .... Alexandria
Warner, James J. .... Perham
§ Wasson, Loren F. .... Alexandria
† Wray, William E. .... Campbell

RAMSEY COUNTY MEDICAL SOCIETY

Regular meetings, last Monday each month except June, July, August
Annual meeting, last Monday in January
Election in November

Number of Members—485

President
Flanagan, Harold F. .... St. Paul
Secretary
Gibbs, Edward C. .... St. Paul
Executive Secretary
Olson, Mrs. Eleanor .... St. Paul
§ Adair, Albert F., Jr. .... St. Paul
†§Ahrens, Albert E. .... St. Paul
Ahrens, Robert M. .... St. Paul
‡§Alden, J. F., Jr. .... St. Paul
§ Arnquist, Andrew S. .... St. Paul
§ Arny, Frederick P. .... St. Paul
§ Arzt, Philip K. .... St. Paul
§ Aurelius, J. Richards .... St. Paul
§ Ausman, Duane R. .... St. Paul
§ Babb, Frank S. .... St. Paul
§ Bacon, Donald K. .... St. Paul
§ Baer, Walter .... St. Paul
§ Balcome, Milton M. .... St. Paul
Barnett, Joseph M. .... St. Paul
‡ Baronofsky, Ivan D. .... St. Paul
† Barsness, Nellie O. N. .... St. Paul
Bauer, Eugene L. .... St. Paul
†§Beals, Hugh .... LaJolla, Calif.
Beck, Charles J. .... North St. Paul
§ Beech, Raymond H. .... St. Paul
§ Beck, Harvey O. .... St. Paul
§ Beer, John J. .... St. Paul
§ Bell, Charles C. .... St. Paul
§ Bellomo, James .... St. Paul
Bellomo, John .... St. Paul
§ Benepe, James L. .... St. Paul
† Bennion, P. H. .... Isway, Montana
§ Bentley, Norman P. .... St. Paul
Bernier, M. J. .... North St. Paul
§ Bernstein, William C. .... St. Paul
§ Bicek, Joseph F. .... St. Paul
† Binger, Henry E. .... Phoenix, Ariz.
Black, Earl J. .... St. Paul
†§Bock, Rolland A. .... St. Paul
† Boeckmann, Egil .... St. Paul
§ Bolender, Harold L. .... St. Paul
§ Bonnello, Frank J. .... St. Paul
§ Borg, Joseph F. .... St. Paul
† Bouma, Lewis R. .... St. Paul
Bouthilet, Florence J. .... St. Paul
§ Brand, George D. .... St. Paul
† Bray, Elwyn R. .... St. Paul
§ Briggs, John F. .... St. Paul
§ Broadie, Thomas E. .... St. Paul
§ Brodie, Walter D. .... St. Paul
† Brown, John C. .... Los Gatos, Calif.
Buckley, J. J. .... St. Paul
§ Bulinski, Theodore J. .... St. Paul
§ Burch, Edward P., II .... St. Paul
† Burch, Frank E. .... St. Paul
§ Burklund, Edwin D. .... St. Paul
§ Burlingame, David A. .... St. Paul
§ Burmeister, Richard O. .... St. Paul
†§Burns, Robert M. .... St. Paul
Burton, Carl G. .... St. Paul
§ Bush, Robert P. .... St. Paul
§ Busher, Herbert H. .... St. Paul
§ Cain, Clark L. .... St. Paul
Callahan, Francis F. .... St. Paul
Canine, James L. .... St. Paul
§ Carley, Walter A. .... St. Paul
*§Carroll, William C. .... St. Paul
Cedarleaf, Cherry B. .... St. Paul
§ Chadbourn, Charles R. .... St. Paul
§ Chatterton, Carl C. .... St. Paul
§ Christiansen, Andrew .... St. Paul
§ Clark, Henry B., Jr. .... Minneapolis
Cochrane, Byron B. .... St. Paul
Coddon, Walter D. .... St. Paul

Cohen, Ellis N. ....St. Paul
§ Colby, Woodard L. ....St. Paul
§ Cole, Wallace H. ....St. Paul
Coleman, John B. ....St. Paul
† Collie, Henry G. ....
St. Petersburg, Fla.
‡§Connolly, Coleman J. ....
Fort Belvoir, Va.
Connor, Charles E. ....St. Paul
§ Cook, Carroll K. ....St. Paul
§ Cooper, Charles C. ....St. Paul
§ Countryman, Roger S. ....St. Paul
§ Craig, David M. ....St. Paul
§ Critchfield, Lyman R. ....St. Paul
Crowley, James H. ....St. Paul
§ Crudo, Vincent D. ....St. Paul
§ Crump, James W. ....St. Paul
§ Culligan, John M. ....St. Paul
§ Culver, L. G. ....St. Paul

Davis, Edward V. ....St. Paul
Dawson, James R. ....St. Paul
Decker, Charles H. ....St. Paul
§ Derauf, Benjamin I. ....St. Paul
§ Deters, Donald C. ....St. Paul
† Dickson, Thomas H. ....St. Paul
§ Drake, Carl B. ....St. Paul
§ Dunn, James N. ....St. Paul

§ Earl, George A. ....St. Paul
§ Earl, John R. ....St. Paul
*§Edlund, Gustaf ....St. Paul
Edwards, Joseph W. ....St. Paul
Edwards, Lloyd G. ....St. Paul
§ Edwards, Thomas J. ....St. Paul
Eelkema, H. Harrison ....St. Paul
§ Eginton, Charles T. ....St. Paul
† Ely, Orriman S. ....So. St. Paul
Emerson, Edward C. ....St. Paul
§ Emmons, R. W. ....St. Paul
§ Endress, Edward K. ....St. Paul
§ Enroth, Oscar E. ....St. Paul
† Ernest, George C. H. ....
St. Petersburg, Fla.
§ Ersfeld, Murray P. ....St. Paul
† Eshelby, E. C. ....St. Paul
‡ Evert, John A., Jr. ....St. Paul

Farkas, John V. ....St. Paul
Fee, John G. ....St. Paul
Feinberg, Milton ....St. Paul
Felder, Davitt A. ....St. Paul
§ Felion, Arthur J. ....St. Paul
*†§Ferguson, James C. ....St. Paul
§ Fesler, Harold H. ....St. Paul
Field, Anthony H. ....Farmington
† Fifer, William J. ....St. Paul
Fink, Daniel L. ....St. Paul
§ Fisher, Dan W. ....St. Paul
§ Flanagan, Harold F. ....St. Paul
§ Flannery, Hubert F. ....St. Paul
Flom, R. P. ....St. Paul
§ Flynn, L. L., Jr. ....St. Paul
† Fogarty, Charles W., Sr. ....St. Paul
§ Fogarty, Charles W., Jr. ....St. Paul
§ Fogelberg, Emil J. ....St. Paul
§ Foley, Frederic E. B. ....St. Paul
Forsythe, James R. ....St. Paul
Fox, LeRoy J. ....St. Paul
§ Freeman, Charles D., Jr. ....St. Paul
Freeman, Gerald I. ....St. Paul
§ Freidman, Louis L. ....St. Paul
§ Fritz, Wallace L. ....St. Paul
§ Froats, Charles W. ....St. Paul
§ Fuller, Benjamin F. ....St. Paul

Galligan, John J. ....St. Paul
§ Garbrecht, Arthur W. ....St. Paul
§ Gardner, Walter P. ....St. Paul
Garrow, Douglas M. ....St. Paul
§ Gehlen, Joseph N. ....St. Paul
*§Ghent, Charles H. ....St. Paul
Gibbs, Edward C. ....St. Paul
§ Gillespie, Delmar R. ....St. Paul
‡ Gilsdorf, Donald A. ....St. Paul
§ Gleason, Wallace A. ....St. Paul
Goldsmith, Joseph W. ....St. Paul
†§Goltz, Edward V. ....St. Paul
§ Grant, Hendrie W. ....St. Paul
§ Gratzek, Thomas ....St. Paul
§ Grau, R. K. ....St. Paul
Gray, Edward F. ....White Bear Lake
*†Gruenhagen, Arnold P. ....St. Paul

Hagen, Paul S. ....St. Paul
Hakanson, Erick Y. ....St. Paul
§ Hall, Barnard ....St. Paul
§ Hammes, Ernest M., Sr. ....St. Paul
§ Hammes, Ernest M., Jr. ....St. Paul
*†Hammond, James F. ....St. Paul
Hannon, Donald W. ....St. Paul
§ Hanson, Harold B. ....St. Paul
Harbaugh, John T. ....St. Paul
§ Harmon, Gaius E. ....St. Paul
§ Hartfiel, William F. ....St. Paul
Hartig, Marjorie ....St. Paul

§ Hartley, Everett C. ....St. Paul
§ Hauser, Victor P. ....St. Paul
§ Hayes, Albert F. ....St. Paul
§ Heck, William W. ....St. Paul
Hedenstrom, Frank G. ....St. Paul
Heilig, William R. ....St. Paul
Henderson, A. J. G. ....No. St. Paul
§ Hengstler, William H. ....St. Paul
† Hensel, Charles N. ....St. Paul
Herman, Samuel M. ....St. Paul
§ Heron, Roy C. ....St. Paul
§ Herrmann, Edgar T. ....St. Paul
Hertz, Myron J. ....St. Paul
†§Hilger, Andrew W. ....St. Paul
§ Hilger, Jerome A. ....St. Paul
Hilger, Laurence D. ....St. Paul
Hilker, Marcus D. ....St. Paul
Hiniker, Louis P. ....St. Paul
§ Hochfilzer, John J. ....St. Paul
Hodgson, Jane E. ....St. Paul
† Holcomb, O. W. ....St. Paul
§ Hollinshead, W. H. ....St. Paul
§ Holmen, Robert W. ....St. Paul
§ Holt, John E. ....St. Paul
Hopkins, George W. ....St. Paul
Howard, Merrill A. ....St. Paul
Howard, Willard S. ....St. Paul
§ Howe, Newell W. ....St. Paul
§ Hullsiek, Harold E. ....St. Paul
§ Hullsiek, Richard B. ....St. Paul
§ Hunter, Murray H. ....Farmington
§ Hurwitz, Milton M. ....St. Paul

Ide, Arthur W. ....White Bear Lake
Ikeda, Kano ....St. Paul
† Ingerson, Carl A. ....St. Paul

Jackson, William C. ....St. Paul
§ James, Ellery M. ....St. Paul
James, John W. ....Mahtomedi
§ Janssen, Martin E. ....St. Paul
† Jarvis, Bruce W. ....St. Paul
Jarvis, Marilyn A. ....St. Paul
Jesion, Joseph W. ....St. Paul
§ Johanson, Waldemar G. ....St. Paul
§ Johnson, Carl E. ....St. Paul
Johnson, Carolyn A. ....St. Paul
§ Johnson, Herbert W. ....St. Paul
† Johnson, Morris B. ....St. Paul
Johnson, R. J. ....St. Paul
† Jones, E. Mendelssohn ....St. Paul

§ Kamman, Gordon R. ....St. Paul
Kaplan, David H. ....St. Paul
Karon, Irvin M. ....St. Paul
§ Kasper, Eugene M. ....St. Paul
Katz, Louis J. ....Long Beach, Calif.
Keefe, Rolland E. ....St. Paul
Kelly, James H. ....St. Paul
†§Kelly, John V. ....St. Paul
§ Kelsey, Chauncey M. ....St. Paul
§ Kenefick, Emmett V. ....St. Paul
§ Kenyon, Thomas J. ....St. Paul
§ Kesting, Herman ....St. Paul
† King, George L. ....Hudson, Wis.
†§Klein, Henry N. ....St. Paul
§ Knutson, Gerald E. ....St. Paul
Knutson, Robert C. ....St. Paul
Koza, Donald W. ....St. Paul
Krezowski, Thomas K. ....St. Paul
§ Kugler, Alex A. ....St. Paul
§ Kuske, Albert W. ....St. Paul
Kusske, Bradley W. ....St. Paul
§ Kusske, Douglas R. ....St. Paul
Kvitrud, Gilbert ....St. Paul

§ Lannin, Bernard G. ....St. Paul
§ Lannin, Donald R. ....St. Paul
Larrabee, Walter F. ....St. Paul
Larson, Eva-Jane ....St. Paul
Larson, James T. ....So. St. Paul
Larson, Kenneth R. ....St. Paul
Larson, Martin L. ....St. Paul
Lax, Morris H. ....St. Paul
†§Leahy, Bartholomew ....St. Paul
§ Leavenworth, R. O., Sr. ....St. Paul
Leavenworth, Richard O., Jr. ....St. Paul
§ Leick, Richard M. ....St. Paul
Leitch, Archibald ....St. Paul
§ Lepak, John A. ....St. Paul
† Lerche, William ....Cable, Wis.
§ Leven, N. Logan ....St. Paul
§ Leverenz, Carleton W. ....St. Paul
Levin, Bert G. ....St. Paul
Levitt, George X. ....St. Paul
§ Lick, Charles L. ....St. Paul
Lick, William J., Jr. ....St. Paul
§ Lien, Richard J. ....St. Paul
† Lightbourn, Edgar L. ....Hastings
Lilleberg, Norbert J. ....St. Paul
§ Lindell, Robert ....St. Paul
Lippman, Hyman S. ....St. Paul
§ Loken, Selmer M. ....St. Paul
Lowe, Earl R. ....So. St. Paul
Lowe, Thomas A. ....So. St. Paul
§ Lundholm, Arthur M. ....St. Paul

§ Lynch, Francis W. ....St. Paul
§ McCabe, James S. ....St. Paul
§ McCain, Donovan L. ....St. Paul
McCarthy, Joseph J. ....St. Paul
† McClanahan, James H. ....
White Bear Lake
McClanahan, Thomas S. ....
White Bear Lake
§ McCloud, Charles N. ....St. Paul
§ McEwan, Alexander ....St. Paul
§ McGroarty, Brian J. ....St. Paul
McKenzie, Eva Elaine ....St. Paul
§ McNeill, J. A. ....St. Paul
‡ Mackoff, Sam M. ....St. Paul
*§Madden, John F. ....St. Paul
Madland, Robert S. ....St. Paul
§ Malerich, J. Anthony ....St. Paul
§ Malerich, J. Anthony, Jr. ....St. Paul
Margolis, Philip M. ....St. Paul
Marks, Roger W. ....St. Paul
§ Martin, Dwight L. ....St. Paul
§ Martineau, Joseph L. ....St. Paul
Mateo, Guillermo ....St. Paul
Matthews, James H. ....Minneapolis
Mazzitello, William F. ....St. Paul
§ Meade, John R. ....St. Paul
§ Mears, Burtis J. ....St. Paul
§ Medelman, John P. ....St. Paul
Melancon, Joseph F. ....St. Paul
§ Menold, William F. ....St. Paul
Merner, Thomas B. ....Faribault
§ Merrick, Robert L. ....St. Paul
†§Meyerding, Edward A. ....St. Paul
Michienzi, Leonard J. ....St. Paul
Midboe, Gilbert ....St. Paul
Miller, Albert G. ....St. Paul
‡ Miller, William T. ....St. Paul
Miller, Z. R. ....St. Paul
§ Milnar, Frank J. ....St. Paul
Mintz, Charles M. ....St. Paul
§ Mishek, Charles J. ....St. Paul
§ Moga, John A. ....St. Paul
Molander, Herbert A. ....St. Paul
§ Monahan, Robert H. ....St. Paul
§ Mooney, Robert D. ....St. Paul
† Moquin, Marie A. ....St. Paul
† Moren, J. Adelaide ....White Bear Lake
Moriarty, Berenice ....St. Paul
Moriarty, Cecile R. ....St. Paul
§ Muller, Albrecht F. ....No. St. Paul
Mundahl, Harold R. ....St. Paul
§ Murphy, Jack T. ....St. Paul
§ Nash, Leo A. ....St. Paul
Neibergs, Lidija ....St. Paul
Neibergs, Pauls ....St. Paul
† Nelson, Louis A., Sr. ....St. Paul
Nelson, Louis A., Jr. ....St. Paul
Nimlos, Kenneth O. ....St. Paul
† Nimlos, Lenore O. ....St. Paul
§ Noble, John F. ....St. Paul
§ Noble, John L. ....St. Paul
† Nollet, Donald J. ....St. Paul
Nuebel, Charles J. ....St. Paul
Nye, Katherine A. ....St. Paul
Nye, Lillian L. ....St. Paul
O'Brien, J. C. ....St. Paul
† O'Connor, Loren J. ....St. Paul
O'Kane, Thomas W. ....St. Paul
§ O'Reilley, Bernard E. ....St. Paul
§ Ockuly, Orville ....St. Paul
§ Ogden, Warner ....St. Paul
§ Ohage, Justus ....St. Paul
§ Olsen, Ralph L. ....St. Paul
Olson, Charles A. ....St. Paul
§ Ostergren, Edward W. ....St. Paul
§ Ouellette, Alfred J. ....St. Paul
§ Owens, Frederick M., Jr. ....St. Paul
Paulson, Elmer C. ....Minneapolis
Paulson, Wallace J. ....Minneapolis
Pearson, Fritz R. ....St. Paul
§ Pearson, Malcolm M. ....St. Paul
Pedersen, Arthur H. ....St. Paul
Peltier, Leonard F. ....Minneapolis
Peterson, David B. ....St. Paul
§ Peterson, Donald H. ....St. Paul
§ Peterson, Harold O. ....St. Paul
Peterson, Joel L. E. ....St. Paul
Peterson, Roy L. ....White Bear Lake
†§Plondke, Fred J. ....St. Paul
§ Plotke, Harry L. ....St. Paul
Polski, Paul G. ....So. St. Paul
Quattlebaum, Frank ....St. Paul
§ Ralph, James R. ....St. Paul
† Ramsey, Walter R. ....St. Paul
Rasmussen, Ramby C. ....St. Paul
§ Ravits, Harold G. ....St. Paul
§ Rea, Charles E. ....St. Paul
Reid, James W. ....St. Paul
§ Reif, Robert W. ....White Bear Lake
Richards, Albert M. ....St. Paul
§ Richards, Ernest T. F. ....St. Paul
Richardson, Edward J., Jr. ....St. Paul
§ Richardson, Robert J. ....St. Paul

Rick, Paul F. W.	St. Paul
Rinkey, Eugene	St. Paul
§ Ritchie, Wallace P.	St. Paul
§ Ritt, Albert E.	St. Paul
Roach, Donald E.	St. Paul
§ Rogers, Sydney F.	St. Paul
† Rogin, Norton	St. Louis Park
Rolig, David H.	St. Paul
§ Rollie, Orris O.	St. Paul
§ Rosenthal, Robert	St. Paul
§ Roth, George C.	St. Paul
†§Rothschild, Harold J.	St. Paul
Rowe, Clarence J., Jr.	St. Paul
§ Roy, Phil. C.	St. Paul
† Ruhberg, George N.	Tarzona, Calif.
§ Rusterholz, Alan P.	St. Paul
§ Ryan, John J.	St. Paul
§ Ryan, Joseph M.	St. Paul
§ Sarnecki, M. M.	St. Paul
† Satterlund, Victor L.	St. Paul
†§Savage, Francis J.	St. Paul
§ Schmidtke, Reinhardt L.	St. Paul
† Schoch, Robert B. J.	St. Paul
§ Schons, Edward	St. Paul
§ Schroeckenstein, Hugo F.	St. Paul
†§Schuldt, Fredk. C.	St. Paul
†§Schulze, Albert G.	St. Paul
§ Schwyzer, Hanns C.	St. Paul
Schwyzer, Marguerite	St. Paul
§ Scott, Eugene E.	St. Paul
Sekhon, Mohan S.	St. Paul
Sells, Richard J.	No. St. Paul
† Senkler, George E.	St. Paul
§ Setzer, Hobert J.	St. Paul
Shannon, William R.	St. Paul
† Shellman, John L.	Pacific Palisades, Calif.
Short, Jacob	St. Paul
Siegel, Clarence	St. Paul
§ Simons, Leander T.	St. Paul
Singer, Benjamin J.	St. Paul
§ Skinner, Abbott	St. Paul
† Skinner, Harvey O.	St. Paul
§ Smisek, Elmer A.	St. Paul
§ Smith, Vernon D. E.	St. Paul
§ Snyder, George Wm.	St. Paul
§ Sohlberg, Olof I.	St. Paul
Sommerdorf, Vernon L.	St. Paul
Sommers, Ben	St. Paul
§ Sorem, Milton B.	St. Paul
Soucheray, Philip H.	St. Paul
§ Souster, Benjamin B.	St. Paul
§ Sowada, Ernest J.	St. Paul
‡ Sprafka, Gregory	New York, N. Y.
†§Sprafka, Joseph L.	St. Paul
§ Sprafka, Joseph M.	St. Paul
§ Steinberg, Charles L.	St. Paul
§ Sterner, Donald C.	St. Paul
*†§Sterner, Ernest G.	St. Paul
§ Sterner, E. R.	St. Paul
§ Sterner, John J.	St. Paul
Stewart, Alexander	St. Paul
§ Stolpestad, Armer H.	St. Paul
§ Stolpestad, Herbert L.	St. Paul
§ Strand, Jack W.	St. Paul
§ Strate, Gordon E.	St. Paul
Straus, M. L.	St. Paul
§ Strem, Edward L.	St. Paul
§ Sturley, Rodney F.	St. Paul
† Swanson, John A.	St. Paul
Swanson, Lawrence J.	St. Paul
§ Swendson, James J.	St. Paul
Swenson, Donald B.	St. Paul
§ Teisberg, John E.	St. Paul
Thompson, Floyd A.	St. Paul
Thoreson, M. C. Bernice	So. St. Paul
§ Tifft, Cyril R.	St. Paul
§ Tongen, Lyle A.	St. Paul
§ Tracht, Robert R.	St. Paul
Travis, James S.	St. Paul
§ Tregilgas, Harold R.	So. St. Paul
Tregilgas, Richard B.	St. Paul
‡ Tucker, Jolyn S.	Alabama
Ubel, Frank A.	St. Paul
Van Bergen, Frederick H.	Minneapolis
Varco, Richard L.	St. Paul
Veirs, Dean M.	St. Paul
Veirs, Ruby J. S.	St. Paul
§ Venables, Alexander E.	St. Paul
‡§von Amerongen, W. W.	St. Paul
§ Waas, Charles W.	St. Paul
§ Walker, Arthur E.	St. Paul
Wall, James O.	St. Paul
‡§Wallinga, Jack	Oakland, Calif.
§ Walsh, Edward F.	St. Paul
§ Walter, Clarence W.	St. Paul
Warren, Cecil A.	St. Paul
Watson, P. Theodore	St. Paul
§ Watson, William H. A.	St. Paul
§ Watson, William J.	Newport
Watz, Clarence E.	St. Paul
§ Webber, Fred L.	St. Paul
§ Weis, Benjamin A.	St. Paul
§ Weisberg, Maurice	St. Paul
§ Wenzel, Gilbert P.	St. Paul
Westover, D. E.	St. Paul
Wetzel, Earl V.	St. Paul
*†Wheeler, Merritt W.	Stillwater
† Wilkinson, Stella L.	St. Paul
† Williams, Arthur B.	St. Paul
§ Williams, Clayton K.	St. Paul
§ Williams, John A.	St. Paul
Williams, Richard A.	St. Paul Park
Wilson, J. Allen	St. Paul
§ Wilson, James V.	St. Paul
† Winnick, Joseph B.	St. Paul
Witthaus, Melvyn E.	Mahtomedi
§ Wolff, Herman J.	St. Paul
Wolkoff, H. J.	St. Paul
Word, Harlan L.	St. Paul
‡ Yamamoto, Joe	Fort Lewis, Wash.
Youngren, Everett R.	St. Paul
Zachman, Leo L.	St. Paul
Zagaria, James F.	St. Paul
§ Zimmermann, Harry B.	St. Paul

RED RIVER VALLEY MEDICAL SOCIETY

(Kittson, Mahnomen, Marshall, Norman, Pennington, Polk, Red Lake and Roseau Counties)
Regular meeting—four each year
Annual meeting, December or January
Election in January

Number of Members—61

President	
THYSELL, HAROLD R.	Crookston
Secretary	
SATHER, RUSSELL O.	Crookston
*†Adkins, C. M.	Thief River Falls
§ Anderson, Wallace E.	Clearbrook
§ Behr, Orlo K.	Crookston
§ Berge, David O.	Roseau
§ Berlin, Anthony S.	Hallock
† Biedermann, Jacob	Thief River Falls
§ Boyer, George S.	Crookston
† Bratrud, Edward	Thief River Falls
Brink, Adlai A.	Baudette
§ Cameron, John H.	Crookston
§ Clapp, Hubert D.	Crookston
§ Covey, Kenneth W.	Mahnomen
§ Dale, Lester N.	Red Lake Falls
†§Delmore, John L.	Roseau
§ Delmore, J. L., Jr.	Roseau
§ Downing, William	Crookston
§ Erickson, Eskil	Halstad
§ Feigal, William M.	Thief River Falls
Flancher, Leon H.	Crookston
§ Greene, Daniel E.	Thief River Falls
§ Haugan, Norman R.	Crookston
*†§Henney, William H.	McIntosh
§ Hirsh, Stanton A.	Crookston
† Hollands, William H.	Fisher
§ Holmstrom Carle H.	Warren
Janecky, Allen G.	Baudette
§ Kinkade, Byron R.	Ada
*†Kirk, George P.	East Grand Forks
§ Klefstad, Lloyd H.	Greenbush
§ Kostick, William R.	Fertile
§ Loken, Theodore	Ada
† Lund, Anthony J.	Cleveland, Ohio
Lynde, Orrin G.	Los Gatos, Calif.
§ McDonald, William J.	Crookston
McHardy, Bryson R.	Karlstad
§ McKaig, Alan M.	Red Lake Falls
§ Martin, George B.	Thief River Falls
§ Mercil, William	Crookston
§ Mueller, Donald R.	Bagley
§ Murn, Thomas G.	Thief River Falls
† Nelson, Henry E.	Crookston
Nelson, Kenneth L.	Warroad
*§Norman, John F.	Crookston
§ Oppegaard, C. L.	Crookston
Parker, Philip J.	Hallock
§ Pumala, Erven E.	Warren
§ Reff, Alan R.	Crookston
§ Roholt, Christian	McIntosh
‡ Rydland, Arne D.	Foley, Alabama
Sather, Edgar L.	Fosston
§ Sather, George A.	Fosston
§ Sather, Richard N.	Fosston
§ Sather, Russell O.	Crookston
§ Schossow, George W.	Erskine
§ Skogerboe, Rudolph	Karlstad
§ Stadem, Clifford	Twin Valley
§ Starekow, Milton	Thief River Falls
Stensgaard, Kermit L.	Thief River Falls
§ Stewart, Donald	Crookston
§ Thysell, Harold R.	Crookston
§ Uhley, Charles G.	Crookston
§ Van Rooy, George T.	Thief River Falls
Wendt, H. Paul	Thief River Falls
§ Wikoff, Howard M.	Crookston
† Wiltrout, Irving G.	Oslo

RENVILLE-REDWOOD COUNTY MEDICAL SOCIETY

Regular meeting, third Tuesday of each month
Annual meeting, November

Number of Members—24

President	
PIERCE, ROBERT B.	Olivia
Secretary	
DIESSNER, ARDELL W.	Redwood Falls
§ Alcorn, William J.	Wabasso
§ Anderson, Chester A.	Hector
Bessesen, Daniel H.	Olivia
§ Billings, Ralph E.	Franklin
† Brand, William A.	Redwood Falls
§ Ceplecha, Stanley F.	Redwood Falls
§ Cosgriff, James A., Sr.	Olivia
Cosgriff, James A., Jr.	Olivia
§ Diessner, Ardell W.	Redwood Falls
§ Dordal, John	Sacred Heart
§ Fawcett, Arthur M.	Renville
† Flinn, Thomas E.	Remer
‡ Furr, Leo O.	Bird Island
§ Haas, J. F.	Fairfax
§ Hinderaker, Harris P.	Bird Island
§ Johnson, William E.	Morgan
§ Knoche, Harvey A.	Morgan
§ Lenz, Joseph R.	Morton
† Mesker, George H.	Olivia
Metz, Donald D.	Buffalo Lake
§ Nelson, Glenn E.	Fairfax
§ Pierce, Robert B.	Olivia
† Preisinger, Joseph W.	Dallas, Texas
§ Strauchler, Jonas	Belview

RICE COUNTY MEDICAL SOCIETY

Regular meeting, third Tuesday of each month
Annual meeting, third Tuesday in October
Number of Members—33

President
MEYER, PAUL F........Faribault
Secretary
NIELSEN, ALVIN M........Northfield

§ Beaton, J. Gordon........Northfield
§ Bruhl, Heinz H........Faribault
†§Dungay, Neil S........Northfield
§ Engberg, Edward J........Faribault
Francis, David W........Morristown
§ Furlow, William L........Faribault
†§Hanson, Adolph M........Faribault
† Huxley, Fredk. R........Faribault
§ Kennedy, George L........Faribault
§ Kolars, James J........Faribault
‡ Kucera, Louis B...Colo. Springs, Colo.
§ Lende, Norman........Faribault
† Lexa, F. J........Lonsdale
§ Mears, Robert F........Northfield
Meyer, Fred'k C........Kenyon
Meyer, Paul F........Faribault
‡ Meyer, Robert P........Faribault
§ Moses, Royal R........Kenyon
§ Nelson, Ernest J........Lonsdale
§ Nielsen, Alvin M........Northfield
§ Nuetzman, Arthur W........Faribault
§ Orr, Burton A........Faribault
Peterson, D. H........Northfield
§ Roberts, Stanley E........Faribault
§ Robilliard, Charles M........Faribault
Rumpf, Carl W........Faribault
§ Rysgaard, George M........Northfield
§ Stevenson, Frank W........Faribault
§ Street, Bernard........Northfield
Studer, Donald J........Faribault
§ Traeger, Carl A........Faribault
§ Weaver, Paul H........Faribault
§ Wilson, Warren E........Northfield

ST. LOUIS COUNTY MEDICAL SOCIETY

(Carlton, Cook, Itasca, Lake and St. Louis Counties)

Regular meeting, second Thursday except July and August
Annual meeting, second Thursday in January
Election, second Thursday in December
Number of Members—279

President
FAWCETT, KEITH R........Duluth
Secretary
HAAVIK, JOHN E........Duluth
Exec. Secretary
Gilbert, Mrs. Margaret B........Duluth

§ Abraham, Arden L........Duluth
§ Adams, Bertram S........Hibbing
Addy, Edward R........Gilbert
Ahola, Kenneth E........Hibbing
§ Ahrens, Curtis F........Duluth
Andrew, William F........Duluth
Antonow, Arthur M........Virginia
Arko, Joseph L........Hibbing
Asta, Joseph J........Duluth
§ Athens, Alvin G........Duluth
§ Aufderheide, Arthur........Duluth
§ Backus, Reno W........Nopeming
§ Bagley, Charles M........Duluth
Bagley, Elizabeth C........Duluth
§ Bagley, William R........Duluth
Baich, Velemir M........Coleraine
§ Bakkila, Henry E........Duluth
§ Bardon, Richard........Duluth
§ Barker, John D........Duluth
Barnes, Richard E........Aurora
† Barney, Leon A........Duluth
§ Barrett, Earl E........Duluth
Bartzen, Peter J........Duluth
§ Becker, Fredk. T........Duluth
§ Benell, Otto E........Virginia
Bepko, Marie K........Cloquet
Berdez, George L........Duluth
§ Bergan, R. O........Duluth
§ Bianco, Anthony J., Sr........Duluth
†§Bianco, Anthony J., Jr........Rochester
† Binet, Henry E........Grand Rapids
§ Blackmore, Sidney C........Biwabik
§ Bloom, Joseph........Duluth
‡ Bolz, J. Arnold......Camp Carson, Colo.
Boman, Paul G........Duluth
Bonner, John L........Eveleth
Bouchelle, McLeomore........Virginia
Bowen, Robert L........Hibbing
† Boyer, Sam H., Sr........Duluth
§ Boyer, Sam H., Jr........Duluth
§ Braun, Ohrmundt C........Grand Rapids
§ Bray, Philip N........Duluth
Brockway, Roger W........Grand Rapids
§ Brooker, Warren J........Duluth
§ Buckley, Robert Peers........Duluth
Burleigh, Edward G........Eveleth
§ Butler, John K........Cloquet
Cantwell, William F...International Falls
* Chapman, Theodore L........Duluth
§ Chermak, Francis G...International Falls
§ Christensen, Clarence H........Duluth
Ciriacy, Edward W........Ely
§ Clark, Clarence L........Duluth
Clark, Elizabeth A........Duluth
§ Clark, Ivan T........Duluth
§ Coll, James J........Duluth
† Collins, Arthur N........Duluth
Colosey, Frederick J........Ely
‡§Conley, Francis W........Ft. Hood, Texas
† Cope, Hershel E........Minneapolis
§ Coventry, William D........Duluth
Cowan, George M........Duluth
Detjen, Edward D........Bigfork
§ Dickson, Franklin H., Jr........Proctor
Doxsee, George C........Chisholm
Doyle, George C........Duluth
§ Dwyer, John J........Duluth
§ Eckman, Philip F........Duluth
§ Eckman, Ralph J........Duluth
† Ekblad, John Wm........Duluth
§ Eklund, Carl D........Duluth
§ Elias, Frank J........Duluth
§ Emanuel, Karl Wm........Duluth
Eppard, Raymond M........Cloquet
Erickson, George P........Hibbing
Erickson, Vernon D........Grand Rapids
Erskine, Gordon N........Grand Rapids
Evensta, John B........Grand Rapids
Farley, Frank G........Hibbing
§ Fawcett, Keith R........Duluth
§ Fellows, Manley F........Duluth
‡ Ferrell, Clarence R........Grand Rapids
§ Feuling, John C........Duluth
Fifield, Malcolm........Duluth
§ Fischer, Mario McC........Duluth
§ Fisketti, Henry........Duluth
§ Flynn, Bernard F........Hibbing
§ Fredericks, Merriam G........Duluth
French, Bayard T........Hibbing
§ Fuller, Josiah........Duluth
§ Gillespie, Malcolm G........Duluth
§ Goldish, Daniel R........Duluth
§ Goldish, Robert J........Duluth
Goodman, Charles........Virginia
§ Gowan, Lawrence R........Duluth
† Graham, Archibald W........Chisholm
§ Grahek, Jack P........Ely
Granquist, Richard D........Chisholm
§ Grinley, Andrew........Grand Rapids
Grohs, William H........Duluth
§ Haavik, John E........Duluth
Halbert, John J........Duluth
† Halliday, P. V........Minneapolis
Halper, Bernard........Hibbing
† Haney, Claude L........Duluth
Hansen, Robert F........Hibbing
Hanson, Ernest O........Carlton
Harrington, Vernon A........Duluth
§ Harris, Carl N........Hibbing
†§Hatch, Walter E........Duluth
*†Hayes, Michael F........Moose Lake
§ Hedberg, Gustaf A........Nopeming
Heiam, William C........Cook
§ Hilding, Anderson C........Duluth
† Hill, Fredk. E........Riverside Calif.
Hill, John P........Virginia
Hirschhoeck, Frank J........Duluth
§ Hoff, Herbert O........Duluth
Hoover, Norman W........Virginia
§ Houkom, Samuel S........Duluth
Hult, John E........Cloquet
Hutchinson, Henry........Moose Lake
‡ Indihar, John E........Oakland, Calif.
Ireland, Gerald W........Nashwauk
Irwin, Homer R........Hibbing
§ Jacobson, Clarence........Chisholm
§ Jacobson, Ferdinand C........Duluth
§ Jensen, Thorvold J........Duluth
§ Jeronimus, Henry J........Duluth
Jessico, Charles M........Duluth
Joffe, Harold H........Virginia
Johnsen, Henry A., Jr........Virginia
‡ Johnson, Edward A........Virginia
§ Johnson, Karl E........Duluth
§ Johnsrud, Luverne W........Duluth
Johnston, Henry W........Virginia
†§Johnston, Rufus O........Hibbing
§ Jolin, Francis M........Bovey
Juntunen, Roy R........Duluth
§ Kelley, Walter........Duluth
§ Kelly, Albert C........Duluth
Kelly, Robert T........Nashwauk
§ Klein, Harry........Duluth
§ Klein, William A........Duluth
§ Knapp, Frank N........Duluth
§ Knoll, W. V........Duluth
§ Kohlbry, Carl O........Duluth
Koskela, Lauri E...Shaker Heights, Ohio
§ Kotchevar, Frank R........Eveleth
Kozberg, Oscar........Moose Lake
§ Krueger, Victor R........Nopeming
§ LaBree, Robert H........Duluth
†§Laird, Arthur T........Duluth
§ Latterell, Kenneth E........Duluth
Law, Harrison E........Virginia
‡ Leek, Joseph H........Augusta, Ga.
Lepak, Francis J........Duluth
Leppo, N. Erkki A........Duluth
Lipinski, Stanley W........Hibbing
§ Litman, Samuel N........Duluth
*‡Lovshin, William C.....Tomahawk, Wis.
† McCarty, Paul D........Ely
† McCoy, Mary K........Duluth
† McDonald, Archibald........Duluth
§ McDonald, Owen G........Duluth
§ McHaffie, Orval L........Duluth
§ McKenna, Maurice J........Grand Rapids
§ McNutt, John R........Duluth
*§MacFarlane, P. H........Chisholm
§ MacRae, Gordon C........Duluth
§ Magney, Fredolph H........Duluth
§ Malmstrom, John A........Virginia
† Marcley, Walter J........Minneapolis
Marrone, Patrick H........Duluth
§ Martin, William B........Duluth
§ Martin, Webster C........Duluth
§ Mayne, Roy M........Nopeming
Mead, Charles H........Duluth
§ Merriman, Lloyd L........Duluth
Miettunen, John B........Chisholm
Moe, Thomas........Moose Lake
§ Moehring, Henry G........Duluth
Mollers, Theodore P........Soudan
Monroe, Paul B........Cloquet
§ Monserud, Nels O........Cloquet
§ Morsman, L. William........Hibbing
§ Moyer, John B........Duluth
Munson, Martin S........Barnum
Murray, Robert A........Hibbing
Nakamura, James Y........Deer River
§ Neff, Walter S........Virginia
§ Nelson, Robert L........Duluth
§ Nicholson, Murdoch A........Duluth
§ Nisius, George F........Duluth
§ Norberg, Carl E........Cloquet
§ Nutting, Roland E........Duluth
§ O'Neill, John C........Duluth
Olson, Albert E........Duluth
§ Olson, Archibald........Duluth
Owens, Ben P........Hibbing
§ Papermaster, Ralph........Two Harbors

*†Parker, Owen W.........Minneapolis
§ Parker, Wilbert H.........Chisholm
§ Parson, E. Irvine.........Duluth
§ Pasek, Antone W.........Cloquet
§ Patch, Orien B.........Duluth
† Pearsall, R. P.........Virginia
§ Pedersen, Roy C.........Duluth
† Pennie, Daniel F.........Duluth
Peterson, Edward N.........Virginia
§ Peterson, John H.........Duluth
§ Pierce, Jack R.........Virginia
Power, John E.........Duluth
Power, John E., Jr.........Duluth
Puumala, Reino H.........Cloquet
Raadquist, Charles S.........Hibbing
Raattama, John.........Keewatin
Rajala, Arnold I.........Grand Rapids
Reed, Paul.........Virginia
Richter, David J.........Virginia
§ Rokala, Henry E.........Virginia
*†§Rood, Dana C.....Santa Barbara, Calif.
Rowe, Olin W.........Duluth
Rowles, Everett K.........Coleraine
§ Rudie, Peter S.........Duluth
Rudie, William A.........Duluth
§ Runquist, John M.........Duluth
Ryan William J.........Duluth
§ Sach-Rowitz, Alvan.........Moose Lake

§ Salter, Reginald A.........Virginia
† Sanford, John B.........Chicago, Ill.
§ Sarff, Oliver E.........Duluth
Sax, Milton H.........Duluth
Sax, Simon G.........Duluth
Schirber, Martin J.........Grand Rapids
§ Schmid, John F.........Duluth
§ Schneider, Laurence E.........Duluth
† Schroder, C. H.........Pottstown, Penn.
Schweiger, Theodore R.........Hibbing
§ Seashore, R. T.........Duluth
§ Sher, David A.........Virginia
Shirai, Shohei.........Coleraine
§ Siegel, John S.........Virginia
§ Sinamark, Andrew.........Hibbing
Sisler, Clifford E.........Grand Rapids
§ Smith, Cyril M.........Duluth
Smith, Wallace R.........Grand Marais
Snyker, Omer E.........Ely
§ Spang, Anthony J.........Duluth
§ Spang, James S.........Duluth
§ Spang, William M.........Duluth
Stein, William A.........Ely
Storsteen, Kenneth A.........Duluth
§ Strandjord, Nels M.........Virginia
†§Strathern, Moses L.........Gilbert
§ Strauss, Eugene C.........Duluth
§ Strewler, Gordon J.........Duluth

§ Strobel, William G.........Duluth
§ Sutherland, Harry N.........Ely
§ Swedberg, William A.........Duluth
§ Swenson, Arnold O.........Duluth
§ Teich, Kenneth W.........Duluth
§ Terrell, Bernard J.........Nopeming
§ Tetlie, James P.........Duluth
Thomas, John V.........Duluth
Tingdale, Carlyle.........Hibbing
Tomhave, Wesley G.........Hibbing
§ Tosseland, Noel E.........Duluth
Tuohy, Edward L.........Duluth
Urberg, Sofus E.........Duluth
Van Ryzin, Donald J.........Duluth
§ Walder, Harold J.........Duluth
† Walker, A. E.........St. Paul
§ Wallace, Martin O.........Duluth
Walter, Frederick H...International Falls
§ Wells, Arthur H.........Duluth
§ Wheeler, Daniel W.........Duluth
‡§Williams, Bruce F. P...Fort Knox, Ky.
Winter, John A.........Duluth
Wolff, John M.........Duluth
†§Ylitalo, William H.........Rochester
§ Young, Thomas O.........Duluth
Zemmers, Roberts.........Duluth
Zick, Luther H.........Grand Rapids
§ Zupanc, Edward.........Duluth

SCOTT-CARVER COUNTY MEDICAL SOCIETY

Regular meeting, second Wednesday of every month
Annual meeting, second Wednesday in June
Number of Members—32

President
Ponterio, James E.........Shakopee
Secretary
Rieschl, Elizabeth K.........Jordan

Bratholdt, James W.........Watertown
† Buck, Fredk. H.........Shakopee
§ Cervenka, Charles F.........New Prague
Clarke, John W.........Watertown
§ Doherty, Elmer M.........New Prague
§ Hebeisen, Milton B.........Chaska
Heinz, Ivy B.........Shakopee
Heinz, Lawrence H.........Shakopee

§ Juergens, Herman M.........Belle Plaine
§ Kucera, Stanley T.........Northfield
Larson, Leighton W.........Waconia
Lukk, Olaf.........Montgomery
†§Martin, Thomas Philip.........Arlington
Nagel, Harold D.........Minneapolis
Navratil, Donald R.........Baltimore, Md.
Ninneman, Newton N.........Waconia
† Novak, Edward E.........New Prague
Olson, Chester J.........Belle Plaine
§ Pearson, Bror F.........Shakopee
† Pearson, Roy T.........Shakopee
Philp, David R.........Watertown

Pogue, Richard E.........Watertown
§ Ponterio, James E.........Shakopee
Rieschl, Elizabeth K.........Jordan
§ Rynda, Edwin R.........New Prague
*†Sanford, James A.........Savage
Sawaryniuk, Iwan.........Waconia
Schimelpfenig, George T.........Chaska
Simmonds, Harry N.........Prior Lake
§ Simons, Bernard H.........Chaska
Stahler, Paul A.........Jordan
†§Westerman, Alvin.........Montgomery
Westerman, Fred.........Montgomery

SOUTHWESTERN MINNESOTA MEDICAL SOCIETY

(Cottonwood, Jackson, Murray, Nobles, Pipestone and Rock Counties)
Regular meetings, on call
Annual meeting, October
Election, October
Number of Members—62

President
Hallin, Roger P.........Worthington
Secretary
Heiberg, Olaf M.........Worthington

§ Arnold, Elmer W.........Adrian
§ Bader, J. L.........Slayton
§ Basinger, Harold P.........Windom
Basinger, Harvey R.........Mountain Lake
§ Basinger, Homer P.........Windom
§ Beckering, Gerrit.........Edgerton
§ Benjamin, Walter G.........Pipestone
§ Bofenkamp, Ferdinand W.........Luverne
§ Boone, Ervin S.........Luverne
†§Bouma, John H.........Topeka, Kansas
† Brown, Alexander H.........Pipestone
‡ Buresh, Kenneth L.........Aurora, Colo.
§ Carlson, John V.........Westbrook
§ Christiansen, Harold A.........Jackson
§ Dawson, Lorin D.........Worthington
*†De Boer, Hermanus.........Edgerton
Dokken, James H.........Windom
Doman, Victor W.........Lakefield

§ Hallin, Roger P.........Worthington
§ Halloran, Walter H.........Jackson
§ Halpern, David J.........Brewster
Harada, Thomas T.........Lake Wilson
§ Harrison, Percy W.........Worthington
§ Heiberg, Olaf M.........Worthington
§ Hoyer, Ludolf J.........Windom
§ Karleen, Bernard N.........Jackson
§ Keyes, Robert W.........Pipestone
§ Kilbride, Edwin A.........Worthington
§ Koenecke, Fred H., Jr.........Lakefield
§ Kotval, Russell J.........Pipestone
§ Laikola, Leslie A.........Adrian
§ Lohmann, John G.........Pipestone
§ Maitland, Edwin T.........Jackson
† Manson, Frank M.........Worthington
§ Martin, Albert C.........Luverne
§ Minge, Raymond K.........Worthington
§ Nealy, Donald E.........Adrian
§ Nicholson, Richard W.........Heron Lake
§ Nywall, Dean D.........Slayton
§ Odland, Donald M.........Luverne

§ Pankratz, Peter J.........Mountain Lake
§ Patterson, Hugh D.........Slayton
§ Pierson, Roy F.........Slayton
§ Piper, William A.........Mountain Lake
Plucker, Milton W.........Worthington
† Ritzinger, Fredk. R.........Lakefield
§ Robinett, Robert W.........Worthington
§ Rohrer, Christian A.........Worthington
§ Rose, John T.........Lakefield
§ Sawtell, Robert R.........Worthington
§ Schade, Fredk. L.........Worthington
§ Schutz, Elmer S.........Mountain Lake
§ Sherman, Charles L.........Luverne
§ Slater, Sidney A.........Worthington
§ Sogge, Ludwig L.........Windom
§ Stam, John.........Worthington
§ Stanley, Court R.........Worthington
Stevenson, Basil M.........Fulda
§ Stratte, Harold C.........Windom
§ Wells, Walter B.........Jackson
§ Williams, Charles A.........Pipestone
† Williams, Leon A.........Minneapolis
‡§Zeller, Nicholas H.........Worthington

STEARNS-BENTON COUNTY MEDICAL SOCIETY

Regular meeting, third Thursday of month
Election in December
Annual meeting, December
Number of Members—69

President
Reif, Henry J.........St. Cloud
Secretary
Coseriu, Vasile G.........St. Cloud

Alden, W. Charles.........Kimball
§ Anderson, E. M.........St. Cloud

Andrews, Bernice F.........Holdingford
§ Autrey, William A.........St. Cloud
§ Baumgartner, Florian H.........Albany
§ Beuning, John B.........St. Cloud
† Brigham, Charles F., Sr.........St. Cloud
§ Brigham, Charles F., Jr.........St. Cloud

† Broker, Henry M.........Minneapolis
§ Buscher, Julius C.........St. Cloud
‡§Cesnik, Robert J.........St. Cloud
§ Clark, Harry B.........St. Cloud
Cleaves, William D.........Sauk Centre
§ Coseriu, Vasile G.........St. Cloud

DeWeese, Robert C. ..... St. Cloud
§ Donaldson, Charles S. ..... St. Cloud
§ Dredge, Thomas E. ..... St. Cloud
DuBois, Julian F., Sr. ..... Sauk Centre
DuBois, Julian F., Jr. ..... Sauk Centre
§ Evans, Leslie ..... Sauk Rapids
‡ Fidelman, Norman E. ..... Foley
§ Fleming, Thomas N. ..... St. Cloud
§ Gaida, Joseph B. ..... St. Cloud
§ Goehrs, Gilman H. ..... St. Cloud
† Goehrs, Henry W. ..... St. Cloud
Grant, John C. ..... Sauk Centre
† Haberman, Emil ..... Osakis
Halenbeck, Philip L. ..... St. Cloud
Henry, Clarence J. ..... Milaca
‡ Henry, J. E. ..... Milaca
Hoehn, David ..... Holdingford
Johnson, Marvin W. ..... Milaca
§ Jones, Richard N. ..... St. Cloud
Keith, Paul J. ..... Milaca
§ Kelly, John F. ..... Cold Spring
‡ Koenig, Robert P. ..... Geneva, N. Y.
† Kohler, Delphin W. ..... Tacoma, Wash.
§ Kuhlmann, Lawrence B. ..... Melrose
† Lewis, Claude B. ..... St. Cloud
§ Libert, John N. ..... St. Cloud
§ Loes, Louis A. ..... St. Cloud
‡§Luckemeyer, Carl J. ..... Ft. Riley, Kansas
†§McDowell, John P. ..... St. Cloud
Meyer, Anthony A. ..... Melrose
§ Milhaupt, E. N. ..... St. Cloud
§ Musachio, Nicholas F. ..... Foley
Myre, Clifford R. ..... Paynesville
§ Neils, Vernon E. ..... Sauk Rapids
§ Nessa, Curtis B. ..... St. Cloud
Nietfeld, Aloys ..... Sauk Centre
§ O'Keefe, James P. ..... St. Cloud
§ Petersen, Robert T. ..... St. Cloud
§ Phares, Otto C. ..... St. Cloud
§ Raetz, Sylvester J. ..... Maple Lake
§ Reif, Henry J. ..... St. Cloud
§ Richards, William B. ..... St. Cloud
Salk, Richard I. ..... Albany
§ Sandven, Nels O. ..... Paynesville
† Schatz, Francis J. ..... St. Cloud
§ Schmitz, Everett J. ..... St. Cloud
† Sherwood, George E. ..... Kimball
Sisk, Harvey E. ..... St. Cloud
§ Stangl, Philip E. ..... St. Cloud
Thuringer, Carl B. ..... St. Cloud
Veranth, Leonard A. ..... St. Cloud
§ Walfred, Karl A. ..... St. Cloud
§ Wenner, Waldemar T. ..... St. Cloud
§ Wittrock, Louis H. ..... Watkins
Zachman, Albert H. ..... Melrose

STEELE COUNTY MEDICAL SOCIETY

Regular meetings, called by the officers
Annual meeting, February

Number of Members—23

President
ARNESEN, JOHN F. ..... Owatonna
Secretary
DEWEY, DONALD H. ..... Owatonna

§ Anderson, Franklin C. ..... Owatonna
§ Arnesen, John F. ..... Owatonna
§ Dewey, Donald H. ..... Owatonna
† Ertel, Edward Q. ..... Ellendale
§ Fischer, John R. ..... Blooming Prairie
§ Halvorsen, Daniel K. ..... Owatonna
Hartung, Elmer H. ..... Claremont
Henry, Kenneth G. ..... Owatonna
Honath, Donald H. ..... Owatonna
Kulstad, Oscar S. ..... Dodge Center
† Kurtin, Henry J. ..... Tucson, Arizona
‡ Kurtin, Joseph J. ..... New York, N. Y.
§ Lundquist, Curt W. ..... Owatonna
§ McEnaney, Clifford T. ..... Owatonna
§ McIntyre, John A. ..... Owatonna
†§Melby, Benedik ..... Blooming Prairie
† Morehead, Dewey E. ..... Owatonna
§ Olson, Albert J. ..... Owatonna
§ Roberts, Oliver W. ..... Owatonna
§ Schaefer, Joseph F. ..... Owatonna
† Senn, Edward W. ..... Owatonna
§ Stransky, Theodore W. ..... Owatonna
§ Wilkowske, Rudolph J. ..... Owatonna

UPPER MISSISSIPPI MEDICAL SOCIETY

(Aitkin, Beltrami, Cass, Clearwater, Crow Wing, Hubbard, Koochiching, Lake of the Woods, Morrison, Todd and Wadena Counties)

Annual meeting, January

Number of Members—95

President
QUANSTROM, VIRGIL E. ..... Brainerd
Secretary
BADEAUX, GEORGE I. ..... Brainerd

Anderson, Werner W. ..... Brainerd
§ Badeaux, George I. ..... Brainerd
*§Beise, Rudolph A. ..... Brainerd
Bender, James H. ..... Brainerd
§ Benson, Alfred H. ..... Little Falls
§ Bolstad, Owen C. ..... Little Falls
§ Borgerson, Arthur H. ..... Long Prairie
‡ Brown, Hector M. ..... Walker
Cardle, George E. ..... Brainerd
Closuit, Frederick C. ..... Aitkin
Cook, Jay M. ..... Staples
§ Coombs, Carl H. ..... Cass Lake
Craig, Clair C. ..... International Falls
Crow, Earl R. ..... South Bend, Ind.
Cushing, Robert L. ..... Brainerd
Davis, Lloyd T. ..... Wadena
§ Davis, Luther F. ..... Wadena
* Davis, Thomas L., Jr. ..... Wadena
Deweese, Wilford J. ..... Bemidji
§ Eiler, John ..... Park Rapids
§ Erickson, Alvin O. ..... Long Prairie
Fitzsimons, William E. ..... Brainerd
§ Fortier, George M. A. ..... Little Falls
Garlock, Arthur V. ..... Bemidji
Garlock, DeWitt W. ..... Highlands, Calif.
Ghostley, Mary C. ..... Bemidji
† Gilmore, Rowland ..... Crookston
Griffin, John W., Jr. ..... Bemidji
Groschupf, Theodore P. ..... Bemidji
§ Grose, Frederick N. ..... Clarissa
‡ Halme, William B. ..... San Francisco, Calif.
§ Hanover, Ralph D. ..... International Falls
Hansen, Milo L. ..... Little Falls
Hartien, Jason K. ..... Bemidji
Healy, Raymond T. ..... Pierz
Heine, George W. ..... Little Falls
Hendricks, Esten J. ..... St. Paul
Higgs, Walter W. ..... Park Rapids
Hildebrand, John E. ..... Bemidji
Hoganson, Donald E. ..... Bemidji
† House, Zachariah E. ..... Burbank, Calif.
Houston, Donald M. ..... Park Rapids
* Hubbard, Otto E. ..... Brainerd
§ Hughes, Bernard J. ..... Brainerd
§ Johnson, Douglas L. ..... Little Falls
† Johnson, Einer W. ..... Bemidji
Johnson, Rudolph E. ..... Mankato
Johnstone, William W. ..... Ah-gwah-ching
§ Kanne, Earl R. ..... Brainerd
Kelley, Roger E. ..... Crosby
Kinports, Edward B. ..... International Falls
§ Knight, Edwin G. ..... Swanville
Larson, LeRoy J. ..... Bagley
Lee, Hubert W. ..... Brainerd
Leinonen, Wendla E. ..... Wadena
§ Lenarz, Albert J. ..... Browerville
Longfellow, Helen W. ..... Brainerd
Lund, Werner J. ..... Staples
Lundsten, Leslie C. ..... Bemidji
§ McLane, William O. ..... Brainerd
§ MacDonald, Roger A. ..... Littlefork
Macheledt, Neil L. ..... Wadena
§ Marshall, Clark M. ..... Crosby
§ Meller, Maurice ..... Brainerd
Mortenson, Howard O. ..... Menahga
§ Mosby, Maurice E. ..... Long Prairie
§ Mulligan, Arthur M. ..... Brainerd
† Nelson, Nesmith P. ..... Minneapolis
Nelson, Robert H. ..... International Falls
Nixon, James B. ..... Crosby
Olson, Lillian A. ..... Ah-gwah-ching
§ Palmer, Harry A. ..... Blackduck
Parker, Charles W. ..... Wadena
§ Parker, Warren E. ..... Sebeka
Pedersen, Robert L. ..... Brainerd
§ Petraborg, Harvey T. ..... Aitkin
Pierce, Charles H. ..... Wadena
§ Quanstrom, Virgil E. ..... Brainerd
§ Ringle, Otto F. ..... Walker
Rozycki, Anthony T. ..... Pine River
Sanderson, Anton G. ..... Anoka
Schmitz, Glen P. ..... Little Falls
‡ Schwyzer, Arnold G. ..... San Juan, P. R.
Simons, Edwin J. ..... Edina
Skaife, William F. ..... Little Falls
Spurbeck, George R. ..... Ah-gwah-ching
§ Stein, Raymond J. ..... Pierz
‡ Stoy, Robert A. ..... Little Falls
Thabes, John A., Jr. ..... Brainerd
Vandersluis, Charles W. ..... Bemidji
§ Watson, Alexander M. ..... Royalton
Watson, Percy T. ..... Northfield
Watson, Sydney W. ..... Royalton
Whittemore, Dexter D. ..... Bemidji
§ Will, Charles B. ..... Bertha
§ Will, W. W. ..... Bertha
Williams, M. M. ..... Ah-gwah-ching
‡ Zeigler, Charles M. ..... Pine River

WABASHA COUNTY MEDICAL SOCIETY

Annual meeting, first Thursday after first Monday in October

Number of Members—13

President
ELLIS, EARL W. ..... Elgin
Secretary
FLESCHE, BERNARD A. ..... Lake City

§ Bayley, E. Covell ..... Lake City
§ Bouquet, Bertram J. ..... Wabasha
§ Bowers, Robert N. ..... Lake City
† Collins, Joseph S. ..... Wabasha
§ Ekstrand, LeRoy M. ..... Wabasha
§ Ellis, Earl Wm. ..... Elgin
§ Flesche, Bernard A. ..... Lake City
§ Gjerde, William P. ..... Lake City
Glabe, Robert A. ..... Plainview
§ Mahle, Donald G. ..... Plainview
§ Ochsner, Clarence G. ..... Wabasha
† Replogle, W. H. ..... Los Angeles, Calif.
Wellman, Thomas G. ..... Clinton, Iowa

WASECA COUNTY MEDICAL SOCIETY

Regular meetings, as decided
Annual meeting, January
Number of Members—10

President
Normann, Stephen T. .... Waseca
Secretary
Florine, Martin C. .... Janesville

§ Davis, Raymond D. .... Waseca
§ Florine, Martin C. .... Janesville
†§Gallagher, Bernard J. .... Waseca
§ Hottinger, Raymond C. .... Janesville
§ McIntire, Homer M. .... Waseca
§ Normann, Stephen T. .... Waseca
§ Oeljen, Siegfried C. G. .... Waseca
§ Olds, George H. .... New Richland
† Swenson, Donald B. .... Minneapolis
§ Swenson, Orvie J. .... Waseca

WASHINGTON COUNTY MEDICAL SOCIETY

(Washington and Dakota Counties)

Regular meetings, second Tuesday in each month
Annual meeting, December
Number of Members—18

President
Carlson, Russel E. .... Stillwater
Secretary
Juergens, Manley F. .... Stillwater

Brabec, Paul F. .... Hastings
§ Carlson, Russel E. .... Stillwater
Fasbender, Herman T. .... Hastings
*†§Haines, James H. .... Stillwater
§ Holcomb, Joel T. .... Marine-on-St. Croix
Humphrey, Wade R. .... Stillwater
§ Jenson, James E. .... Stillwater
*§Johnson, Ray G. .... Stillwater
Josewski, Raymond J. .... Stillwater
§ Juergens, Manley F. .... Stillwater
Just, Herman J. .... Hastings
Kulzer, Norbert J. .... Hastings
§ McCarten, Francis M. .... Stillwater
Menshcha, Nicholas .... Forest Lake
† Poirier, Joseph A. .... Forest Lake
Racer, Harley J. .... Stillwater
Ruggles, George M. .... Forest Lake
§ Sherman, Carnot H. .... Bayport
§ Stuhr, John W. .... Stillwater
Van Meier, Henry .... Stillwater

WEST CENTRAL MINNESOTA MEDICAL SOCIETY

(Big Stone, Pope, Stevens and Traverse Counties)

Regular meetings, one Tuesday in March, May, September and November
Annual meeting, November
Number of Members—25

President
Eide, O. A. .... Hancock

Secretary
Watson, Robert M. .... Morris

§ Arneson, Arthur I. .... Morris
§ Behmler, Fred'k Wm. .... Morris
†§Bergan, Otto .... Clinton
† Bolsta, Charles .... Ortonville
‡§Bucher, Foster D. .... N. Y., N. Y.
†§Eberlin, Edward A. .... Glenwood
§ Eide, O. A. .... Hancock
† Elsey, James R. .... Glenwood
*†Fitzgerald, E. T. .... Rialti, Calif.
§ Giesen, Allan F. .... Starbuck
Good, Roy H. .... Glenwood
Hedemark, Homer H. .... Ortonville
Hedemark, Truman A. .... Ortonville
Karn, Jacob F. .... Ortonville
§ Kooda, Jennings C. .... Morris
Lee, Gordon E. .... Glenwood
§ Lindberg, Alfred L. .... Wheaton
†§Linde, Herman .... Cyrus
§ Magnuson, Allen E. .... Wheaton
§ Merrill, Robert W. .... Morris
Muir, Walter F. .... Browns Valley
*§O'Donnell, D. M. .... Ortonville
§ Oliver, Irwin L. .... Graceville
† Ransom, Matthias L. .... Hancock
§ Rossberg, Raymond A. .... Morris
§ Swedenburg, Paul A. .... Glenwood
§ Watson, Robert M. .... Morris

WINONA COUNTY MEDICAL SOCIETY

Regular meeting, first Monday in January, April, July and October
Annual meeting, first Monday in January
Number of Members—32

President
Heise, Philip vR. .... Winona
Secretary
Wilson, Louis J. .... Winona

*†Benoit, Frank T. .... Winona
Boardman, Dalmon V. .... Winona
§ Christensen, Eli E. .... Winona
§ Finkelnburg, William O. .... Winona
‡ Haesly, Warren W. .... Winona
§ Hartwich, Roger F. .... Winona
Heise, Carl vR. .... Winona
§ Heise, Herbert vR. .... Winona
§ Heise, Paul vR. .... Winona
§ Heise, Philip vR. .... Winona
Heise, William vR. .... Winona
Hughes, Sidney O. .... Winona
§ Johnston, Leonard F. .... Winona
Keyes, John D. .... Winona
§ Loomis, G. L. .... Winona
† McLaughlin, Edmund M. .... Winona
§ Mattison, Percy A. .... Winona
§ Meinert, Albert E. .... Winona
Neumann, Conrad A. .... Winona
§ Page, Raymond L. .... St. Charles
†§Robbins, Charles P. .... Winona
§ Roemer, Henry J. .... Winona
§ Rogers, Charles W. .... Winona
§ Rollins, Pat .... St. Charles
§ Satterlee, Howard W. .... Lewiston
§ Schmidt, Hilmar R. .... Winona
*§Steiner, Irving W. .... Winona
§ Tweedy, John A. .... Winona
Tweedy, Robert B. .... Winona
Vollmer, Fredk. J. .... Winona
Wilson, Louis J. .... Winona
§ Wilson, Rolland H. .... Winona
§ Woltjen, Myron J. .... Rushford
§ Younger, Louis I. .... Winona

WRIGHT COUNTY MEDICAL SOCIETY

Regular meeting, first Tuesday of every third month
Annual meeting, first Tuesday in October
Number of Members—17

President
Sandeen, Robert M. .... Buffalo
Secretary
Catlin, Theodore J. .... Buffalo

‡ Abullarade, Jose A. .... Cokato
§ Anderson, Waldo P. .... Buffalo
§ Bendix, Lester H. .... Annandale
†§Catlin, John J. .... Buffalo
§ Catlin, Theodore J. .... Buffalo
§ Ellison, Frank E. .... Monticello
‡ Fetzek, Albert D. .... St. Michael
§ Greenfield, Wm. Theodore .... Cokato
Grundset, Ole J. .... Montrose
§ Guilfoile, Pierre F. .... Delano
§ Hall, William E. .... Maple Lake
§ Hart, William E. .... Monticello
§ Sandeen, Robert M. .... Buffalo
§ Shragg, Robert I. .... Howard Lake
§ Smorstok, Matthew B. .... Monticello
§ Thielen, Robert D. .... St. Michael
§ Thomas, William H. .... Howard Lake

Alphabetic Roster

Key to Symbols:

***Deceased; †Associate, Junior Associate, Residency, Affiliate or Life Member; ‡In Service**

Aanes, Almer M.	Red Wing
Abbott, Albert R.	Rochester
Abraham, Arden L.	Duluth
Abramson, Milton	Minneapolis
Abullarade, Jose A.	Cokato
Achor, Richard W. P.	Rochester
Adair, Albert F., Jr.	St. Paul
Adams, Bertram S.	Hibbing
Adams, Richard C.	Rochester
Addy, Edward R.	Gilbert
Adkins, Charles D.	Minneapolis
*†Adkins, Charles M.	Thief River Falls
Adkins, Galen H.	Anoka
Affeldt, Daniel E.	Kasson
Aga, John H.	Rochester
Agustsson, Hreidar	Minneapolis
Ahern, Eugene E.	Minneapolis
Ahlfs, Jacob J.	Caledonia
Ahola, Kenneth E.	Hibbing
† Ahrens, Albert E.	St. Paul
Ahrens, Curtis F.	Duluth
Ahrens, Robert M.	St. Paul
† Aitkens, Herbert B.	LeCenter
Akins, Willard M.	Red Wing
Albrecht, H. H.	Chisago City
Alcorn, William J.	Wabasso
‡ Alden, J. F., Jr.	St. Paul
Alden, W. Charles	Kimball
Alexander, Harlan A.	Minneapolis
Aling, Charles A.	Minneapolis
Allen, Edgar V. N.	Rochester
Allen, George S.	Cannon Falls
Allen, John H.	Montevideo
Allison, David D.	Litchfield
Althausen, Theo. L., Jr.	Minneapolis
† Altnow, Hugo O.	Coral Gables, Fla.
Amatuzio, Donald S.	Minneapolis
† Amberg, Samuel	Rochester
Andersen, Howard A.	Rochester
Andersen, Silas C.	Minneapolis
Anderson, Arnold S.	St. Louis Park
Anderson, Chester A.	Hector
Anderson, Chester A.	Madison
Anderson, David M.	Minneapolis
Anderson, David P., Jr.	Austin
‡ Anderson, Donald C.	Miami, Fla.
† Anderson, Edward D.	Minneapolis
Anderson, Ernest M.	St. Cloud
Anderson, Ernest R.	Minneapolis
Anderson, Frank J.	Minneapolis
Anderson, Franklin C.	Owatonna
Anderson, Harold J.	Austin
Anderson, James J.	Mankato
Anderson, John T.	Minneapolis
Anderson, Karl W.	Minneapolis
Anderson, Margaret C.	Mankato
Anderson, Mark J.	Rochester
Anderson, Markham J.	Rochester
Anderson, Milton W.	Rochester
Anderson, Richard E.	Willmar
Anderson, Richard W.	St. Paul
Anderson, Roger L.	Minneapolis
Anderson, U. Schuyler	Minneapolis
Anderson, Waldo P.	Buffalo
Anderson, Wallace E.	Clearbrook
Anderson, Wallace E.	Lakeville
Anderson, Wallace R.	Austin
Anderson, Werner W.	Brainerd
Anderson, William H.	Minneapolis
Anderson, William T.	Minneapolis
Andreassen, Einar C.	St. Paul
Andreassen, Rolf L.	Minneapolis
Andresen, K. D'A.	Minneapolis
Andrew, William F.	Duluth
Andrews, Bernice F.	Holdingford
Andrews, Robert S.	Minneapolis
† Andrews, Roy N.	Mankato
Ankner, Frank J.	Minneapolis
Antonow, Arthur M.	Virginia
* Archer, Willard E.	Anoka
† Ardan, Nicholas I., Jr.	Rochester
Arends, Archabald L.	Minneapolis
Arey, S. Lane	Minneapolis
Arhelger, Stuart	Minneapolis
Arko, Joseph L.	Hibbing
Arlander, Clarence E.	Minneapolis
Arling, Leonard S.	Minneapolis
Arms, James J.	Minneapolis
Armstrong, Ralph S.	Winnebago
Arndt, Harry Wm.	Detroit Lakes
Arnesen, John F.	Owatonna
Arneson, Arthur I.	Morris
Arnold, Anna W.	Minneapolis
Arnold, Elmer Wm.	Adrian
Arnquist, Andrew S.	St. Paul
Arny, Frederick P.	St. Paul
Arvidson, Carl G.	Minneapolis
Arzt, Philip K.	St. Paul
Asta, Joseph J.	Duluth
Athens, Alvin G.	Duluth
Atmore, William G.	Rochester
Aufderheide, Arthur C.	Duluth
† Aune, Martin	Minneapolis
† Aurand, William H.	Minneapolis
Aurelius, J. Richards	St. Paul
Ausman, Duane R.	St. Paul
Autrey, William A.	St. Cloud
Austrian, Sol	Cosmos
‡ Baars, Conrad W.	Camp Cook, Calif.
Babb, Frank S.	St. Paul
Backus, Reno W.	Nopeming
Bacon, Donald K.	St. Paul
Badeaux, George I.	Brainerd
Bader, J. L.	Slayton
Baer, Walter	St. Paul
‡ Bagby, George W.	Ft. Leonard Wood, Mo.
Baggenstoss, Archie H.	Rochester
Baggenstoss, Osmond J.	Minneapolis
Bagley, Charles M.	Duluth
Bagley, Elizabeth C.	Duluth
Bagley, Russell W.	Minneapolis
Bagley, William R.	Duluth
Baich, Velemir M.	Coleraine
Bain, Robert C.	Rochester
Bair, Hugo L.	Rochester
Baird, Joseph W.	Minneapolis
Baird, Raymond L.	Lake Crystal
Baken, Melvin P.	Minneapolis
Baker, A. B.	Minneapolis
Baker, Alfred T.	Minneapolis
Baker, George S.	Rochester
† Baker, Harry R.	Hayfield
‡ Baker, Hillier L., Jr.	West Point, N. Y.
Baker, Jeannette L.	Fergus Falls
*†Baker, Looe	Minneapolis
Baker, Milton E.	Minneapolis
Baker, Norman H.	Fergus Falls
Baker, Russell L.	Hayfield
Bakkila, Henry E.	Duluth
Balcome, Milton M.	St. Paul
Baleisis, Peter	Minneapolis
† Balfour, Donald C.	Rochester
Balfour, William M.	Rochester
Balkin, Samuel G.	Minneapolis
Balogh, Charles J.	Minneapolis
Bank, Harry E.	San Francisco, Calif.
Banner, Edward A.	Rochester
Barber, Tracy E.	Austin
Bardon, Richard	Duluth
Bargen, J. Arnold	Rochester
Barker, John D.	Duluth
Barker, Nelson W.	Rochester
Barlow, Loren C.	Rochester
Barnes, Arlie R.	Rochester
Barnes, Richard E.	Aurora
Barnett, Joseph M.	St. Paul
† Barney, Leon A.	Duluth
Barno, Alex	St. Louis Park
‡ Baronofsky, Ivan D.	St. Paul
Barr, James S.	Elmore
Barr, Lowell C.	Albert Lea
Barr, Maxwell M.	Minneapolis
Barr, Robert N.	Minneapolis
Barr, Ronald W.	Montevideo
Barrett, Earl E.	Duluth
Barron, Jesse J.	Minneapolis
† Barron, Moses	Minneapolis
‡ Barron, S. Steven	Minneapolis
Barry, Maurice J., Jr.	Rochester
† Barsness, Nellie O. N.	St. Paul
Bartholomew, Lloyd G.	Rochester
Bartzen, Peter J.	Duluth
Basinger, Harold P.	Windom
Basinger, Harvey R.	Mountain Lake
Basinger, Homer P.	Windom
Bastron, James A.	Rochester
Batdorf, B. Niles	Good Thunder
Bauer, Eugene L.	St. Paul
Baumgartner, Florian H.	Albany
† Baxter, Stephen H.	Minneapolis
Bayley, E. Covell	Lake City
‡ Bayard, Edwin D.	Scott Air Force Base, Ill.
Beach, Northrop	Minneapolis
Beahrs, Oliver H.	Rochester
† Beals, Hugh	LaJolla, Calif.
Beaton, J. Gordon	Northfield
Beck, Charles J.	No. St. Paul
† Becker, Arnetta M.	Minneapolis
Becker, Frederick T.	Duluth
Beckering, Gerrit	Edgerton
Beddow, Ralph M.	Rochester
Bedford, Edgar Wm.	Minneapolis
Beech, Raymond H.	St. Paul
Beek, Harvey O.	St. Paul
Beer, John J.	St. Paul
Behmler, Fred'k Wm.	Morris
Behr, Orlo K.	Crookston
Beirstein, Samuel	Minneapolis
* Beise, Rudolph A.	Brainerd
Beiswanger, Richard H.	Minneapolis
Bell, Charles C.	St. Paul
† Bell, E. T.	Minneapolis
Bellomo, James	St. Paul
Bellomo, John	St. Paul
Bellville, Titus P.	Minneapolis
Belzer, Meyer S.	Minneapolis
Bender, James H.	Brainerd
Bendix, Lester H.	Annandale
† Benedict, William L.	Rochester
Benell, Otto E.	Virginia
Benepe, James L.	St. Paul
Benesh, Louis A.	Minneapolis
Benjamin, Edwin G.	Minneapolis
Benjamin, Harold G.	Minneapolis
Benjamin, Walter G.	Pipestone
† Bennion, P. H.	Isway, Mont.
*†Benoit, Frank T.	Winona
Benson, Alfred H.	Little Falls
Bentley, Norman P.	St. Paul
Bepko, Marie K.	Cloquet
Berdez, George L.	Duluth
Berg, Arnold	Hawley
Berg, Clinton C.	Excelsior
† Bergan, Otto	Clinton
Bergan, R. O.	Duluth
Berge, David O.	Roseau
Berge, Harry L.	Mora
Berge, Kenneth	Rochester
Berger, Alex G.	Minneapolis
Bergh, George S.	Minneapolis
Bergh, Solveig M.	Minneapolis
Berglund, Eldon B.	Minneapolis
Bergman, Oscar B.	St. James
* Bergquist, Karl E.	Battle Lake
† Berkman, David M.	Oronoco
Berkman, John M.	Rochester
Berkwitz, Nathaniel J.	Minneapolis
Berlin, Anthony S.	Hallock
Berman, Reuben	Minneapolis
Bernatz, Philip E.	Rochester
Bernier, M. J.	No. St. Paul
Bernstein, Irving C.	Minneapolis
Bernstein, William C.	St. Paul
Bessesen, Alfred N., Jr.	Minneapolis
Bessesen, Daniel H.	Olivia
*†Bessesen, William A.	Minneapolis
Betts, Charles S.	Rochester
Beuning, John B.	St. Cloud
Bianco, Anthony J.	Duluth
† Bianco, Anthony J., Jr.	Rochester
Bicek, Joseph F.	St. Paul
Bickel, William H.	Rochester
† Biedermann, Jacob	Thief River Falls
Bieter, Raymond N.	Minneapolis
† Bigelow, Charles E.	Dodge Center
Bigler, Earl E.	Perham
Bigler, Ivan E.	Perham
Bilka, Paul J.	Minneapolis
Billings, Ralph E.	Franklin
Binder, Manuel R.	Minneapolis
† Binet, Henry E.	Grand Rapids
† Binger, Henry E.	Phoenix, Ariz.
Biornson, Robert G.	Minneapolis
Black, B. Marden	Rochester
Black, Earl J.	St. Paul
Black, William A.	New Ulm
Blackmore, Sidney C.	Biwabik
Blake, Allen J.	Hopkins
Blake, James A.	Hopkins
Blake, Paul S.	Hopkins
† Blakey, Adam R.	Osakis
Bloedel, Traugott J. G.	Osseo
Blomberg, Robert D.	Minneapolis
Blomberg, William R.	Princeton
Bloom, Joseph	Duluth

Bloom, Norman B.....Minneapolis
Blumberg, Henry B.....
French Camp, Calif.
Blumenthal, Jacob S.....Minneapolis
Boardman, Dalmon V.....Winona
† Bock, Ronald A.....St. Paul
* Bockman, Michael W. H.....
Hot Springs Nat'l. Park, Ark.
Bodaski, Albert A.....Tyler
‡ Bodelson, A. H.....Little Rock, Ark.
† Boeckmann, Egil.....St. Paul
Boehrer, John J.....Minneapolis
Bofenkamp, Benjamin.....Minneapolis
Bofenkamp, F. William.....Luverne
‡ Bohn, Donald G.....Minneapolis
Boies, Lawrence R.....Minneapolis
Bolender, Harold L.....St. Paul
Boline, Clifford A.....Battle Lake
† Bolsta, Charles.....Ortonville
Bolstad, Owen C.....Little Falls
‡ Bolz, J. Arnold.....Camp Carson, Colo.
Boman, Paul G.....Duluth
Bonello, Frank J.....St. Paul
Bonner, John L.....Eveleth
‡ Bonnet, John D.....Rochester
Boody, George J., Jr.....Dawson
Boone, Ervin S.....Luverne
† Booth, Albert E.....Minneapolis
† Boreen, Clifton A.....Minneapolis
Borg, Joseph F.....St. Paul
Borgerson, Arthur H.....Long Prairie
† Borgeson, Egbert J.....St. Paul
Borman, Chauncey N.....Minneapolis
Borowicz, Leonard A.....Minneapolis
Bosland, Howard G.....Willmar
Bossard, John W.....Rochester
† Bossert, Clarence S.....Mora
‡ Boswell, J. Thornton.....Portsmouth, Va.
† Botham, Richard J.....Rochester
Bottolfson, Bottolf T.....Moorhead
Bouchelle, McLemore.....Virginia
† Bouma, John H.....Topeka, Kans.
† Bouma, Lewis R.....St. Paul
Bouquet, Bertram J.....Wabasha
Bouthilet, Florence J.....St. Paul
Bowen, Robert L.....Hibbing
† Bowers, Dorrance.....Rochester
Bowers, Gordon G.....Minneapolis
Bowers, Robert N.....Lake City
Bowes, Donald E.....Rochester
Boyd, David A., Jr.....Rochester
† Boyd, George K.....Pittsburgh, Pa.
Boyer, George S.....Crookston
† Boyer, Samuel H., Sr.....Duluth
Boyer, Samuel H., Jr.....Duluth
Boynton, Ruth E.....Minneapolis
Boysen, Herbert.....Madelia
† Boysen, Peter.....Bemidji
Braasch, John W.....Rochester
† Braasch, William F.....Rochester
Brabec, Paul F.....Hastings
Brackett, Ralph D.....Rochester
Brand, George D.....St. Paul
† Brand, William A.....Redwood Falls
Brandenburg, Robert O.....Rochester
Bratholdt, James W.....Watertown
Bratrud, Arthur F.....Minneapolis
† Bratrud, Edward.....Thief River Falls
Bratrud, Theodore E.....Minneapolis
Bratrude, Earl J.....St. James
Braun, Ohrmundt C.....Grand Rapids
Braun, Robert A.....Rochester
† Bray, Elwyn R.....St. Paul
Bray, Philip N.....Duluth
‡ Breitenbucher, Robert B.....
Columbia, S. C.
Brekke, Harvey J.....Minneapolis
Bretzke, Carl O.....Hutchinson
Bridge, Allyn G.....Minneapolis
Bridge, Ezra V.....Cannon Falls
Briggs, John F.....St. Paul
† Brigham, Charles F., Sr.....St. Cloud
Brigham, Charles F., Jr.....St. Cloud
Brill, Alice K.....Minneapolis
Brink, Adlai A.....Baudette
Brink, Donald M.....Hutchinson
Broadbent, James C.....Rochester
Broadie, Thomas E.....St Paul
Brockway, Roger W.....Grand Rapids
Broders, C. W.....Rochester
Brodie, Walter D.....St. Paul
† Broker, Henry M.....Minneapolis
Brooker, Warren J.....Duluth
† Brooks, Charles N.....Minneapolis
Brown, Alexander E.....Rochester
† Brown, Alexander H.....Pipestone
† Brown, Edgar D.....Paynesville
‡ Brown, Hector M.....Walker
Brown, Henry A.....Rochester
Brown, Ian A.....Minneapolis
Brown, Joe R.....Rochester
† Brown, John C.....Los Gatos, Calif.
Brown, Philip W.....Rochester

Brown, Roland G.....Rochester
Brown, William D.....Minneapolis
Bruhl, Heinz H.....Faribault
Brunsting, Louis A.....Rochester
Brusegard, James F.....Red Wing
‡ Bucher, Foster D.....N. Y., N. Y.
Buchstein, Harold F.....Minneapolis
† Buck, Frederick H.....Shakopee
Buckley, J. J.....St. Paul
Buckley, Robert P.....Duluth
Buie, Louis A.....Rochester
Buirge, Raymond E.....Minneapolis
Bulinski, Theodore J.....St. Paul
Bulkley, Kenneth.....Minneapolis
Bumpus, Frank T.....Rochester
Bunker, Bevan W.....Anoka
Burch, Edward P., II.....St. Paul
† Burch, Frank E.....St. Paul
Burchell, Howard B.....Rochester
‡ Buresh, Kenneth L.....Aurora, Colo.
Burke, Edmund C.....Rochester
Burklund, Edwin C.....St. Paul
Burleigh, Edward G.....Eveleth
Burlingame, David A.....St. Paul
Burmeister, Richard O.....St. Paul
Burnett, Joseph W.....New Ulm
Burnham, Wesley H.....Minneapolis
Burns, Catherine.....Albert Lea
Burns, Floyd M.....Milan
Burns, M. Alpheus.....Milan
† Burns, Robert M.....St. Paul
Burseth, Edgar C.....Mora
Burton, Carl G.....St. Paul
Buscher, Julius C.....St. Cloud
Bush, Robert P.....St. Paul
Bushard, Wilfred J.....Minneapolis
Busher, Herbert H.....St. Paul
Butler, John K.....Cloquet
Butt, Hugh R.....Rochester
Butturff, Carl R.....Freeborn
Butzer, John A.....Mankato
Butzer, John F.....Mankato
Buzzelle, Leonard K.....Minneapolis

Cable, Morris L.....Minneapolis
Cabot, Clyde M.....Minneapolis
Cady, Laurence H.....Minneapolis
Cain, Clark L.....St. Paul
Cain, James C.....Rochester
Cain, James H.....Hoffman
Cairns, Robert J.....Redwood Falls
† Calhoun, F. W.....Albert Lea
Callahan, Francis F.....St. Paul
Cameron, Isabell L.....Minneapolis
Cameron, John H.....Crookston
Camp, Ray Junior.....Madison
† Campagna, Mario J.....Rochester
Campbell, Donald C.....Rochester
Campbell, Lowell M.....Minneapolis
Campbell, Orwood J.....Minneapolis
Canfield, Wayne W.....St. Peter
Canine, James L.....So. St. Paul
Cantwell, William F.....International Falls
Caplan, Leslie.....Minneapolis
Card, William H.....Minneapolis
Cardle, George E.....Brainerd
Carey, James B.....Minneapolis
Carlander, Lester W.....Minneapolis
Carley, Walter A.....St. Paul
Carlson, Carl E.....Alexandria
Carlson, John V.....Westbrook
Carlson, Lawrence.....Minneapolis
Carlson, Leonard T.....Minneapolis
Carlson, Russel E.....Stillwater
Carlson, Vernon J.....Moorhead
Caron, Robert P.....Minneapolis
Carr, David T.....Rochester
Carr, William J.....Minneapolis
Carroll, John J.....Winsted
* Carroll, William C.....St. Paul
Carryer, Haddon McC.....Rochester
Carthey, Frank J.....New Ulm
Caspers, Carl G.....Minneapolis
† Catlin, John J.....Buffalo
Catlin, Theodore J.....Buffalo
† Cavanor, Frank T.....Minneapolis
Cedarleaf, Cherry B.....St. Paul
Ceder, Elmer T.....Minneapolis
Ceplecha, Stanley F.....Redwood Falls
Cervenka, Charles F.....New Prague
‡ Cesnik, Robert J.....St. Cloud
Chadbourn, Charles R.....St. Paul
† Chadbourn, Wayne A.....Minneapolis
Chalgren, William S.....Mankato
Challman, Samuel A.....Minneapolis
† Chambers, Winslow C.....Blue Earth
Chapin, Lemuel E.....Rochester
* Chapman, Theodore L.....Duluth
Chatterton, Carl C.....St. Paul
Chavez, Demetrio A.....Minneapolis
Chermak, Francis G.....International Falls
Chesler, Merrill D.....Minneapolis
† Chesley, Albert J.....Minneapolis

Childs, Donald S., Jr.....Rochester
Chisholm, Tague C.....Minneapolis
Christensen, Clarence H.....Duluth
Christensen, Eli E.....Winona
Christensen, Llewellyn E.....Salem, Ore.
Christensen, Norman A.....Rochester
Christiansen, Andrew.....St. Paul
Christiansen, Harold A.....Jackson
Chunn, Stanley S.....Willmar
Ciriacy, Edward W.....Ely
Clagett, Oscar T.....Rochester
Clapp, Hubert D.....Crookston
Clark, Clarence L.....Duluth
Clark, Edward C.....Rochester
Clark, Elizabeth A.....Duluth
Clark, Harry B.....St. Cloud
Clark, Henry B., Jr.....Minneapolis
Clark, Ivan T.....Duluth
Clark, Leslie Wm.....Spring Valley
Clark, Malcolm D.....Minneapolis
Clarke, Eric K.....Minneapolis
Clarke, John W.....Watertown
Clay, Lyman B.....Minneapolis
† Clayburgh, Bennie J.....Rochester
Claydon, Howard F.....Red Wing
Cleaves, William D.....Sauk Centre
† Clement, John B.....Lester Prairie
Clifford, George W.....Alexandria
Clifton, Theodore A.....Chatfield
Closuit, Fredk. C.....Aitkin
Cochrane, Byron B.....St. Paul
Cochrane, Ray F.....Minneapolis
Coddon, Walter D.....St. Paul
Coe, John I.....Minneapolis
Cohen, Bernard A.....Minneapolis
Cohen, Ellis N.....Minneapolis
Cohen, Ephraim B.....Minneapolis
Cohen, Maynard M.....St. Paul
Cohen, Sumner S.....Oak Terrace
Colby, M. Y., Jr.....Rochester
Colby, Woodard L.....St. Paul
Cole, James S.....Minneapolis
Cole, Wallace H.....St. Paul
Coleman, John B.....St. Paul
Coll, James J.....Duluth
† Collie, Henry G.....St. Petersburg, Fla.
† Collins, Arthur N.....Duluth
† Collins, Joseph S.....Wabasha
Collins, Loren E.....Rochester
Colosey, Frederick J.....Ely
Combacker, Leon C.....Fergus Falls
Comfort, Mandred W.....Rochester
Compton, Russell F.....Rochester
Condit, William H.....Minneapolis
‡ Conley, Francis W.....Ft. Hood, Texas
Conley, Robert H.....Mankato
‡ Connolly, Coleman J.....Ft. Belvoir, Va.
Connor, Charles E.....St. Paul
Cook, Carroll K.....St. Paul
Cook, Edward N.....Rochester
Cook, Jay M.....Staples
Cooley, Jack C.....Rochester
Coombs, Carl H.....Cass Lake
Cooper, Charles C.....St. Paul
Cooper, John P.....Excelsior
† Cooper, Maurice D.....Winnebago
Cooper, Milton.....Rochester
Cooper, Robert R.....Minneapolis
† Cope, Hershel B.....Virginia
Corbin, Kendall B.....Rochester
Corniea, Albert D.....Minneapolis
Correa, Dale H.....Minneapolis
Corrigan, Cyril J.....Minneapolis
Coseriu, Vasile G.....St. Cloud
Cosgriff, James A., Sr.....Olivia
Cosgriff, James A., Jr.....Olivia
Costello, Addis.....Rochester
Coulter, Harold E.....Madelia
Counseller, Virgil S.....Rochester
Countryman, Roger S.....St. Paul
Counts, Robert N.....Rochester
Covell, Walter W.....St. Peter
Coventry, Markham B.....Rochester
Coventry, William D.....Duluth
Covey, Kenneth W.....Mahnomen
Cowan, Donald W.....Minneapolis
Cowan, George M.....Duluth
Craig, Clair C.....International Falls
Craig, David M.....St. Paul
Craig, M. Elizabeth.....Minneapolis
‡ Craig, Richard M.....Rochester
Craig, Winchell McK.....Rochester
Cranmer, Richard R.....Minneapolis
Cranston, Robert W.....Minneapolis
Creevy, Charles D.....Minneapolis
Creighton, Ralph H.....Minneapolis
Critchfield, Lyman R.....St. Paul
Cronwell, Bernhard J.....Austin
Crow, Earl R.....South Bend, Ind.
Crowley, James H.....St. Paul
Crudo, Vincent D.....St. Paul
Crumbley, James J.....Rochester
Crump, James W.....St. Paul

Culligan, John M.	St. Paul
Culligan, Leo C.	Minneapolis
Culp, Ormond S.	Rochester
Culver, L. G.	St. Paul
Cundy, Donald T.	Minneapolis
Curtis, Rauen A.	LeCenter
Cushing, Robert L.	Brainerd
† Cutts, George	Minneapolis
Dady, Elmer E.	Minneapolis
Daehlin, Rolf	Fergus Falls
Daggett, Donald R.	Minneapolis
Dahl, Elmer O.	Minneapolis
‡ Dahl, James C.	Minneapolis
Dahl, John A.	Minneapolis
Dahlin, David C.	Rochester
‡ Dahlstet, John P.	San Ysidro, Calif.
† Daignault, Oscar	Benson
Dale, Lester N.	Red Lake Falls
‡ Daly, David	Rochester
† Daniel, Donald H.	Minneapolis
† Danielson, Karl A.	Litchfield
Danielson, Lennox	Litchfield
Danyluk, Michael	Minneapolis
Daugherty, Guy W.	Rochester
David, Reuben	Minneapolis
Davis, Austin C.	Rochester
Davis, Charles T.	Rochester
Davis, Edward V.	St. Paul
Davis, George D.	Rochester
Davis, Harry L.	Rochester
Davis, Jay C.	Minneapolis
Davis, Lloyd T.	Wadena
Davis, Luther F.	Wadena
Davis, Raymond D.	Waseca
Davis, Robert E.	Rochester
* Davis, Thomas L.	Wadena
Davis, William I.	Mound
Dawe, Clyde J.	Rochester
Dawson, James R.	St. Paul
Dawson, Lorin D.	Worthington
Dearing, William H.	Rochester
*†DeBoer, Hermanus	Edgerton
Decker, Charles H.	St. Paul
Decker, David G.	Rochester
DeGeest, James H.	Goodhue
DeKruif, Hendrik	Fergus Falls
† Delmore, J. L., Sr.	Roseau
Delmore, J. L., Jr.	Roseau
Del Plaine, C. W.	Minneapolis
Demo, Robert A.	Albert Lea
Derauf, Benjamin I.	St. Paul
Deters, Donald C.	St. Paul
Detjen, Edward D.	Big Fork
Deveraux, Thomas J.	Wayzata
Devine, Kenneth D.	Rochester
DeWall, Richard A.	Minneapolis
DeWeerd, James H.	Rochester
DeWeese, Robert C.	St. Cloud
Deweese, Wilford J.	Bemidji
Dewey, Donald H.	Owatonna
Dickman, Roy W.	Minneapolis
Dickson, Franklin H., Jr.	Proctor
† Dickson, Thomas H.	St. Paul
Diefenbach, Eugene J., Jr.	Minneapolis
Diehl, Harold S.	Minneapolis
Dierker, Heinrich	Minneapolis
Diessner, A. W.	Redwood Falls
Diessner, Grant R.	Rochester
Diessner, Henry D.	Minneapolis
Dille, Donald E.	Litchfield
† Dines, David E.	Rochester
Dixon, Claude F.	Rochester
Doane, Joseph C.	Rochester
Dobson, M. W.	Mankato
Dockerty, Malcolm B.	Rochester
Dodds, William C.	Detroit Lakes
Dodge, Henry W., Jr.	Rochester
Doehlert, Charles A., Jr.	Rochester
Doherty, Elmer M.	New Prague
Dokken, James H.	Windom
Doman, Victor Wm.	Lakefield
Doms, Vernon A.	Elbow Lake
Donaldson, Charles S.	St. Cloud
Donatelle, Edward P.	Minneapolis
† Donin, J. F.	Winnipeg, Man., Can.
Donoghue, Francis E.	Rochester
Donovan, Daniel L.	Albert Lea
Dordal, John	Sacred Heart
Dorge, Richard I.	Minneapolis
Dornbach, Robert A.	Excelsior
† Dornblaser, Harry B.	Los Gatos, Calif.
Dorsey, George C.	Minneapolis
Douglas, Kenneth W.	Sandstone
Douglass, Bruce E.	Rochester
‡ Dovenmuehle, Robt. H.	Durham, N. C.
Downing, William C.	Crookston
† Doxey, Gilbert L.	Minneapolis
Doxsee, George C.	Chisholm
Doyle, George C.	Duluth
Doyle, James R.	Rochester
Doyle, Lawrence O.	Minneapolis
Drake, Carl B.	St. Paul
† Drake, Charles R.	Minneapolis
† Dredge, Homer P.	Sandstone
Dredge, Thomas E.	Minneapolis
Drexler, George W.	Blue Earth
Drill, Herman E.	Hopkins
† Drips, Della G.	Oronoco
Dry, Thomas J.	Rochester
Dubbe, Frederick H.	New Ulm
DuBois, J. F., Sr.	Sauk Centre
DuBois, J. F., Jr.	Sauk Centre
Duff, Edwin R.	Minneapolis
Dummer, Donald J.	Minneapolis
Duncan, James W.	Moorhead
† Dungay, Neil S.	Northfield
Dunlap, Earl H.	Minneapolis
Dunn, Jack, Jr.	Rochester
Dunn, James N.	St. Paul
Dupont, Joseph A.	Excelsior
Duryea, Willis M.	Minneapolis
DuShane, James W.	Rochester
† Dutton, C. E.	Minneapolis
Dvorak, Benjamin A.	Minneapolis
Dwan, Paul F.	Minneapolis
Dworsky, Samuel D.	Minneapolis
Dwyer, John J.	Duluth
† Dyer, John Allen	Rochester
‡ Dykstra, Peter C.	West Point, N. Y.
Dysterheft, Adolf F.	Gaylord
Earl, George A.	St. Paul
Earl, John R.	St. Paul
Eaton, Lealdes M.	Rochester
Eberley, Tohe S.	Benson
† Eberlin, Edward A.	Glenwood
Eckdale, John E.	Marshall
Eckman, Philip F.	Duluth
Eckman, Ralph J.	Duluth
Edelmann, Robert B.	Rochester
Eder, Walter P.	Minneapolis
* Edlund, Gustaf	St. Paul
Edwards, Jesse E.	Rochester
Edwards, Joseph W.	St. Paul
Edwards, Lloyd G.	St. Paul
Edwards, Thomas J.	St. Paul
Eelkema, H. Harrison	St. Paul
Egge, Sanford G.	Albert Lea
Eginton, Charles T.	St. Paul
Ehrenberg, Claude J.	Minneapolis
Ehrlich, Sol P.	Minneapolis
Eich, Matthew A.	Minneapolis
Eichhorn, Edmund P.	Minneapolis
Eide, O. A.	Hancock
Eiler, John	Park Rapids
Eisenstadt, David H.	Minneapolis
Eisenstadt, William S.	Minneapolis
Eitel, George D.	Minneapolis
† Ekblad, John W.	Duluth
Eklund, Carl D.	Duluth
Ekstrand, LeRoy M.	Wabasha
Elias, Frank J.	Duluth
Elkins, Earl C.	Rochester
Ellertson, Leonard M.	Albert Lea
Ellinger, Albert J.	Willmar
Elliot, Harold J.	Hayfield
Ellis, Earl Wm.	Elgin
Ellis, F. Henry	Rochester
Ellison, David E.	Minneapolis
Ellison, Ellis	Minneapolis
Ellison, Frank E.	Monticello
† Elsey, James R.	Glenwood
Elstner, Howard L.	Rochester
† Ely, Orriman S.	So. St. Paul
Emanuel, Karl Wm.	Duluth
Emerson, Edward C.	St. Paul
Emerson, Edwin E.	Osakis
Emmett, John L.	Rochester
Emmons, R. W.	St. Paul
Emond, Albert J.	Farmington
Emond, Joseph S.	Farmington
Endress, Edward K.	St. Paul
Engberg, Edward J.	Faribault
Engel, Joseph P.	Minneapolis
Engelhart, Peter C.	Minneapolis
Englund, Elvin F.	Minneapolis
Engstrand, Oscar J.	Minneapolis
Engstrom, Denton P.	Minneapolis
Engstrom, Robert	Mankato
Enroth, Oscar E.	St. Paul
† Ensrud, Earl Richard	Rochester
Eppard, Raymond M.	Cloquet
Erdal, Ove A.	Albert Lea
Erich, John B.	Rochester
Erickson, Alvin O.	Long Prairie
Erickson, Clifford O.	Minneapolis
Erickson, Donald J.	Rochester
Erickson, Eskil	Halstad
Erickson, George P.	Hibbing
Erickson, John G.	Rochester
Erickson, Laurence F.	Minneapolis
Erickson, Myron E.	Minneapolis
Erickson, Reuben F.	Minneapolis
Erickson, Vernon D.	Grand Rapids
Ericson, Reinhold M.	Wayzata
† Ericson, Swan	Le Sueur
† Ernest, Geo. C. H.	St. Petersburg, Fla.
Ersfeld, Murray P.	St. Paul
Erskine, Gordon M.	Grand Rapids
† Ertel, Edward Q.	Ellendale
Esensten, Sidney	Minneapolis
† Eshelby, E. C.	St. Paul
Estes, J. Earle	Rochester
Estrem, Ralph L.	Fergus Falls
Estrem, Robert D.	Fergus Falls
† Eusterman, George B.	Rochester
Eustermann, John J.	Mankato
Evans, Edward T.	Minneapolis
Evans, Leslie	Sauk Rapids
Evans, Robert D.	Minneapolis
† Evarts, Arrah B.	Rochester
Evensta, John B.	Grand Rapids
‡ Evert, John A., Jr.	St. Paul
Ewen, Edgar F.	Rochester
Faber, John E.	Rochester
† Fabi, Mario Nestor	Rochester
† Fahr, George E.	Minneapolis
Fallon, Virgil T.	Dawson
Falls, John L.	Red Wing
Fansler, Walter A.	Minneapolis
Farkas, John V.	St. Paul
Farley, Frank G.	Hibbing
Fasbender, Herman T.	Hastings
Faucett, Robert L.	Rochester
Faulconer, Albert, Jr.	Rochester
Fawcett, Arthur M.	Renville
Fawcett, Keith R.	Duluth
Fee, John G.	St. Paul
Feeney, John M.	Minneapolis
‡ Feigal, David W.	A. F. B., Wyoming
Feigal, William M.	Thief River Falls
Feinberg, Milton	St. Paul
Feinberg, Philip	Minneapolis
Feinstein, Julius Y.	Minneapolis
Felder, Davitt A.	St. Paul
Feldmann, Floyd M.	N. Y., N. Y.
Felion, Arthur J.	St. Paul
Fellows, Manley F.	Duluth
Fenger, E. P. K.	Oak Terrace
Fergeson, James O.	Rochester
* Ferguson, James C.	St. Paul
Ferguson, William C.	Walnut Grove
‡ Ferrell, C. R.	San Antonio, Texas
Ferris, Deward O.	Rochester
Fesenmaier, Otto B.	New Ulm
Fesler, Harold H.	St. Paul
‡ Fetzek, Albert D.	St. Michael
Feuling, John C.	Duluth
‡ Fidelman, N. E.	Ft. Sheridan, Ill
Field, Anthony H.	Farmington
Field, Charles W.	Minneapolis
Fieldman, E. Jay	Rochester
† Fifer, William R.	St. Paul
Fifield, Malcolm M.	Duluth
Figi, Frederick A.	Rochester
Fingerman, David L.	Minneapolis
Fink, Daniel L.	St. Paul
Fink, Leo W.	Minneapolis
Fink, Walter H.	Minneapolis
Finkelnburg, William O.	Winona
Fisch, Herbert M.	Austin
Fischer, John R.	Blooming Prairie
Fischer, Marion McC.	Duluth
Fisher, Dan W.	St. Paul
Fisher, Isadore I.	Minneapolis
Fisketti, Henry	Duluth
† Fitzgerald, Don F.	Wayzata
*†Fitzgerald, Edward T.	Rialto, Calif.
Fitzsimons, William E.	Brainerd
† Fjeldstad, Christian A.	Minneapolis
Flanagan, Harold F.	St. Paul
Flanagan, Leonard G.	Austin
Flancher, Leon H.	Crookston
Flannery, Hubert F.	St. Paul
Fleeson, William H.	Minneapolis
Fleming, Dean S.	Hopkins
Fleming, Thomas N.	St. Cloud
Flesche, Bernard A.	Lake City
Fliehr, Richard R.	Minneapolis
Flink, Edmund B.	Minneapolis
Flinn, James B.	Redwood Falls
† Flinn, Thomas E.	Remer
Floersch, Adrian J.	Glencoe
Flom, Martin G.	Moorhead
Flom, Reynold P.	St. Paul
‡ Flom, Robert S.	Columbus, Ga.
Florine, Martin C.	Janesville
Fly, Orceneth A., Jr.	Rochester
Flynn, Bernard F.	Hibbing
Flynn, L. L., Jr.	St. Paul
† Fogarty, C. Wm., Sr.	St. Paul
Fogarty, C. Wm., Jr.	St. Paul
Fogelberg, Emil J.	St. Paul
Foker, Leslie W.	Minneapolis
Foley, Frederick E. B.	St. Paul
Folken, Frank G.	Albert Lea
Folsom, Louis B.	Minneapolis
Ford, Burton C.	Marshall
Ford, William H.	Minneapolis

Forsythe, James R....St. Paul
Fortier, George M. A....Little Falls
Foss, Edward L....Rochester
Foster, Orley W....Minneapolis
Fowler, Lucius Haynes....Minneapolis
Fox, Donald P....Minneapolis
Fox, James Rogers....Minneapolis
Fox, LeRoy J....St. Paul
† Franchere, Fred. Wm....Lake Crystal
Francis, David W....Morristown
Frane, Donald B....Minneapolis
† Frear, Rosemary....Minneapolis
Fredericks, George M....Minneapolis
Frederickson, Alice C....Willmar
Fredricks, Merriam G....Duluth
Freeman, Charles D., Jr....St. Paul
Freeman, Craig....Minneapolis
Freeman, Donald W....St. Louis Park
Freeman, Gerald I....St. Paul
† Freeman, John P....Glenville
Friedman, Louis L....St. Paul
French, Bayard T....Hibbing
French, Lyle A....Minneapolis
Frey, Richard J....Minneapolis
Friberg, Joseph....Minneapolis
Fricke, Robert E....Rochester
Fried, Louis A....Minneapolis
Friedell, Aaron....Minneapolis
Friedell, George....St. Louis Park
Friedman, Harry S....Minneapolis
Friedman, Jack....Minneapolis
Friend, Charles A....Minneapolis
Fritsche, Albert....New Ulm
Fritsche, Carl J....New Ulm
Fritsche, Theodore R....New Ulm
Fritz, Wallace L....St. Paul
Froats, Charles W....St. Paul
† Frost, John B....Minneapolis
Frost, Russell H....Oak Terrace
Frykman, Howard M....Minneapolis
Fugina, George R....Mankato
Fuller, Alice H....Minneapolis
Fuller, Benjamin F....St. Paul
Fuller, Josiah....Duluth
Funk, Victor K....Oak Terrace
Furlow, William L....Faribault
Furman, Lucie C....Minneapolis
Furr, Leo O....Bird Island

Gaard, Richard C....Minneapolis
Gacusana, Jose M....Mahnomen
Gaebe, Milton B....Clara City
Gaida, Joseph B....St. Cloud
† Gallagher, Bernard J....Waseca
Gallett, Lester E....Minneapolis
Galligan, John J....St. Paul
† Galligan, Margaret M....Minneapolis
* Galloway, John B....Minneapolis
† Gambill, Carl M....Rochester
Gambill, Earl E....Rochester
Gamble, Elbert J....Bricelyn
Gammell, John H....Minneapolis
Garbrecht, Arthur Wm....St. Paul
Gardner, Victor H., Sr....Fairmont
Gardner, Walter P....St. Paul
Garlock, Arthur V....Bemidji
Garlock, Dewitt H....Highlands, Calif.
Garrett, Charles M., Jr....Rochester
Garrow, David M....St. Paul
Garske, George L....Minneapolis
Garten, Joseph L....Minneapolis
Garvey, James T....Minneapolis
‡ Gastineau, Clifford F....Rochester
† Gatchell, Frank G....Rochester
Gaunt, William D....Rochester
Gaviser, David....Minneapolis
Gee, Vernon R....Rochester
Gehlen, Joseph N....St. Paul
Geib, Marvin J....Moorhead
Geiser, Peter M....Alexandria
Geraci, Joseph E....Rochester
Geurs, Benjamin R....Mankato
* Ghent, Charles H....St. Paul
Ghormley, Ralph K....Rochester
Ghostley, Mary C....Bemidji
Gibbs, Edward C....St. Paul
Gibbs, Robert W....Minneapolis
Giebenhain, John N....Minneapolis
Giere, Joseph C....Minneapolis
Giere, Richard W....Minneapolis
Giere, Silas W....Benson
Giesen, Allan F....Starbuck
† Giffin, Herbert Z....Rochester
Giffin, Mary E....Rochester
Gifford, R. W., Jr....Silver Springs, Md.
Gilbert, Maurice G....Minneapolis
Gillespie, Delmar R....St. Paul
Gillespie, Malcolm G....Duluth
Giman, Lloyd C....Willmar
† Gilmore, Rowland....Crookston
‡ Gilsdorf, Donald A....St. Paul
Gingold, Benjamin A....Minneapolis
‡ Ginsberg, R. L....San Antonio, Texas

† Girvin, Richard B....Minneapolis
Gjerde, William P....Lake City
Glabe, Robert A....Plainview
Glaeser, John H....Minneapolis
Gleason, Wallace A....St. Paul
† Glew, Wm. Bainbridge....Rochester
Goblirsch, Andrew P....Sleepy Eye
Goehrs, Gilman H....St. Cloud
† Goehrs, Henry W....St. Cloud
Goff, John R....Rochester
Goldberg, Isadore M....Minneapolis
Goldish, Daniel R....Duluth
Goldish, Robert J....Duluth
Goldman, Theodore I....Minneapolis
Goldner, Meyer Z....Minneapolis
Goldsmith, Joseph W....St. Paul
‡ Goldstein, Norman P....Bethesda, Md.
† Goltz, Edward V....St. Paul
Goltz, Robert W....Minneapolis
Good, C. Allen, Jr....Rochester
Good, Hoff D....Minneapolis
Good, Roy H....Glenwood
Goodchild, William R....Minneapolis
Goodman, Charles E....Virginia
Gordon, Philip E....Minneapolis
Gordon, Sewell S....St. Louis Park
Gowan, Lawrence R....Duluth
† Grace, Joseph B....Rochester
† Graham, Archibald W....Chisholm
Grahek, Jack P....Ely
‡ Grais, Melvin L....Colo. Springs, Colo.
Granquist, Richard D....Chisholm
Grant, Hendrie W....St. Paul
Grant, John C....Sauk Centre
Grant, Suzanne....Minneapolis
Grattan, Robert T....Rochester
Gratzek, Frank R. E....Minneapolis
Gratzek, Thomas....St. Paul
Grau, R. K....St. Paul
Graves, Richard B....Red Wing
Gray, Edward F....White Bear
† Gray, Frank D....Marshall
Gray, Howard K....Rochester
Gray, Royal C....Minneapolis
Green, Paul A....Rochester
Green, Robert A....St. Louis Park
† Green, Robert C., Jr....LaGrosse, Va.
Greenberg, Albert J....Minneapolis
Greene, Daniel E....Thief River Falls
Greene, Laurence F....Rochester
Greenfield, Irving....Minneapolis
Greenfield, William T....Cokato
† Greishiemer, Esther M.
Philadelphia, Pa.
Gridley, John W....Arlington
Griebie, Grant L....Brownton
Griffin, George D., Jr....Rochester
Griffin, John W., Jr....Bemidji
Griffin, R. P....Benson
Grimes, Burton P....St. Peter
Grimes, Marian....Minneapolis
Grimmell, Francis J....Minneapolis
Grindlay, John H....Rochester
Grinley, Andrew V....Grand Rapids
Grise, William B....Austin
Groch, Sigmund N....Rochester
Grohs, William H....Duluth
Gronvall, Paul R....Minneapolis
Groschupf, Theodore P....Bemidji
Grose, Frederick N....Clarissa
Grosh, John Livingston....Rochester
Gross, John B....Rochester
Grotting, John K....Minneapolis
*†Gruenhagen, Arnold P....St. Paul
Grundset, Ole J....Montrose
Guilbert, G. D....Minneapolis
Guilfoile, Pierre J....Delano
Gullickson, Glenn, Jr....Minneapolis
† Gullickson, Andrew....Longmont, Colo.
Gully, Raymond J....Cambridge
Gunlaugson, Frederick C....Minneapolis
Gushurst, Edward G....Minneapolis
Gustason, Harold T....Minneapolis
‡ Guy, Jack A....New London

Haas, J. F....Fairfax
Haavik, John E....Duluth
Habein, Harold C., Sr....Rochester
‡ Habein, H. C., Jr....Wiesbaden, Ger.
Haherer, Helen R....Minneapolis
† Haberman, Emil....Osakis
Haes, Julius E....Mankato
‡ Haesly, Warren W....Winona
‡ Hagedorn, Albert B....New York, N. Y.
Hagen, Kristofer....Richfield
† Hagen, Olaf J....Moorhead
Hagen, Paul S....St. Paul
Hagen, Wayne S....Minneapolis
† Haggard, G. D....Minneapolis
† Haigler, Frank H., Jr....Rochester
*†Haines, James H....Stillwater
Haines, Samuel F....Rochester
Hakanson, Erick Y....St. Paul

Halbert, John J....Duluth
Halenbeck, Philip L....St. Cloud
Hall, Barnard....St. Paul
Hall, Harry B....Minneapolis
Hall, Wendell H....Minneapolis
Hall, William E....Maple Lake
* Hallberg, Charles A....Minneapolis
Hallberg, Olav E....Rochester
† Hallenbeck, Dorr F....Rochester
Hallenbeck, George A....Rochester
† Halliday, Phillip V....Minneapolis
Hallin, Roger P....Worthington
Halloran, Walter H....Jackson
‡ Halme, W. B....San Francisco, Calif.
Halper, Bernard....Hibbing
Halpern, David J....Brewster
Halpin, Joseph E....Rush City
Halvorsen, Daniel K....Owatonna
Halvorson, James W....Zumbrota
Hamlon, John S....Fergus Falls
Hammar, Lawrence M....Mankato
Hammes, Ernest M., Sr....St. Paul
Hammes, Ernest M., Jr....St. Paul
*†Hammond, James F....St. Paul
† Haney, Claude L....Duluth
Hankerson, Robert G....Minnesota Lake
Hanlon, David G....Rochester
Hannah, Hewitt B....Minneapolis
Hanover, Ralph D....International Falls
Hansen, Cyrus O....Minneapolis
Hansen, Erling W....Minneapolis
Hansen, Milo Luverne....Little Falls
Hansen, Olga S....Minneapolis
Hansen, Robert E....Hibbing
‡ Hansen, Rollin M....Falls Church, Va.
Hansen, Theodore M....Albert Lea
† Hanson, Adolph M....Faribault
Hanson, Ernest O....Cloquet
Hanson, Everett C....New York Mills
Hanson, Harlow J....Minneapolis
Hanson, Harold B....St. Paul
Hanson, Henry V....Minneapolis
Hanson, LeRoy W....Pelican Rapids
Hanson, Lewis....Frost
Hanson, Malcolm B....Minneapolis
Hanson, Mark C. L....Minneapolis
Hanson, Norbert O....Rochester
Hanson, William A. H....Minneapolis
Happe, Lawrence J....Minneapolis
Harada, Thomas T....Lake Wilson
Harbaugh, John T....St. Paul
† Hardy, William M....Rochester
Hare, Robert L....Rochester
Harmon, Gaius E....St. Paul
Harrington, Stuart W....Rochester
Harrington, Vernon A., Jr....Duluth
Harris, Carl N....Hibbing
† Harris, Evelyn S....Seattle, Wash.
Harris, Leon D....Minneapolis
Harris, Lloyd E....Rochester
Harrison, Percy W....Worthington
Hart, William E....Monticello
Hartfiel, Herbert A....Montevideo
Hartfiel, William F....St. Paul
Hartig, Marjorie....St. Paul
Hartjen, Jason K....Bemidji
Hartley, Everett C....St. Paul
Hartman, Evelyn E....Minneapolis
Hartman, Howard R....Rochester
Hartnagel, G. F....Red Wing
Hartung, Elmer H....Claremont
Hartwich, Roger F....Winona
Hass, Frederick M....Minneapolis
† Hassett, Roger G....Mankato
Hastings, DeForest R....Minneapolis
Hastings, Donald W....Minneapolis
† Hatch, Walter E....Duluth
Haugan, Norman R....Crookston
Hauge, Erling T....Minneapolis
Hauge, Malvin I....Clarkfield
Haugen, George W....Minneapolis
Haugen, John A....Minneapolis
Hauser, Donald C....Minneapolis
Hauser, George W....Minneapolis
Hauser, Victor P....St. Paul
Havel, Robert J....Minneapolis
Haven, Walter K....Minneapolis
Havens, Fred Z....Riverside, Calif.
† Havens, John G. W....Austin
Hawkinson, Raymond P....Minneapolis
Hawley, George M. B., II....Red Wing
Hay, Lyle J....Minneapolis
Hayes, Albert F....St. Paul
† Hayes, James M....Minneapolis
*†Hayes, Michael F....Moose Lake
Hayles, Alvin B....Rochester
Hays, Albert T....Minneapolis
Head, Douglas....Minneapolis
Healy, John R....Rochester
Healy, Raymond T....Pierz
Hebbel, Robert....Minneapolis
Hebeisen, Milton B....Chaska
Heck, Frank J....Rochester

	Heck, William W.	St. Paul
	Hedberg, Gustaf A.	Nopeming
	Hedemark, Homer H.	Ortonville
	Hedemark, Truman A.	Ortonville
	Hedenstrom, Frank G.	St. Paul
	Hedenstrom, Paul H.	Cambridge
	Hedenstrom, Philip C.	Marshall
	Hedin, Raymond F.	Red Wing
	Hedlund, Charles J.	Owatonna
	Heegaard, William G.	Alexandria
†	Hegge, Olav H.	Austin
†	Hegge, Rolv S.	Austin
	Heiam, William C.	Cook
	Heiberg, Emmett A.	Fergus Falls
	Heiberg, Olaf M.	Worthington
	Heilig, William R.	St. Paul
†	Heilman, Dorothy M. H.	San Diego, Calif.
	Heilman, Fordyce R.	Rochester
	Heimark, John J.	Rochester
	Heimark, Julius J.	Fairmont
	Heine, George W.	Little Falls
	Heinz, Ivy B.	Shakopee
	Heinz, Lawrence H.	Shakopee
	Heise, Carl vR.	Winona
	Heise, Herbert vR.	Winona
	Heise, Paul vR.	Winona
	Heise, Philip vR.	Winona
	Heise, William vR.	Winona
‡	Heisler, John J.	Minneapolis
	Helferty, John K.	Boise, Idaho
*†	Helland, Gustav M.	Spring Grove
†	Helland, John W.	Spring Grove
	Heller, Edgar E.	Mankato
†	Helmholz, Henry F.	Rochester
	Helseth, Hovald K.	Pelican Rapids
	Hempel, Dean J.	Minneapolis
†	Hempstead, B. E.	Rochester
	Hench, Philip S.	Rochester
	Henderson, Arthur J. G.	No. St. Paul
	Henderson, Edward D.	Rochester
	Henderson, John W.	Rochester
	Henderson, Lowell L.	Rochester
*†	Henderson, Melvin S.	Rochester
†	Henderson, Robert Earl	Rochester
	Hendricks, Esten J.	St. Paul
†	Hendrickson, John F.	Minneapolis
	Hengstler, William H.	St. Paul
*†	Henney, William H.	McIntosh
	Henrikson, Earl C.	Minneapolis
	Henry, Clarence J.	Milaca
†	Henry, Clifford E.	Kirksville, Mo.
‡	Henry, Harold W.	Benton, Ill.
‡	Henry, J. E.	Milaca
	Henry, Kenneth G.	Owatonna
	Henry, Martin R.	St. Peter
†	Hensel, C. N.	St. Paul
	Hepper, Norman G.	Rochester
	Herbert, Willis	Minneapolis
	Herman, Samuel M.	St. Paul
	Hermann, Harold W.	Minneapolis
	Hermanson, Peter E.	Hendricks
	Heron, Roy C.	St. Paul
	Herrmann, Edgar T.	St. Paul
†	Hertel, Garfield E.	Austin
	Hertz, Myron J.	St. Paul
	Hesla, Inman A.	Austin
†	Hewitt, Edith S.	Rochester
†	Hewitt, Richard M.	Rochester
†	Heyerdale, O. C.	Rochester
†	Higgins, John H.	Minneapolis
	Higgs, Walter W.	Park Rapids
	Hildebrand, J. E.	Bemidji
	Hilding, Anderson C.	Duluth
†	Hilger, Andrew Wm.	St. Paul
	Hilger, Jerome A.	St. Paul
	Hilger, Laurence D.	St. Paul
	Hilker, Marcus D.	St. Paul
	Hill, Earl	Minneapolis
	Hill, Elmer M.	Minneapolis
†	Hill, Frederick E.	Riverside, Calif.
	Hill, John P.	Virginia
	Hill, John R.	Rochester
†	Hill, Richard Woolsey	Rochester
†	Hillis, Samuel J.	East Bradenton, Fla.
	Hinckley, Robert G.	Minneapolis
	Hinderaker, Harris P.	Bird Island
	Hines, Edgar A., Jr.	Rochester
	Hiniker, Louis P.	St. Paul
	Hiniker, Peter J.	Le Sueur
	Hinz, Walter E.	Willmar
	Hirschboeck, Frank J.	Duluth
	Hirsh, Stanton A.	Crookston
	Hirshfield, Frank R.	Minneapolis
	Hitchcock, Claude R.	Minneapolis
	Hochfilzer, John J.	St. Paul
	Hodapp, Robert V.	Willmar
	Hodgson, Corrin H.	Rochester
	Hodgson, Jane E.	St. Paul
	Hodgson, John R.	Rochester
	Hoehn, David	Holdingford
	Hoeper, Philip G.	Mankato
	Hoff, Herbert O.	Duluth
	Hoffbauer, Frederick	Minneapolis
	Hoffert, Henry E.	Minneapolis
	Hoffman, Roy A.	Minneapolis
	Hoffman, Walter L.	Minneapolis
	Hoganson, D. E.	Bemidji
†	Hoidale, Andrew	Tracy
	Holcomb, Joel T.	Marine-on-St. Croix
†	Holcomb, O. W.	St. Paul
	Holian, Darwin K.	Albert Lea
	Holland, C. R.	Rochester
†	Hollands, William H.	Fisher
	Hollenhorst, Robert W.	Rochester
	Hollinshead, W. H.	St. Paul
	Holm, Donald F.	Benson
	Holman, Colin B.	Rochester
	Holmberg, Conrad J.	Minneapolis
	Holmen, Robert W.	St. Paul
	Holmes, Alva E.	Rush City
	Holmstrom, Carle H.	Warren
	Holt, Allen H.	Rochester
	Holt, John E.	St. Paul
	Holzapfel, Fred C.	Minneapolis
	Honath, Donald H.	Owatonna
	Hoover, Norman W.	Virginia
	Hopkins, George W.	St. Paul
‡	Horns, H. L.	Colo. Springs, Colo.
	Horns, Richard C.	Minneapolis
	Horton, Bayard T.	Rochester
	Hottinger, Raymond C.	Janesville
	Houglum, Arvid J.	Lake Park
†	Houkom, Bjarne	T. T., East Africa
	Houkom, Samuel S.	Duluth
†	House, Zachariah E.	Burbank, Calif.
	Houston, Donald M.	Park Rapids
	Houts, Joseph C.	Dassel
	Hovde, Rolf	Winthrop
	Hovland, Melvin L.	Minneapolis
	Howard, Marshall I.	Mankato
	Howard, Merrill A.	St. Paul
	Howard, Robert B.	Minneapolis
	Howard, Solomon E.	Minneapolis
	Howard, Willard S.	St. Paul
	Howe, Newell W.	St. Paul
	Howell, Carter W.	Minneapolis
	Howell, Llewelyn P.	Rochester
	Howell, Milton M.	Gencoe
	Hoyer, Ludolf J.	Windom
	Hruza, William J.	Madelia
*	Hubbard, Otto E.	Brainerd
	Hubin, Edwin G.	Sandstone
	Hudec, Elwyn R.	Echo
	Hudson, George E.	Minneapolis
	Huebert, Dan W.	Hutchinson
	Huenekens, Edgar J.	Minneapolis
†	Huffington, H. L.	Mankato
	Huffington, Herb L., Jr.	Waterville
	Hughes, Bernard J.	Brainerd
	Hughes, Sidney O.	Winona
	Huizenga, Kenneth A.	Rochester
	Hullsiek, Harold E.	St. Paul
	Hullsiek, Richard B.	Ft. Snelling
	Hult, John E.	Cloquet
	Hultkrans, Rudolph E.	Minneapolis
	Humphrey, E. W., Sr.	Moorhead
	Humphrey, Wade R.	Stillwater
	Hunt, Arthur B.	Rochester
†	Hunt, R. C.	Fairmont
	Hunt, William	Fergus Falls
	Hunter, James S.	Rochester
	Hunter, Murray H.	Farmington
†	Hurd, Annah	Minneapolis
	Hurwitz, Milton M.	St. Paul
	Hustad, Edward G.	Montevideo
	Hutchinson, Dorothy W.	Oak Terrace
	Hutchinson, Henry	Moose Lake
†	Huxley, Frederick R.	Faribault
	Hymes, Charles	Minneapolis
†	Hynes, John E.	Minneapolis
†	Ide, Arthur W., Sr.	St. Paul
	Ide, Arthur W., Jr.	Minneapolis
	Idstrom, Linneus G.	Minneapolis
	Ikeda, Kano	St. Paul
‡	Indihar, John E., Jr.	Oakland, Calif.
†	Ingerson, Carl A.	St. Paul
	Inglis, William	Redwood Falls
†	Irvine, Harry G.	Minneapolis
	Irwin, Homer R.	Hibbing
	Iverson, Rolf M.	Minneapolis
	Ivins, John C.	Rochester
	Jackman, Raymond J.	Rochester
	Jacks, Quentin D.	Rochester
	Jackson, Richard L.	Rochester
	Jackson, William C.	St. Paul
	Jacobs, Douglas L.	Willmar
†	Jacobs, Johannes C.	Willmar
	Jacobson, Clarence	Chisholm
	Jacobson, Clifford W.	Breckenridge
	Jacobson, Ferdinand C.	Duluth
	Jacobson, Loren J.	Minneapolis
	Jacobson, Wyman E.	St. Louis Park
	James, Ellery M.	St. Paul
	James, John W.	Mahtomedi
	Janecky, Allen G.	Baudette
	Janes, Joseph M.	Rochester
	Janssen, Martin E.	St. Paul
†	Jarvis, Bruce W.	St. Paul
	Jarvis, Marilyn A.	St. Paul
	Jay, Alan R.	Minneapolis
	Jeffries, William L.	Minneapolis
‡	Jensen, Adrian R.	Roslindale, Mass.
	Jensen, Alvin M.	Brownton
	Jensen, Harry C.	Minneapolis
†	Jensen, Marius J.	Minneapolis
	Jensen, Nathan K.	Minneapolis
	Jensen, Reynold A.	Minneapolis
	Jensen, Thorvold J.	Duluth
	Jenson, James E.	Stillwater
	Jerome, Bourne	Minneapolis
	Jerome, Elizabeth E.	Minneapolis
	Jeronimus, Henry J.	Duluth
	Jesion, Joseph W.	St. Paul
	Jessico, Charles M.	Duluth
‡	Jeub, Robert P.	San Antonio, Texas
	Joffe, Harold H.	Virginia
	Johanson, Waldemar G.	St. Paul
	Johnsen, Henry A., Jr.	Virginia
	Johnson, Adelaide McF.	Rochester
	Johnson, Aldridge F.	Isle
	Johnson, Arthur B.	Minneapolis
	Johnson, August E.	Minneapolis
	Johnson, Carl E.	St. Paul
	Johnson, Carl Eric	Rochester
	Johnson, Carolyn A.	St. Paul
‡	Johnson, Curtis M.	Dawson
	Johnson, C. Percy	Tyler
	Johnson, Douglas L.	Little Falls
‡	Johnson, Edward A.	Virginia
†	Johnson, Einer W., Sr.	Bemidji
	Johnson, Einer, W., Jr.	Rochester
	Johnson, Emil W.	Minneapolis
	Johnson, Frank E.	Minneapolis
	Johnson, Harry A.	Minneapolis
	Johnson, Herbert W.	St. Paul
	Johnson, Hobart C.	North Mankato
†	Johnson, James A.	Minneapolis
	Johnson, John W.	Minneapolis
†	Johnson, Julius	Minneapolis
	Johnson, Karl E.	Duluth
	Johnson, Malcolm R.	Minneapolis
	Johnson, Marvin W.	Milaca
†	Johnson, Morris B.	St. Paul
	Johnson, Norman P.	Minneapolis
	Johnson, Norton T.	Minneapolis
	Johnson, Olga H.	Moorhead
	Johnson, R. J.	St. Paul
	Johnson, Ralph B.	Lanesboro
*	Johnson, Ray G.	Stillwater
	Johnson, Reinald G.	Minneapolis
	Johnson, Reuben A.	Minneapolis
	Johnson, Robert E.	Minneapolis
	Johnson, Roger S.	Wayzata
	Johnson, Rudolph E.	Mankato
	Johnson, Vilheim M.	Dawson
	Johnson, William E.	Morgan
	Johnson, Youbert T.	Minneapolis
	Johnsrud, Luverne W.	Hibbing
	Johnston, Henry W.	Virginia
	Johnston, Leonard F.	Winona
†	Johnston, R. O.	Fort Lauderdale, Fla.
	Johnstone, William W.	Ah-gwah-ching
	Jolin, Francis M.	Bovey
†	Jones, Alvah W.	Red Wing
	Jones, David G.	Minneapolis
†	Jones, E. Mendelssohn	St. Paul
†	Jones, H. W., Jr.	Brookline, Mass.
	Jones, Orville H.	Mankato
	Jones, Richard H.	Minneapolis
	Jones, Richard N.	St. Cloud
	Jones, William R.	Minneapolis
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	Jordan, Lewis S.	Granite Falls
†	Jordan, Stanley	Rochester
	Josewich, Alexander	Minneapolis
	Josewski, Raymond J.	Stillwater
	Joyce, George L.	Rochester
	Judd, Allen S.	Minneapolis
	Judd, Edward S., Jr.	Rochester
	Judd, Walter H.	Washington, D. C.
	Juergens, Herman M.	Belle Plaine
†	Juergens, John L.	Belle Plaine
	Juergens, Manley F.	Stillwater
	Juers, Edward H.	Red Wing
†	Juliar, Richard O.	Los Angeles, Calif.
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	Jurdy, Mitchell J.	Minneapolis
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	Kabrick, Ola A.	St. Peter
	Kadesky, Harold B.	Minneapolis
	Kaiser, Milton L.	New Ulm
	Kalin, Oscar T.	Minneapolis
	Kallestad, Leonard L.	Minneapolis
	Kamman, Gordon R.	St. Paul
*†	Kamp, Byron A.	Albert Lea
	Kanne, Earl R.	Brainerd

Kantar, Bruce L.	Minneapolis
Kaplan, David H.	St. Paul
Kaplan, Harold A.	Minneapolis
Kaplan, John J.	Minneapolis
Kapsner, Alfred T.	Princeton
Karleen, Bernard N.	Jackson
Karleen, Conrad I.	Minneapolis
Karlen, Markle	Minneapolis
Karn, Jacob F.	Ortonville
Karon, Irvine M.	St. Paul
Kasper, Eugene M.	St. Paul
Kath, Reinhard H.	Arlington
Katz, Louis J.	Long Beach, Calif.
Katz, Yale J.	Minneapolis
Kaufman, Edward J.	Appleton
Kaufman, Herschel J.	Minneapolis
Kaufman, Walter B.	Mankato
Kaufman, William C.	Appleton
Kearney, Rochfort W.	Mankato
Kearns, Thomas P.	Rochester
Keating, Francis R., Jr.	Rochester
Keefe, Rolland E.	St. Paul
Keil, Marcus A.	Albert Lea
Keith, Haddow M.	Rochester
Keith, Norman M.	Rochester
Keith, Paul J.	Milaca
Keithahn, Elmer E.	Sleepy Eye
Kelby, Gjert M.	Minneapolis
Kelley, Roger E.	Crosby
Kelley, Walter M.	Duluth
Kelly, Albert C.	Duluth
Kelly, Charles F.	Minneapolis
Kelly, James H.	St. Paul
Kelly, John F.	Cold Spring
Kelly, John P.	Minneapolis
Kelly, John V.	St. Paul
Kelly, Robert T.	Nashwauk
Kelsey, Carleton G.	St. Paul
Kelsey, Chauncey M.	St. Paul
Kemp, Alphonse F.	Mankato
Kenefick, Emmett V.	St. Paul
Kennedy, Claude C.	Minneapolis
Kennedy, George L.	Faribault
Kennedy, Jane F.	Minneapolis
Kennedy, Roger L. J.	Rochester
Kent, George B.	Phoenix, Ariz.
Kenyon, Thomas J.	St. Paul
Kerkhof, Arthur C.	Minneapolis
Kernohan, James W.	Rochester
Kesting, Herman	St. Paul
Kevern, Jay L.	Henning
Keyes, John D.	Winona
Keyes, Robert W.	Pipestone
Keys, John R.	Rochester
Kiely, James P.	Rochester
Kiely, Joseph M.	Joliet, Ill.
Kierland, Robert R.	Rochester
Kiesler, Frank, Jr.	Minneapolis
Kilbride, Edwin A.	Worthington
Kilby, Ralph Allen	Rochester
Kimmel, George C.	Red Wing
King, Edgar A.	Minneapolis
King, Frances W.	Oak Terrace
† King, George L.	Hudson, Wis.
Kinkade, Byron R.	Ada
Kinports, Ed. B.	International Falls
Kinsella, Thomas J.	Minneapolis
Kippen, Neil	Breckenridge
Kirby, Thomas J., Jr.	Rochester
Kirklin, B. R.	Rochester
Kirklin, John W.	Rochester
Kistler, Alvin J.	Minneapolis
Kitzberger, Peter J.	New Ulm
Klakeg, Clayton H.	Rochester
‡ Klass, Donald W.	Rochester
Klefstad, Lloyd H.	Greenbush
Klein, Harry	Duluth
† Klein, Henry N.	St. Paul
Klein, William A.	Duluth
Klima, William W.	Stewart
Kline, Richard F.	Wayzata
Knapp, Frank N.	Duluth
Knapp, Miland E.	Minneapolis
Knight, Edwin G.	Swanville
† Knight, James Harry	Rochester
Knight, Ralph T.	Minneapolis
† Knight, Ray R.	Minneapolis
Knoche, Harvey A.	Morgan
Knoll, W. V.	Duluth
Knudsen, Helen L.	Minneapolis
Knutson, Gerhard E.	St. Paul
Knutson, Lewis A.	Spring Grove
Knutson, Robert C.	St. Paul
Koelsche, Giles A.	Rochester
Koenecke, Fred H., Jr.	Lakefield
‡ Koenig, Robert P.	Geneva, N. Y.
Koenigsberger, Charles	Mankato
Koepcke, Gerald M.	Minneapolis
Kohlbry, Carl O.	Duluth
† Kohler, Delphin W.	Tacoma, Wash.
Kolars, James J.	Faribault
Koller, Hermann M.	Minneapolis
Koller, Louis R.	Minneapolis
Koller, Robert L.	Minneapolis
Kooda, Jennings C.	Morris
Korchik, John P.	Minneapolis
Korda, Henry A.	Pelican Rapids
Kosiak, John, Jr.	Minneapolis
Koskela, Lauri E.	Shaker Heights, O.
Kostick, William R.	Fertile
Kotchevar, Frank R.	Eveleth
Kottke, Frederick J.	Minneapolis
Kotval, Russell J.	Pipestone
Koucky, Rudolph W.	Minneapolis
Kovack, Freeman D.	Minneapolis
Koza, Donald W.	St. Paul
Kozberg, Oscar	Moose Lake
Kraemer, George N.	Fairmont
Kragh, Lyle O.	Braham
Krause, C. W.	Fairmont
Kremen, Arnold J.	N. Y., N. Y.
Kreuzer, Titus C.	Marshall
Krezowski, Thomas K.	St. Paul
Krieser, Albert E.	Anoka
Kroboth, Frank J., Jr.	Rochester
‡ Kroll, Harry G.	Rochester
Krout, Robert M.	Rochester
Krueger, Victor R.	Nopeming
Krusen, Frank H.	Rochester
Kruzick, S. J.	Sleepy Eye
Krystosek, Lee A.	Clara City
Kucera, Frank J.	Hopkins
‡ Kucera, Louis B.	Colo. Springs, Colo.
Kucera, Stanley T.	Northfield
Kucera, William J., Sr.	Minneapolis
Kucera, William J., Jr.	Minneapolis
Kugler, Alex A.	St. Paul
Kuhlmann, Lawrence B.	Melrose
† Kuhn, Arthur J.	Rochester
Kulstad, Oscar S.	Dodge Center
Kulzer, Norbert J.	Hastings
Kunkel, Melvin G.	Rochester
Kunkel, Wm. M., Jr.	Harrisburg, Pa.
† Kurtin, Henry J.	Tuscon, Ariz.
‡ Kurtin, Joseph J.	N. Y., N. Y.
Kuske, Albert W.	St. Paul
† Kusske, Arthur L.	New Ulm
Kusske, Bradley, W.	St. Paul
Kusske, Douglas R.	St. Paul
Kusz, Clarence V.	Minneapolis
Kvale, Walter F.	Rochester
Kvitrud, Gilbert	St. Paul
LaBree, John W.	St. Louis Park
LaBree, Robert H.	Duluth
† LaGaard, Sheldon M.	Minneapolis
Lacy, Paul E.	Rochester
Laikola, Leslie A.	Adrian
† Laird, Arthur T.	Duluth
† Lajoie, John M.	Minneapolis
Lake, Clifford F.	Rochester
Lamb, H. Douglas	Minneapolis
Lang, Leonard A.	Minneapolis
‡ Lange, Robert D.	Washington, D. C.
Langhoff, Arthur H.	Mankato
Lannin, Bernard G.	St. Paul
Lannin, Donald R.	St. Paul
Lapierre, Arthur P.	Minneapolis
Lapierre, Jean T.	Minneapolis
Larrabee, Walter F.	St. Paul
Larsen, Frank Wm.	Minneapolis
Larson, Arnold	Detroit Lakes
Larson, Clarence M.	Minneapolis
Larson, Donald M.	Minneapolis
Larson, Eva Jane	St. Paul
Larson, Gerald E.	Cambridge
Larson, James T.	So. St. Paul
Larson, Kenneth R.	St. Paul
Larson, Leighton	Waconia
Larson, Lawrence M.	Minneapolis
Larson, Leonard M.	Oak Terrace
Larson, LeRoy J.	Bagley
Larson, Loren J.	Minneapolis
Larson, Martin L.	St. Paul
Larson, Milo H.	Lake Benton
Larson, Oliver E.	Red Wing
Larson, Paul N.	Minneapolis
Larson, Ralph H.	Anoka
† Larson, Roger C.	Minneapolis
Latterell, Kenneth E.	Duluth
† LaVake, Rae T.	Minneapolis
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Law, William M.	Rochester
Lax, Morris H.	St. Paul
Laymon, Carl W.	Minneapolis
*†Lazar, Henry L.	Excelsior
Lazarte, Jorge A.	Rochester
† Leahy, Bartholomew	St. Paul
Leavenworth, R. O., Sr.	St. Paul
Leavenworth, R. O., Jr.	St. Paul
† Leavitt, H. H.	Minneapolis
† Lebowske, Joseph A.	Minneapolis
Leck, Paul C.	Austin
Leddy, Eugene T.	Rochester
Leo, Hubert W.	Brainerd
Lee, Gordon E.	Glenwood
Lee, Norman J.	Tracy
Lee, Philip R.	Rochester
† Lee, Walter N.	Claremont, Calif.
‡ Leek, Joseph H.	Augusta, Ga.
Leemhuis, Andrew J.	Minneapolis
Leibold, Herbert H.	Parkers Prairie
Leick, Richard M.	St. Paul
Leiferman, Robert J.	Minneapolis
Leinonen, Wendla E.	Wadena
Leitch, Archibald	St. Paul
Leland, Harold R.	Minneapolis
*†Lemon, Willis S.	Clifton Forge, Va.
Lenander, Mellvin E.	St. Peter
Lenarz, Albert J.	Browerville
Lende, Norman	Faribault
Lenz, Joseph R.	Morton
Lenz, Otto A.	Minneapolis
Leonard, Lawrence J.	Minneapolis
Leonard, Samuel	Minneapolis
† Leopard, Brand A.	Brownsville, Texas
Lepak, Francis J.	Duluth
Lepak, John A.	St. Paul
Leppo, N. Erkki A.	Duluth
† Lerche, William	Cable, Wis.
Lerner, A. Ross	Minneapolis
Lester, Malcolm J., Jr.	Truman
Leuallen, Edmund C.	Rochester
Leven, N. Logan	St. Paul
Leverenz, Carleton W.	St. Paul
Levin, Bert G.	St. Paul
Levitt, George X.	St. Paul
Lewis, Arthur J.	Henning
Lewis, Barton L.	Rochester
Lewis, Charles W.	Henning
† Lewis, Claude B.	St. Cloud
Lewis, F. John	Minneapolis
† Lexa, Frank J.	Lonsdale
Libert, John N.	St. Cloud
Lick, Charles L.	St. Paul
Lick, Louis C.	Rochester
Lick, William J., Jr.	St. Paul
† Liedloff, Adolph G.	Mankato
Lien, Richard J.	St. Paul
Liffrig, William W.	Red Wing
† Lightbourn, Edgar L.	Hastings
Lilleberg, Norbert J.	St. Paul
Lillehei, C. Walton	Minneapolis
Lillehei, Elmer J.	Robbinsdale
† Lillie, Harold I.	Rochester
Lillie, John C.	Rochester
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‡ Lind, C. J., Jr.	Ft. S. Houston, Tex.
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Lindberg, Alfred L.	Wheaton
† Lindberg, Arvid C.	Minneapolis
Lindberg, Vernon L.	Minneapolis
Lindberg, Winston R.	Minneapolis
Lindblom, Alton E.	No. Mankato
Lindblom, Maurice L.	Minneapolis
† Linde, Herman	Cyrus
Lindell, Robert	St. Paul
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Lindner, Janus C.	Minneapolis
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† Linner, Henry P.	Minneapolis
Linner, John H.	Minneapolis
Linner, Paul W.	Minneapolis
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Lippman, Hyman S.	St. Paul
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Lippmann, Emanuel S.	Minneapolis
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Litman, Abraham B.	Minneapolis
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Logan, George B.	Rochester
† Logefeil, Rudolph C.	Minneapolis
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Loken, Selmur M.	St. Paul
Loken, Theodore	Ada
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Longfellow, Henry W.	Brainerd
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Louisell, Charles	Fairmont
Love, Frederick A.	Carlos
Love, J. Grafton	Rochester
Lovett, Beatrice R.	Oak Terrace

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Lowe, Earl R.....So. St. Paul
Lowe, Thomas A.....So. St. Paul
Lowry, Elizabeth C.....Minneapolis
Lowry, Paul.....Minneapolis
Lowry, Thomas.....Minneapolis
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‡ Luckemeyer, C. J.....Ft. Riley, Kansas
† Ludwig, Clarence John.....Rochester
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Lufkin, Nathaniel H.....Minneapolis
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Lund, George W.....Minneapolis
Lund, Werner J.....Staples
Lundberg, Ruth I.....Minneapolis
Lundblad, Robert M.....Clara City
Lundblad, Roy A.....Minneapolis
Lundblad, Stanley W.....Minneapolis
Lundeberg, Karl R.....Minneapolis
Lundell, Carl L.....Granite Falls
Lundholm, Arthur M.....St. Paul
Lundquist, Curt W.....Owatonna
Lundquist, Virgil J. P.....Minneapolis
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Lyons, James H.....Minneapolis
Lysne, Henry.....Minneapolis
Lysne, Myron.....Minneapolis
Lysyj, Anatol.....Minneapolis
Lyzenga, Anton G.....Minneapolis

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McCaffrey, Fabian J.....Minneapolis
McCain, Donovan L.....St. Paul
McCann, Eugene J.....Minneapolis
McCannel, Malcolm A.....Minneapolis
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McCarthy, Donald .....St. Paul
McCarthy, Joseph J.....St. Paul
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White Bear Lake
McClanahan, Thos. S.
White Bear Lake
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McDaniel, S. P.....Fergus Falls
† McDaniel, Orianna.....Minneapolis
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McDonald, John R.....Rochester
McDonald, Owen G.....Duluth
McDonald, William J.....Crookston
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McEnaney, Clifford T.....Owatonna
McEwan, Alexander.....St. Paul
McFarland, Arthur H.....Minneapolis
McFarlane, John A.....Rochester
McGandy, Robert F.....Minneapolis
McGeary, George E.....Minneapolis
McGroarty, Brian J.....St. Paul
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McHardy, Bryson R.....Karlstad
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McIntire, Homer M.....Waseca
McIntyre, John A.....Owatonna
McKaig, Alan M.....Red Lake Falls
McKaig, Carle B.....Pine Island
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McKenna, Elizabeth M.....Austin
McKenna, Jay K.....Austin
McKenna, Maurice J.....Grand Rapids
McKenzie, Charles H.....Minneapolis
McKenzie, Eva Elaine.....St. Paul
McKibben, Helena.....Rochester
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McKinney, Frank S.....Minneapolis
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McManus, William F.....Princeton
McMurtrie, William B.....Minneapolis
McNear, George R., Jr.....Mankato
McNeil, John J.....Minneapolis
McNeill, J. A.....St. Paul
McNutt, John R.....Duluth
McPheeters, Herman O.....Minneapolis
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McWhorter, Henry E.....Rochester

MacCarty, Collin S.....Rochester
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MacDonald, Daniel A.....Minneapolis
MacDonald, John W.....Minneapolis
MacDonald, Roger A.....Little Fork
* MacFarlane, P. Harvie.....Chisholm
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MacKinnon, Donald C.....Minneapolis
MacRae, Gordon C.....Duluth
Mach, Frank B.....Minneapolis
Macklin, W. E., Jr.....Willmar
‡ Mackoff, Sam M.....St. Paul
† Madalin, Herbert E.....Rochester
* Madden, John F.....St. Paul
Madison, Mitchell S.....Rochester
Madland, Robert S.....St. Paul
Maeder, Edward C.....Minneapolis
Maertz, William F.....St. Peter
Magath, Thomas B.....Rochester
Magney, Fredolph H.....Duluth
Magnuson, Allen E.....Wheaton
‡ Magnuson, Raymond C.....Cambridge
Mahle, Donald G.....Plainview
Mahowald, Aloys.....Fergus Falls
Maitland, Edwin T.....Jackson
† Maland, Clarence O .....Minneapolis
Malerich, J. Anthony.....St. Paul
Malerich, J. Anthony, Jr.....St. Paul
Malmstrom, John A.....Virginia
Mandel, Sheldon L.....Minneapolis
Manger, William M.....Rochester
Mankey, James C.....Minneapolis
Mankin, Harold T.....Cambridge, Mass.
Mankin, Haven W.....Rochester
Mann, Frank D.....Rochester
‡ Manning, Phil R.....Los Angeles, Calif.
† Manson, Frank M.....Worthington
March, Kenneth A.....Cambridge
† Marcley, Walter J.....Minneapolis
Marking, George H.....Minneapolis
Marks, Roger W.....St. Paul
Marrone, Patrick H.....Duluth
Marshall, Clark M.....Crosby
Martens, Theodore G.....Rochester
Martin, Albert C.....Luverne
Martin, Dwight L.....St. Paul
Martin, George B.....Thief River Falls
Martin, George R.....Minneapolis
Martin, Gordon M.....Rochester
† Martin, Thomas Philip.....Arlington
Martin, Webster C.....Duluth
Martin, William B.....Duluth
Martin, William J.....Rochester
Martineau, Joseph L.....St. Paul
Martinson, Carl J .....Wayzata
Martinson, Elmer J.....Wayzata
Masson, Duncan M.....Rochester
† Masson, James C.....Rochester
Masson, James K.....Rochester
Mateo, Guillermo.....St. Paul
Mathieson, Don R.....Rochester
Matthews, James H.....Minneapolis
Mattill, Peter M.....Oak Terrace
Mattison, Percy A.....Winona
Mattson, Albert D.....St. James
Mattson, Hamlin A. N.....Minneapolis
‡ Maus, Philip.....New Orleans, La.
Maxeiner, Stanley R., Sr.....Minneapolis
Maxeiner, Stanley R., Jr.....Minneapolis
Mayne, John Gregory.....Rochester
Mayne, Roy M .....Nopeming
Mayo, Charles W.....Rochester
Mazzitello, William F.....St. Paul
Mead, Charles H .....Duluth
Meade, John R.....St. Paul
Mears, Burtis J.....St. Paul
Mears, Robert F.....Northfield
Medelman, John P.....St. Paul
Meinert, Albert E.....Winona
Melancon, Joseph F.....St. Paul
† Melby, Benedik.....Blooming Prairie
Meller, Maurice.....Brainerd
Meller, Robert L.....Minneapolis
† Melzer, George R.....Lyle
Menges, Charles G. H.....Rochester
Menold, William F.....St. Paul
Mensheha, Nicholas.....Forest Lake
Mercil, William F.....Crookston
Merkert, Charles E.....Minneapolis
Merkert, George L.....Minneapolis
Merner, Thomas B.....Faribault
† Merrick, Charlotte T.....St. Paul
Merrick, Robert L.....St. Paul
Merrill, Robert W.....Morris
Merriman, Lloyd L.....Duluth
Merritt, Wallace A.....Rochester
† Mesker, George H.....Olivia
Metcalf, Norman B.....Princeton
Metz, Donald D.....Buffalo Lake
Meyer, Alvin J.....Minneapolis
Meyer, Anthony A.....Melrose
† Meyer, Ette L.....Minneapolis
Meyer, Fredk. C.....Kenyon

Meyer, Paul F.....Faribault
Meyer, Robert J.....Minneapolis
‡ Meyer, Robert P.....Faribault
† Meyerding, Edward A.....St. Paul
† Meyerding, Henry Wm.....Rochester
Michael, Joseph C.....Minneapolis
Michel, Henry H.....Minneapolis
Michels, Roger P.....Willmar
Michelson, Henry E.....Minneapolis
Michienzi, Leonard J.....St. Paul
† Mickelsen, Emma F.....Minneapolis
Mickelson, John C.....Mankato
Midboe, Gilbert T.....St. Paul
Midthune, A. S.....Lake Park
Miettunen, John B.....Chisholm
Milhaupt, E. N.....St. Cloud
Miller, Albert G.....St. Paul
Miller, Arden L.....Minneapolis
Miller, Harold E.....Minneapolis
‡ Miller, Herman.....Grand Meadow
Miller, Hugo E.....Minneapolis
Miller, John C.....Minneapolis
Miller, Roland D.....Rochester
† Miller, Victor I.....Mankato
† Miller, William Anthony.....N. Y. Mills
Miller, William J.....Ft. Wayne, Ind.
‡ Miller, William T.....St. Paul
Miller, Winston R.....Red Wing
Miller, Z. R.....St. Paul
Millett, D. Keith.....Minneapolis
Mills, Stephen D.....Rochester
Milnar, Frank J.....St. Paul
Milton, John S.....Minneapolis
Minge, Raymond K.....Worthington
Minsky, Armen A.....Minneapolis
Mintz, Charles M.....St. Paul
Mishek, Charles J.....St. Paul
Mitby, Irving L.....Minneapolis
Mitchell, Berton D.....Minneapolis
Mitchell, Edwards C.....Minneapolis
Mitchell, Mancel T.....Minneapolis
‡ Mixer, Harry W.....Ft. Hood, Texas
Moberg, Clarence W.....Detroit Lakes
Moberg, Thomas D.....Minneapolis
Moe, John H.....Minneapolis
Moe, Thomas.....Moose Lake
Moehn, John T.....Minneapolis
Moehring, Henry G.....Duluth
Moen, J. K., Jr.....Minneapolis
Moersch, Frederick P.....Rochester
Moersch, Herman J .....Rochester
Moga, John A.....St. Paul
Molander, Herbert A.....St. Paul
Molenaar, Robert E.....Cannon Falls
Mollers, Theodore P.....Soudan
† Monahan, Elizabeth S.....Minneapolis
Monahan, Robert H.....St. Paul
Monroe, Paul B.....Cloquet
Monserud, Nels O.....Cloquet
Monson, Einer M.....Minneapolis
Monson, Leonard J.....Canby
Montgomery, Hamilton.....Rochester
Mooney, Robert P.....St. Paul
Moore, Chris H.....Rochester
Moore, Irvin H.....Minneapolis
Moores, Kenneth D.....Rochester
Moorhead, Marie.....Minneapolis
Moos, Daniel J.....Minneapolis
† Moquin, Marie A.....St. Paul
† Morehead, Dewey E.....Owatonna
† Moren, J. Adelaide.....White Bear Lake
Morgan, Hugh O.....Amboy
Moriarty, Berenice.....St. Paul
Moriarty, Cecile R.....St. Paul
Mork, Arthur H.....Anoka
Mork, Frank E.....Anoka
Morlock, Carl G.....Rochester
Morrison, Charlotte J.....Minneapolis
Morse, Morton P.....LeRoy
Morsman, L. Wm.....Hibbing
Mortensen, J. D.....Rochester
Mortensen, Nels G.....Minneapolis
Mortenson, Howard O.....Menahga
Mosby, Maurice E.....Long Prairie
Moses, Royal R.....Kenyon
Mosser, Donn G.....Minneapolis
‡ Moulton, K. B.....St. James
Mouritsen, Glenn J.....Fergus Falls
Moyer, John B.....Duluth
Moyer, Leonard B.....Minneapolis
Mueller, Donald R.....Bagley
Muesing, William J.....New Ulm
Muir, Walter F.....Browns Valley
Mulder, Donald W.....Rochester
Mulholland, William M.....Minneapolis
Muller, Albrecht E.....No. St. Paul
Mulligan, Arthur M.....Brainerd
Mundahl, Harold R.....St. Paul
Munson, Martin S.....Barnum
Murn, Thomas G.....Theif River Falls
Murphy, Edmund P.....Minneapolis
Murphy, Jack T.....St. Paul
Murphy, Joseph E.....Marshall

Murray, Elisabeth..........Minneapolis
Murray, Robert A..........Hibbing
Musachio, Nicholas F..........Foley
Muske, Marvin..........Minneapolis
Mussey, Mary E..........Rochester
Mussey, Robert D..........Rochester
Musty, Nicholas J..........Minneapolis
Myers, Cortland..........Rochester
Myers, Jay A..........Minneapolis
Myers, Richard L..........Rochester
Myers, Thomas T..........Rochester
Myhre, James A..........Minneapolis
Myre, Clifford R..........Paynesville
Myre, Theodore T..........Rochester

Naegeli, Frank..........Fergus Falls
Nagel, Harold D..........Minneapolis
Nakamura, James Y..........Deer River
Nash, Eldore B..........Eden Valley
Nash, Leo A..........St. Paul
Naslund, Ames W..........Minneapolis
Nauth, Bernard S..........Minneapolis
Navratil, Donald R..........Baltimore, Md.
Neal, Joe M..........St. Paul
Nealy, Donald E..........Adrian
Neary, Richard P..........Minneapolis
Neel, Harry B..........Albert Lea
Neff, Walter S..........Virginia
Nehring, Jesse P..........Preston
Neibergs, Lidija..........St. Paul
Neibergs, Pauls..........St. Paul
Neils, Vernon E..........Sauk Rapids
Nelson, Bernette G..........Minneapolis
Nelson, Bernice A..........Minneapolis
Nelson, Carleton A..........Worthington
Nelson, C. Barton..........Minneapolis
Nelson, Clayton E. J..........Albert Lea
Nelson, Edward N..........Minneapolis
Nelson, Ernest J..........Lonsdale
Nelson, Glenn E..........Fairfax
Nelson, Harvey..........Minneapolis
Nelson, Henry E..........Crookston
Nelson, Kenneth L..........Warroad
Nelson, Lloyd S..........Minneapolis
Nelson, Louis A..........St. Paul
Nelson, Louis A., Jr..........St. Paul
Nelson, Maxine O..........Minneapolis
Nelson, Maynard C..........Minneapolis
Nelson, Melvin S..........Granite Falls
Nelson, Nesmith P..........Minneapolis
Nelson, O. L. Norman..........Minneapolis
Nelson, Robert H..........International Falls
Nelson, Robert L..........Duluth
Nelson, Roy A..........Fergus Falls
Nelson, Wallace I..........Minneapolis
Nelson, Wilburn O. B..........Fergus Falls
Nelson, William C..........Rochester
Nerenberg, Samuel T..........Minneapolis
Nesbitt, Samuel..........Minneapolis
Nesheim, Martin O..........Emmons
Nessa, Curtis B..........St. Cloud
Nesse, J. A..........Austin
Nesset, Lawren B..........Minneapolis
Nesset, William D..........Minneapolis
Neumaier, Arthur..........Glencoe
Neuman, Harold W..........Rochester
Neumann, Conrad A..........Lewiston
Neumeister, Charles A..........Minneapolis
Nice, Charles M..........Minneapolis
Nichols, Donald R..........Rochester
Nicholson, Murdoch A..........Duluth
Nicholson, Richard W..........Heron Lake
Nickerson, John R..........Fairmont
Nickerson, Neil D..........Fairmont
Nielsen, Alvin M..........Northfield
Nietfeld, Aloys B..........Sauk Centre
Nilson, Helmer J..........No. Mankato
Nimlos, Kenneth O..........St. Paul
Nimlos, Lenore O..........St. Paul
Ninneman, Newton N..........Waconia
Nisius George..........Duluth
Nixon, James B..........Crosby
Noble, John F..........St. Paul
Noble, John L..........St. Paul
Nollet, Donald J..........St. Paul
Noonan, William J..........Minneapolis
Noran, Axel S. N..........Minneapolis
Noran, Harold H..........Minneapolis
Norberg, Carl E..........Cloquet
Nord, Robert E..........Edina
Nordin, Gustaf T..........Minneapolis
Nordland, Martin, Sr..........Minneapolis
Nordland, Martin, Jr..........Minneapolis
Nordman, Willard F..........Mora
Norman, John F..........Crookston
Normann, Stephen T., Jr..........Waseca
Norris, Neil T..........Caledonia
Noth, Henry W..........Minneapolis
Novak, Edward E..........New Prague
Nudell, Gerald..........Minneapolis
Nuebel, Charles J..........St. Paul
Nuessle, Walter G..........Springfield
Nuetzman, Arthur W..........Faribault
Nutting, Roland E..........Duluth

Nydahl, Malvin J..........Minneapolis
Nye, Katherine A..........St. Paul
Nye, Lillian L..........St. Paul
Nygren, William T..........Braham
Nylander, Emil G..........Minneapolis
Nywall, Dean D..........Slayton

O'Brien, J. C..........St. Paul
O'Brien, Louis T..........Breckenridge
† O'Connor, Daniel C..........Eden Valley
† O'Connor, Loren J..........St. Paul
* O'Donnell, D. M..........Ortonville
O'Donnell, James E..........Minneapolis
O'Hanlon, John A..........Minneapolis
O'Kane, Thomas W..........St. Paul
O'Keefe, James P..........St. Cloud
‡ O'Keefe, Mathew..........Rochester
† O'Leary, John B..........Minneapolis
O'Leary, Paul A..........Rochester
O'Neil, Richard L..........Rochester
O'Neill, John C..........Duluth
O'Phelan, E. Harvey..........Minneapolis
O'Reilley, Bernard E..........St. Paul
‡ O'Shaughnessy, E. J..........Cp. Kilmer, N. J.
† Oberg, Carl M..........Minneapolis
Ochsner, Clarence G..........Wabasha
Ockuly, Orville..........St. Paul
Odel, Howard M..........Rochester
Odland, Donald M..........Luverne
Odland, Mark E..........Detroit Lakes
Odland, Olin..........Granite Falls
Oeljen, Siegfried C. G..........Waseca
Ogden, Warner..........St. Paul
Ohage, Justus..........St. Paul
Olavs, Olga..........Minneapolis
Olds, George H..........New Richland
Olive, John T., Jr..........Mankato
Oliver, Irwin L..........Graceville
Oliver, James..........Moorhead
Olmanson, Edmund G..........St. Peter
Olsen, Arthur M..........Rochester
Olsen, Edward George..........Minneapolis
Olsen, Ralph L..........St. Paul
Olson, Albert E..........Duluth
Olson, Albert J..........Owatonna
Olson, Alton C..........Minneapolis
Olson, Archibald O..........Duluth
Olson, Carl J..........Minneapolis
Olson, C. Kent..........Minneapolis
Olson, Charles A..........St. Paul
Olson, Chester J..........Belle Plaine
Olson, Detlof M..........Minneapolis
Olson, Duane O. C..........Gaylord
Olson, Ernest A..........Pine Island
Olson, Grant E..........West Concord
Olson, Gregory M..........Litchfield
Olson, Lillian A..........Ah-gwah-ching
† Olson, Olof A..........Minneapolis
Olson, Rolland A..........Wayzata
† Onifer, Theodore Michael..........Rochester
Onsgard, L. Kenneth..........Houston
Opfell, Richard W..........Rochester
Oppegaard, C. L..........Crookston
Oppen, E. Gerhard..........Minneapolis
Oppen, Melvin G..........Wayzata
Opshal, Lawrence J..........Willmar
Opstad, Earl T..........Oak Terrace
Orr, Burton A..........Faribault
‡ Orwoll, Harold S..........Japan
Osborn, Donald O..........Austin
Osborn, John E..........Miamisburg, Ohio
Ostergaard, Erling..........Evansville
Ostergren, Edward W..........St. Paul
Ostling, Burton C..........Kerkhoven
Otto, Henry C..........Frazee
Ouellette, Alfred J..........St. Paul
Ourada, Anthony L..........Ceylon
Owen, Charles A., Jr..........Rochester
Owens, Ben P..........Hibbing
Owens, Frederick M., Jr..........St. Paul
Owens, William A..........Montevideo

Page, Raymond L..........St. Charles
Palen, Benjamin J..........Minneapolis
Palmer, Clinton F..........Albert Lea
Palmer, Harry A..........Blackduck
Palmerton, Ernest S..........Fergus Falls
Pankratz, Peter J..........Mountain Lake
Papermaster, Ralph..........Two Harbors
Papermaster, Theodore C..........Minneapolis
‡ Paris, Jaime..........Rochester
Park, Wilford E..........Minneapolis
Parker, Charles W..........Wadena
Parker, Harry L..........Rochester
*† Parker, Owen W..........Minneapolis
Parker, Philip J..........Hallock
Parker, Robert L..........Rochester
Parker, Warren E..........Sebeka
Parker, Wilbert H..........Chisholm
Parkhill, Edith M..........Rochester
Parkin, Thomas W..........Rochester
Parson, E. Irvine..........Duluth
Parson, E. Lillian B..........Elbow Lake
Parson, Lester R..........Elbow Lake

Parsons, R. A..........St. James
Parsons, Ralph L..........Monterey
‡ Parsons, W. B., Jr..........Coronado, Calif.
Pasek, Antone W..........Cloquet
Pasek, Edward A..........Minneapolis
Patch, Orien B..........Duluth
Patrick, Robert T..........Rochester
Pattee, James J..........Minneapolis
Patterson, Hugh D..........Slayton
† Patterson, William L..........Fergus Falls
Paulson, Elmer C..........Minneapolis
Paulson, John A..........Rochester
† Paulson, Theodore S..........Fergus Falls
Paulson, Wallace J..........Minneapolis
Paynter, Camen R..........Great Falls, Mont.
Pearce, Francis M., Jr..........Rochester
† Pearsall, R. P..........Virginia
Pearson, Bror F..........Shakopee
Pearson, Fritz R..........St. Paul
Pearson, Malcolm M..........St. Paul
† Pearson, Roy T..........Shakopee
Pease, Gertrude L..........Rochester
Pedersen, Arthur H..........St. Paul
Pedersen, Robert L..........Brainerd
Pedersen, Roy C..........Duluth
Peltier, Leonard F..........Minneapolis
Pemberton, Albert H..........Rochester
† Pemberton, John..........Rochester
† Penhall, Fletcher W..........Willmar
Penk, Engward L..........Springfield
Penn, George E..........Mankato
† Pennie, Daniel F. V..........Duluth
Peppard, Thomas A..........Minneapolis
Perlman, Everett C..........Minneapolis
Perlman, Herschel L..........Minneapolis
Perry, Harold..........Rochester
Person, John P..........Albert Lea
Pertl, Albert L..........Canby
Peteler, Jennings C. L..........Minneapolis
Peters, Gustavus A..........Rochester
Petersen, Arthur B..........Rochester
Petersen, D. H..........Northfield
Petersen, Deane A..........Minneapolis
Petersen, Glenn L..........Minneapolis
*† Petersen, John R..........Minneapolis
Petersen, Magnus C..........Rochester
Petersen, Peter C..........Minneapolis
Petersen, Robert T..........St. Cloud
Petersen, William E..........Minneapolis
Peterson, Alice H..........Minneapolis
Peterson, David B..........St. Paul
Peterson, Donald H..........St. Paul
Peterson, Edward N..........Virginia
Peterson, Harold O..........St. Paul
Peterson, Henry..........Minneapolis
Peterson, Herbert W..........Minneapolis
Peterson, Joel L. E..........St. Paul
Peterson, John H..........Duluth
Peterson, Kenneth A..........Marshall
Peterson, Kenneth H..........Hutchinson
Peterson, Nordahl P..........Minneapolis
Peterson, Oliver H., Sr..........Minneapolis
† Peterson, Oliver H., Jr..........Minneapolis
Peterson, Palmer A..........Minneapolis
Peterson, Peter E..........Minneapolis
Peterson, Roy A..........Vesta
Peterson, Roy L..........White Bear Lake
Peterson, Stanley C..........Austin
Peterson, Willard C..........Minneapolis
Peterson, Willard E..........Willmar
Peterson, Willard H..........Spring Valley
Petit, Julien V..........Minneapolis
Petit, Leon J..........Minneapolis
Petraborg, Harvey T..........Aitkin
Pettersen, George R..........Mabel
Pettet, John R..........Rochester
Pewters, John T..........Minneapolis
Peyton, William T..........Minneapolis
Phares, Otto C..........St. Cloud
Phelps, Kenneth A..........Minneapolis
Philp, David R..........Watertown
Pierce, Charles H..........Wadena
Pierce, Jack R..........Virginia
Pierce, Robert B..........Olivia
Pierson, Roy F..........Slayton
† Piper, Monte C..........LaCanada, Calif.
Piper, William A..........Mountain Lake
Place, Virgil A..........Rochester
Plasha, Matthew K..........Anoka
Plass, Herbert F. R..........Minneapolis
† Platou, Erling S..........Minneapolis
Pleissner, Karl W..........Minneapolis
Plimpton, Nathan C., Jr..........Minneapolis
† Plondke, Fred J..........St. Paul
Plotke, Harry L..........St. Paul
Plucker, Milton W..........Worthington
Pogue, Richard E..........Watertown
Pohl, John F. M..........Minneapolis
† Poirier, Joseph A..........Forest Lake
Polley, Howard F..........Rochester
† Pollock, David K..........Minneapolis
Polski, Paul G..........So. St. Paul
Polzak, Jacob A..........Minneapolis
Pone, John..........Cambridge

Ponterio, James E..........Shakopee
Pool, Thomas L..........Rochester
† Poppe, Frederick H..........Minneapolis
Porter, Oliver M..........Atwater
Potek, David..........Minneapolis
Potter, Robert B..........Hopkins
Pougiales, Mary L..........Rochester
‡ Powelson, Myron H..........Rochester
Power, John E., Sr..........Duluth
Power, John E., Jr..........Duluth
* Prangen, Avery DeH..........Rochester
† Pratt, Fred J., Sr..........Minneapolis
Pratt, Fred J., Jr..........Minneapolis
‡ Pratt, George F.....San Francisco, Calif.
Pratt, Joseph H., Jr..........Rochester
† Preisinger, Joseph W..........Dallas, Texas
Preston, Paul J..........Minneapolis
Price, William E..........Minneapolis
Prickman, Louis E..........Rochester
Priest, Robert E..........Minneapolis
Priestley, James T..........Rochester
† Prim, Joseph A..........Minneapolis
Prins, Leo R..........Albert Lea
Proeschel, Ray K..........Willmar
Proffitt, William E..........Minneapolis
Proshek, Charles E..........Minneapolis
Pruitt, Raymond D..........Rochester
Pugh, David G..........Rochester
Pumala, Erven E..........Warren
Purnell, Don C..........Rochester
Purves, George H..........Hendricks
Puumala, Reino H..........Cloquet

Quanstrom, Virgil E..........Brainerd
Quattlebaum, Frank..........St. Paul
Quello, Robert O. B..........Minneapolis
Quiggle, Arthur B..........Minneapolis
† Quist, Henry W., Sr..........Minneapolis
Quist, Henry W., Jr..........Minneapolis

Raadquist, Charles S..........Hibbing
Raattama, John..........Keewatin
Racer, Harley J..........Stillwater
Raetz, Sylvester J..........Maple Lake
Rajala, Arnold I..........Grand Rapids
Ralph, James R..........St. Paul
Ralston, Donald E..........Rochester
† Ramsey, Walter R..........St. Paul
Randall, Lawrence M..........Rochester
Randall, Raymond V..........Rochester
Ransom, H. Robert..........Osseo
† Ransom, Matthias L..........Hancock
Rasmussen, Ramby C..........St. Paul
Rasmussen, Waldemar C..........Rochester
Ravits, Harold G..........St. Paul
† Rawls, Thompson T..........Rochester
Rayner, Ralph R..........Glencoe
Rea, Charles E..........St. Paul
Reader, Donald R..........Minneapolis
Reed, Paul..........Virginia
Reff, Alan R..........Crookston
Regnier, Edward A..........Minneapolis
Reid, James W..........So. St. Paul
Reif, Harold A..........Minneapolis
Reif, Henry J..........St. Cloud
Reif, Robert W..........White Bear Lake
† Reifsnyder, Wm. Henry, III..Rochester
Reiley, Richard E..........Minneapolis
† Reineke, George F..........New Ulm
Reitemeier, Richard J..........Rochester
Reitmann, John H..........Anoka
Re Mine, William H., Jr..........Rochester
Remole, William D..........Minneapolis
Remsberg, R. R..........Tracy
† Replogle, William H...Los Angeles, Cal.
Resch, Joseph A..........Minneapolis
Rice, Carl O..........Minneapolis
‡ Rice, Frank B..........Minneapolis
Rice, Fred A..........Minneapolis
Rice, Hagbart G..........Moorhead
Rice, Roberta G..........Grand Island, Neb
Richards, Albert M..........St. Paul
Richards, Ernest T. F..........St. Paul
Richards, William B..........St. Cloud
Richardson, Edward J., Jr..........St. Paul
Richardson, Robert J..........St. Paul
Richdorf, Lawrence F..........Minneapolis
Richter, David J..........Virginia
Rick, Paul F. W..........St. Paul
Rieke, Wellington W..........Wayzata
Rieschl, Elizabeth K..........Jordan
Rigler, Leo G..........Minneapolis
Rigler, Robert G..........Rochester
‡ Ringer, Merritt G., Jr..........Rochester
Ringle, Otto F..........Walker
Rinkey, Eugene..........St. Paul
Riordan, Elsie M..........Minneapolis
‡ Ripepi, James D..........New York, N. Y.
Risch, Ronald E..........Minneapolis
Risser, Alden F..........Stewartville
Ritchie, Wallace P..........St. Paul
Ritt, Albert E..........St. Paul
† Ritzinger, Frederick R..........Lakefield

Rizer, Dean K..........Minneapolis
† Rizer, Robert I..........Minneapolis
Roach, Donald E..........St. Paul
Robb, Edwin F..........Minneapolis
† Robbins, Charles P..........Winona
Robbins, Owen F..........Minneapolis
† Roberts, Byron H..........Minneapolis
Roberts, Lewis J..........Minneapolis
Roberts, Oliver W..........Owatonna
Roberts, Stanley E..........Faribault
Roberts, Stanley W..........Minneapolis
† Roberts, William B..........Minneapolis
Robertson, Paul A..........Austin
Robilliard, Charles M..........Faribault
Robinett, Robert W..........Worthington
Robinson, Cortland O..........Crystal
Rocknem, Robert E..........Minneapolis
Rockwell, C. V..........Minneapolis
Rockwood, Philo H..........Fergus Falls
† Rodda, Frederick C..........Minenapolis
† Rodenbaugh, Fredrich Hase....Rochester
Rodgers, Richard S..........Minneapolis
Roehlke, Arthur B..........Elk River
Roemer, Henry J..........Winona
Rogers, Charles W..........Winona
Rogers, Sidney F..........St. Paul
† Rogin, Norton..........St. Louis Park
Rogne, William G..........Spring Grove
Roholt, Christian L..........McIntosh
Rohrer, Christian..........Worthington
Rokala, Henry E..........Virginia
Rolig, David H..........St. Paul
Rollie, O. O..........St. Paul
Rollins, Pat..........St. Charles
‡ Rollins, Troy G..........Cass Lake
Rome, Howard P..........Rochester
Romness, Kenneth B..........Mound
*†Rood, Dana C.....Santa Barbara, Calif.
Rooke, E. D..........Rochester
Rorem, Joseph A..........Appleton
† Rosander, Phyllis..........Minneapolis
Rose, John T..........Lakefield
Rosenbaum, David L..........Minneapolis
Rosendahl, Frederick G..........Minneapolis
Rosenfield, Abraham B..........Minneapolis
Rosenow, John H..........Minneapolis
Rosenthal, F. Harold..........Austin
Rosenthal, Robert..........St. Paul
† Rosenwald, Reuben M..........Anoka
Ross, Alexander J..........Minneapolis
Rossberg, Raymond A..........Morris
Rossen, Ralph X..........Minneapolis
Rotenberg, Robert J..........Minneapolis
Roth, Frederick D..........Mankato
Roth, George C..........St. Paul
Rothnem, Morris..........Minneapolis
† Rothschild, Harold J..........St. Paul
Rothwell, Walter S..........Rochester
* Rotnem, Orville M.....Iowa City, Iowa
Roust, Henry A..........Montevideo
Rovelstad, Randolph A..........Rochester
Rovelstad, Roger..........Fairmont
Rowe, Clarence J., Jr..........St. Paul
Rowe, Olin W..........Duluth
*†Rowe, William H..........Fairmont
Rowles, Everett K..........Coleraine
Roy, Phil C..........St. Paul
Rozycki, Anthony..........Pine River
Ruchie, Warren H..........Willmar
Rucker, Charles W..........Rochester
Rucker, William H..........Minneapolis
Rud, Norman E..........Minneapolis
† Rudell, Gustave L..........Minneapolis
Rudie, Clifford N..........St. Peter
Rudie, Peter S..........Duluth
Rudie, William D..........Duluth
Rudolph, Frank A..........Sandstone
Ruggieri, Bartholomew..........Rochester
Ruggles, George M..........Forest Lake
† Ruhberg, George M..........Tarzona, Calif.
Rumpf, Carl W..........Faribault
Runquist, John M..........Duluth
Rushton, Joseph G..........Rochester
Russ, Homer H..........Blue Earth
Russeth, Arthur N..........Minneapolis
Rusten, Elmer M..........Minneapolis
Rusterholz, Alan P..........St. Paul
Rutledge, Lloyd H..........Detroit Lakes
Ryan, John J..........St. Paul
Ryan, Joseph M..........St. Paul
Ryan, Robert F..........Rochester
Ryan, William J..........Duluth
Rydburg, Wayne C..........Minneapolis
Ryding, Vincent..........Albert Lea
‡ Rydland, Arne D..........Foley, Ala.
Rygh, Harold N..........Atwater
Rynda, Edwin R..........New Prague
Rynearson, Edward H..........Rochester
Rysgaard, George M..........Northfield

St. Cyr, Harry M., Jr..........Robbinsdale
St. Cyr, Kenneth J..........Robbinsdale
Sabanas, Alvina O..........Rochester
† Sabin, Frederick Chapman......Rochester
Sach-Rowitz, Alvan..........Moose Lake
Sadler, William P., Jr..........Minneapolis
Saffert, Cornelius A..........New Ulm
Safirescu, Sorim R..........Minneapolis
Sahr, Walter G..........Hutchinson
Salassa, Robert M..........Rochester
Saliterman, Bernard I..........Minneapolis
Salk, Richard J..........Albany
Salter, Reginald A..........Virginia
Samuelson, Leonard G..........Mankato
Samuelson, Samuel..........Minneapolis
Sandeen, Robert M..........Buffalo
Sanderson, Anton G..........Anoka
Sanderson, David J..........Fergus Falls
Sandt, Karl E..........Minneapolis
Sandven, Nels O..........Paynesville
† Sanford, Arthur H..........Rochester
*†Sanford, James A..........Lakeville
† Sanford, John B..........Chicago, Ill.
Sanford, Raymond A..........Mankato
Sarff, Oliver E..........Duluth
Sargent, Edward C..........Austin
Saranecki, Merchislan M..........St. Paul
† Satersmoen, Theodore......Pelican Rapids
Sather, Edgar L..........Fosston
Sather, Edgar R..........Alexandria
Sather, George A..........Fosston
Sather, Richard N..........Fosston
Sather, Russell O..........Crookston
Satterlee, Howard W..........Lewiston
† Satterlund, Victor L..........St. Paul
Sauer, William G..........Rochester
† Savage, Francis J..........St. Paul
Sawaryniuk, Iwan..........Waconia
Sawatzky, William A..........Minneapolis
Sawtell, Robert R..........Worthington
Sax, Milton H..........Duluth
Sax, Simon G..........Duluth
Saxman, Gertrude O..........Georgetown
Sayre, George P..........Rochester
Sborov, Abe M..........Minneapolis
Schaaf, Frederick H. K..........Minneapolis
Schaar, Frances E..........Minneapolis
Schade, Frederick L..........Worthington
Schaefer, Joseph F..........Owatonna
Schaefer, Kenneth F..........Minneapolis
Schaefer, Wesley G..........Minneapolis
Schamber, Walter F..........Parkers Prairie
† Schatz, Francis J..........St. Cloud
Scheidel, Alois McK..........Rochester
Scheifley, Charles H..........Rochester
† Scheldrup, N. H..........Minneapolis
Scherer, Leslie Raymond......Minneapolis
Scherling, Sidney S..........Minneapolis
Schiele, Burtrum C..........Minneapolis
Schimelpfenig, George..........Chaska
Schirber, Martin J..........Grand Rapids
† Schirger, Alexander..........Rochester
Schissel, Gregory A..........Minneapolis
Schmid, John F..........Duluth
Schmidt, Herbert W..........Rochester
Schmidt, Hilmar R..........Winona
† Schmidt, Paul A..........Aurora, Ill.
Schmidt, Paul G..........Granite Falls
Schmidt, Ruben F..........Alden
Schmidt, W. Robert..........Minneapolis
Schmidtke, Reinhardt L..........St. Paul
† Schmitt, S. C..........Los Angeles, Calif.
Schmitz, Anthony A..........Mankato
Schmitz, Everett J..........St. Cloud
Schmitz, Glenn P..........Little Falls
Schneck, Jack I..........Waukesha, Wis.
Schneider, Lawrence E..........Duluth
Schneider, Paul J..........Adams
Schnell, Frederick S..........Litchfield
† Schoch, Robert B. J..........St. Paul
Schoeneberger, P. B..........Perham
† Scholpp, Otto W..........Hutchinson
Schons, Edward..........St. Paul
Schottler, Max E..........Minneapolis
† Schroder, Charles H..........Pottstown, Pa.
Schroeckenstein, Hugo F..........St. Paul
Schroeder, Albert J..........Minneapolis
Schroeppel, John E..........Winthrop
Schulberg, Verne A..........Gaylord
† Schuldt, Frederick C..........St. Paul
Schultz, Alvin L..........Minneapolis
† Schultz, J. Albert..........Albert Lea
Schultz, J. Harold..........Minneapolis
Schultz, Peter J..........Minneapolis
† Schulze, Albert G..........St. Paul
Schulze, William M..........Minneapolis
Schumacker, John W..........Robbinsdale
Schutz, Elmer S..........Mountain Lake
Schwartz, Carl A..........Rochester
Schwartz, E. Robert..........Minneapolis
Schwartz, Virgil J..........Minneapolis
Schwarz, Bert E..........Rochester
Schweiger, Theodore R..........Hibbing
Schweinfurth, J. D..........Rochester
‡ Schwyzer, Arnold G..........
San Juan, Puerto Rico
Schwyzer, Hanns C..........St. Paul

Schwyzer, Marguerite	St. Paul
Sckossow, George W.	Erskine
Scott, Eugene E.	St. Paul
Scott, Horace G.	Minneapolis
Scudamore, Harold K.	Rochester
Seaberg, John A.	Minneapolis
Seashore, R. T.	Duluth
‡ Seay, James Elbert, III	Rochester
Seery, Thomas M.	Austin
Seham, Max	Minneapolis
Seifert, Milton H.	Excelsior
Seifert, Otto J.	New Ulm
Sekhon, Mohan S.	St. Paul
† Selby, John B.	Rochester
Seldon, Thomas H.	Rochester
Seljeskog, S. R.	Minneapolis
Sells, Richard S.	No. St. Paul
Selmo, Joseph D.	Norwood
Semsch, Robert D.	Minneapolis
† Senkler, George E.	St. Paul
† Senn, Edward W.	Owatonna
Setzer, Hobert J.	St. Paul
† Seybold, Herbert M.	Rochester
Shandorf, James F.	Minneapolis
Shannon, William R.	St. Paul
Shaperman, Eva P.	Minneapolis
Shapiro, Sidney K.	Minneapolis
Sharp, David D.	Minneapolis
Shaver, Ward	Fergus Falls
Shaw, Howard A.	Minneapolis
Shea, Andrew W.	Minneapolis
Sheedy, Chester L.	Austin
Sheldon, Warren N.	Minneapolis
† Shellman, John L.	Pacific Palisades, Calif.
Sheppard, Charles G.	Hutchinson
Sher, David A.	Virginia
Sher, Lewis	Minneapolis
Sherman, Alfred G.	Albert Lea
Sherman, Carnot H.	Bayport
Sherman, Charles L.	Luverne
* Sherman, Hubert T.	Cambridge
Sherman, Lloyd F.	Minneapolis
Sherman, Royal V.	Red Wing
† Sherwood, George E.	Kimball
Shick, Richard M.	Rochester
Shields, Jack W.	Rochester
Shillington, Maurice A.	Minneapolis
Shirai, Shohei	Coleraine
Shocket, Everett	Rochester
Short, Jacob	St. Paul
Shragg, Robert I.	Howard Lake
Shronts, John F.	Minneapolis
Sibley, John A.	Rochester
Siegel, Clarence	St. Paul
Siegel, John S.	Virginia
Siegmann, William C.	Minneapolis
Siekert, R. G.	Rochester
Silas, Ralph M.	Minneapolis
‡ Silver, Arthur W.	Rochester
Silver, John D.	Minneapolis
Simison, Carl	Barnesville
Simmonds, Harry N.	Prior Lake
† Simon, Howard B.	Rochester
Simons, Bernard H.	Chaska
Simons, Edwin J.	Edina
† Simons, Jalmar H.	Minneapolis
Simons, Leander T.	St. Paul
Simonson, Donald B.	Minneapolis
Simonton, Kinsey MacL.	Rochester
† Simpson, Ellery D.	Phoenix, Ariz.
Sinamark, Andrew	Hibbing
Singer, Benjamin J.	St. Paul
Sinykin, Melvin B.	Minneapolis
Siperstein, David M.	Minneapolis
Sisk, Harvey E.	St. Cloud
Sisler, Clifford E.	Grand Rapids
Sisterman, Thomas J.	Minneapolis
† Sivertsen, Andrew	Minneapolis
† Sivertsen, Ivar	Minneapolis
Sjoding, J. Donald	Mankato
Sjostrom, Lawrence E.	St. Peter
Skaife, William F.	Little Falls
Skaug, Harold M.	Chatfield
Skinner, Abbott	St. Paul
† Skinner, Harvey O.	St. Paul
Skjold, Arthur C.	Minneapolis
Skogerboe, R. B.	Karlstad
† Skrdla, Willard B.	Rochester
Slater, Sidney A.	Worthington
Slocumb, Charles H.	Rochester
Smid, Arthur C.	Rochester
Smiley, John T.	Minneapolis
Smisek, Elmer A.	St. Paul
Smisek, Frank M. E.	Minneapolis
Smith, Adam M.	Minneapolis
Smith, Archie M.	Minneapolis
Smith, Baxter A., Jr.	Minneapolis
Smith, Cyril M.	Duluth
Smith, Don V.	Blue Earth
† Smith, Frederick L.	Rochester
Smith, George R.	Hutchinson
Smith, Graham G.	Minneapolis
Smith, Harry J.	Lake Crystal
Smith, Harry L.	Rochester
† Smith, Homer R.	Minneapolis
* Smith, Leon G.	Montevideo
Smith, Lloyd A.	Willmar
Smith, Lucian A.	Rochester
Smith, Meredith P.	Rochester
† Smith, Margaret I.	Gardena, Calif
† Smith, Myron W.	Red Wing
Smith, Norvin R.	Willmar
Smith, Paul M.	Lake Crystal
Smith, Reginald A.	Rochester
† Smith, Ross H., Jr.	Rochester
Smith, Theodore S.	Minneapolis
Smith, Vernon D. E.	St. Paul
Smith, Wallace R.	Grand Marais
Smith, William G.	Rochester
Smith, William T.	Minneapolis
Smorstok, Matthew B.	Monticello
Smyth, John J.	Lester Prairie
Snyder, Clifford D.	Kiester
Snyder, George W.	St. Paul
Snyker, Omer E.	Ely
Soderlind, Ragnar T.	Minneapolis
Sogge, Ludwig L.	Windom
Sohlberg, Olof I.	St. Paul
† Sohmer, A. E. J.	Mankato
Solhaug, Samuel B.	Minneapolis
Solhaug, Samuel B., Jr.	Minneapolis
Solsem, Frederick N. S.	Spicer
Solvason, Harold M.	Minneapolis
Sommerdorf, Vernon L.	St. Paul
Sommers, Ben	St. Paul
Sommerness, M. Duane	Fergus Falls
Sonnesyn, Nels N.	Le Sueur
Sorem, Milton B.	St. Paul
Sorum, F. T.	Willmar
Soucheray, Philip H.	St. Paul
Soule, Edward H.	Rochester
Souster, Benjamin B.	St. Paul
Sowada, Ernest J.	St. Paul
Spain, W. Thomas	Princeton, N. J.
Spang, Anthony J.	Duluth
Spang, James S.	Duluth
Spang, William M.	Duluth
Spano, Joseph P.	Minneapolis
Spear, Harold C.	New Haven, Conn.
Spencer, Bernard J.	Minneapolis
Spencer, Jean A.	Rochester
Sperl, Michael P., Jr.	Rochester
Spink, Wesley W.	Minneapolis
Spittel, John A., Jr.	Rochester
Sponsel, Kenath H.	Minneapolis
‡ Sprafka, Gregory A.	N. Y., N. Y.
† Sprafka, Joseph L.	St. Paul
Sprafka, Joseph M.	St. Paul
Sprague, Randall G.	Rochester
† Spratt, Charles N.	Minneapolis
Spurbeck, George H.	Ah-gwah-ching
Spurzem, Raymond J.	Anoka
Stadem, Clifford J.	Twin Valley
Stahler, Paul A.	Jordan
Stahl, George W.	Austin
‡ Stahn, Louis H.	El Paso, Texas
Stahr, Aubrey C.	Hopkins
Stam, John	Worthington
Stanford Charles E.	Minneapolis
Stangl, Philip E.	St. Cloud
Stanley, Court R.	Worthington
Starekow, Milton D.	Thief River Falls
Starr, Grier F.	Rochester
Staub, Henry P.	Minneapolis
Stauffer, Maurice H.	Rochester
Steffens, Leon A.	Red Wing
† Stein, Harold A.	Rochester
Stein, Raymond J.	Pierz
Stein, William A.	Ely
Steinberg, Charles L.	St. Paul
* Steiner, Irving W.	Winona
‡ Steiner, Leon E.	Philadelphia, Pa.
Steinhilber, Richard M.	Rochester
Stelter, Lloyd A.	Minneapolis
Stemsrud, Harold L.	Alexandria
Stennes, John L.	Minneapolis
Stensgaard, Kermit L.	Thief River Falls
Stenstrom, Annette	Minneapolis
Sterner, Donald C.	St. Paul
* Sterner, Ernest G.	St. Paul
Sterner, E. R.	St. Paul
Sterner, John J.	St. Paul
Sterrie, Norman A.	Minneapolis
† Stevens, Grant M.	Rochester
Stevenson, Basil M.	Fulda
Stevenson, Frank W.	Faribault
Stewart, Alexander	St. Paul
† Stewart, Allan H.	Rochester
Stewart, Donald E.	Crookston
Stewart, Marvin J.	Minneapolis
Stewart, Rolla I.	Minneapolis
Stickney, J. M.	Rochester
† Stiegler, Farrell S.	St. Paul
Stilwell, George G.	Rochester
Stillwell, George K.	Rochester
Stillwell, Water C.	Mankato
Stoesser, Albert V.	Minneapolis
Stolpestad, Armer H.	St. Paul
Stolpestad, Herbert L.	St. Paul
Stoltz, Robert C.	Minneapolis
† Stomel, Joseph	Blackfoot, Idaho
Stone, Stanley P.	Minneapolis
Storsteen, Kenneth A.	Duluth
‡ Stoy, Robert A.	Little Falls
† Strachauer, Arthur C.	Minneapolis
Strand, Jack W.	St. Paul
Strandjord, Nels M.	Virginia
Stransky, Theodore W.	Owatonna
Strate, Gordon E.	St. Paul
Strathern, Carleton S.	St. Peter
† Strathern, Fred P.	St. Peter
† Strathern, Moses L.	Gilbert
Stratte, Alf K.	Pine City
Stratte, Harold C.	Windom
Strauchler, Jona	Belview
Straus, M. L.	St. Paul
Strauss, Eugene C.	Duluth
Street, Bernard	Northfield
Strem, Edward L.	St. Paul
Strewler, Gordon J.	Duluth
Strickler, Jacob H.	Minneapolis
Strobel, William G.	Duluth
Stroebel, Chas. F., Jr.	Rochester
Strom, Gordon W.	Minneapolis
Stromgren, Delph T.	Minneapolis
Stromme, William B.	Minneapolis
Strunk, Clarence A.	Minneapolis
Struthers, A. Morgan	Rochester
Struxness, David F.	Glencoe
Studer, Donald J.	Faribault
Stuhler, Louis G.	Rochester
Stuhr, John W.	Stillwater
Sturges, Robert L.	Minneapolis
Sturley, Rodney F.	St. Paul
† Subby, Walter	Minneapolis
Sukov, Marvin	Minneapolis
Sullivan, Raymond M.	Minneapolis
Sullivan, Robert E.	Minneapolis
Sutherland, Harry N.	Ely
Sutherland, W. H.	New London
Sutton, Harris R.	Hoffman
Svien, Hendrik J.	Rochester
Swain, Francis M.	Minneapolis
† Swanson, John A.	St. Paul
Swanson, Lawrence J.	West St. Paul
Swanson, Roy E.	Minneapolis
Swedberg, William A.	Duluth
Swedenburg, Paul A.	Glenwood
Sweetser, Horatio B.	Minneapolis
Sweetser, Theo. H., Sr.	Minneapolis
Sweetser, Theo. H., Jr.	Minneapolis
† Sweitzer, Samuel E.	Minneapolis
† Swendseen, Carl G.	Graceville
Swendson, James J.	St. Paul
Swenson, Arnold O.	Duluth
† Swenson, Donald B.	St. Paul
† Swenson, Donald B.	Minneapolis
Swenson, Orvie J.	Waseca
Swenson, Roy G.	North Branch
Symmonds, Richard E.	Rochester
Syverton, Jerome	Minneapolis
Tam, Ernest C.	Minneapolis
Tangen, George M.	Minneapolis
Tani, George T.	Rochester
Tanquist, Edwin J.	Alexandria
Taub, Robert G.	Rochester
Taylor, Joseph H.	Minneapolis
Teich, Kenneth W.	Duluth
Teisberg, John E.	St. Paul
Tenner, Robert J.	Minneapolis
Terrell, Bernard J.	Nopeming
Tesch, Gordon H.	Elk River
Tetlie, James P.	Duluth
Thabes, J. A., Jr.	Brainerd
Thayer, Ellsworth A.	Fairmont
Thielen, Robert D.	St. Michael
Thiem, Chester E.	Mankato
Thill, Leonard J.	Balaton
† Thomas, George E.	Minneapolis
Thomas, John V.	Duluth
Thomas, William H.	Howard Lake
Thomes, A. Boyd	Minneapolis
Thompson, Arthur	Minneapolis
Thompson, A. Henry	Litchfield
Thompson, Carl O.	Hendricks
Thompson, Floyd A.	St. Paul
Thompson, Gershom J.	Rochester
Thompson, Willis H.	Minneapolis
Thomson, James M.	Austin
Thoreson, M. C. Bernice	So. St. Paul
Thorsen, David S.	Minneapolis
Thorson, Stuart V.	Minneapolis
Thuringer, Carl B.	St. Cloud
Thysell, Desmond M.	Minneapolis
† Thysell, Fred A.	Moorhead
Thysell, Harold R.	Crookston
Thysell, Vernon D.	Hawley
Tichy, Fae Y.	Minneapolis

	Tifft, Cyril R.	St. Paul
	Tihen, Edward N.	Rochester
	Tillisch, Jan H.	Rochester
†	Tingdale, August C.	Minneapolis
	Tingdale, Carlyle	Hibbing
	Tinkham, Robert G.	Minneapolis
	Titrud, Leonard A.	Minneapolis
	Tobin, John D.	Minneapolis
	Tomhave, Wesley G.	Hibbing
	Tongen, Lyle A.	St. Paul
	Tosseland, Noel E.	Duluth
	Trach, Benedict B.	Minneapolis
	Tracht, Robert R.	St. Paul
	Traeger, Carl A.	Faribault
	Travis, James S.	St. Paul
	Traxler, J. F.	Henderson
	Tregilgas, Harold R.	So. St. Paul
	Tregilgas, Richard B.	St. Paul
	Troost, Henry B.	Mankato
	Trow, James E.	Minneapolis
	Trow, William H.	Minneapolis
	Trueman, Harold S.	Minneapolis
	Truesdale, Clark W.	Glencoe
	Tsai, Shih Hao	Oak Terrace
	Tucker, Jolyn S.	Alabama
	Tucker, Richard C.	Minneapolis
	Tudor, Richard B.	Minneapolis
	Tuohy, Edward L.	Duluth
†	Turnacliff, Dale D.	St. Paul
	Tweedy, John A.	Winona
	Tweedy, Robert B.	Winona
	Twiggs, Leo F.	Austin
	Twomey, John E.	Minneapolis

	Ubel, Frank A.	St. Paul
	Ude, Walter H.	Minneapolis
	Uhley, Charles G.	Crookston
	Uihlein, Alfred	Rochester
†	Ulrich, Henry L.	Minneapolis
	Ulvestad, Harold S.	Minneapolis
	Underdahl, Laurentius O.	Rochester
	Undine, Clyde A.	Minneapolis
	Urberg, Sofus E.	Duluth
	Utne, John R.	Rochester
	Utz, David C.	Rochester

†	Vadheim, Alfred L.	Tyler
†	Valentine, Walter H.	Tracy
	Van Bergen, Fredk. H.	Minneapolis
	Van Cleve, Horatio P., Jr.	Austin
	Vandersluis, Charles W.	Bemidji
	Van Herik, Martin	Rochester
	Van Meier, Henry	Stillwater
	Van Patter, Ward	Seattle, Wash.
	Van Rooy, George T.	Thief River Falls
	Van Ryzin, Donald J.	Duluth
	Varco, Richard L.	St. Paul
	Vaughan, Victor M.	Truman
	Vaughn, Louis D.	Rochester
	Veirs, Dean M.	St. Paul
	Veirs, Ruby J. S.	St. Paul
	Venables, Alexander E.	St. Paul
	Veranth, Leonard A.	St. Cloud
	Verby, John E., Jr.	Rochester
	Vermund, Halvor	Minneapolis
	Vezina, John C.	Mapleton
	Vik, A. Elliott	Minneapolis
	Vik, Melvin	Anoka
	Virnig, Hildegard J.	Caledonia
	Virnig, Mark P.	Wells
	Virnig, Richard P.	Wells
	Vitols, T. M.	Minneapolis
	Vogel, Howard A. L.	New Ulm
	Vollmer, Frederick J.	Winona
‡	von Amerongen, W. W.	Chandler, Ariz.
‡	Von Drasek, Joseph	Mankato

	Waas, Charles W.	St. Paul
	Wagener, Henry P.	Rochester
	Wagner, Norman W.	Benson
	Wagoner, James M.	Harmony
	Wahlquist, Harold F.	Minneapolis
	Wakefield, Elmer G.	Rochester
†	Wakim, Khalil G.	Rochester
	Walder, Harold J.	Duluth
	Waldron, Carl W.	Hopkins
	Walfred, Karl A.	St. Cloud
†	Walker, A. E.	St. Paul
	Walker, Arthur E.	St. Paul
	Wall, Carl R.	Minneapolis
	Wall, James O.	St. Paul
	Wallace, Martin O.	Duluth
†	Waller, Joseph D.	Pine City
‡	Wallinga, Jack	Oakland, Calif.
	Walonick, Albert L.	St. Louis Park
	Walsh, Edward F.	St. Paul
	Walsh, Francis M.	Minneapolis
	Walsh, William T.	Minneapolis
	Walter, Clarence Wm.	St. Paul
	Walter, Frederick H.	International Falls
	Walter, William E.	Wanamingo
	Walters, Waltman	Rochester
	Wandke, Otto E.	Fairmont
‡	Wang, Jun-Chuan	Honolulu, Hawaii
	Wangensteen, Owen H.	Minneapolis
	Ward, Bert B.	Rochester
	Ward, Louis E.	Rochester
	Ward, Percy A.	Minneapolis
	Warner, James J.	Perham
	Warren, Cecil A.	St. Paul
	Wasmund, Clarence W.	Red Wing
	Wasson, Loren F.	Alexandria
	Watkins, Charles H.	Rochester
	Watkins, John A.	Wells
	Watson, Alexander M.	Royalton
	Watson, C. Gordon	Minneapolis
	Watson, Cecil J.	Minneapolis
	Watson, Eleanor J.	Rochester
	Watson, John R.	Rochester
	Watson, P. Theodore	St. Paul
	Watson, Percy T.	Northfield
	Watson, Robert M.	Morris
	Watson, Sydney Wm.	Royalton
	Watson, William H. A.	St. Paul
	Watson, William J.	Newport
	Watz, Clarence E.	St. Paul
	Waud, Robert E.	Rochester
	Waugh, John M.	Rochester
†	Weaver, Myron Mc.	Vancouver, B. C., Canada
	Weaver, Paul H.	Faribault
	Webb, Edgar A.	Minneapolis
	Webb, Roscoe C.	Minneapolis
	Webber, Fred L.	St. Paul
	Webber, Richard J.	St. Louis Park
	Weber, Harry M.	Rochester
	Weber, Lowell W.	Minneapolis
	Weed, Lyle A.	Rochester
†	Weeks, Richard E.	Rochester
	Wehr, M. B.	Rochester
	Weiner, Alan D.	Rochester
	Weir, James F.	Rochester
	Weis, Benjamin A.	St. Paul
	Weisberg, Maurice	St. Paul
	Weisberg, Raphael J.	Minneapolis
	Weiss, Carl A.	Hastings
	Welch, John S.	Rochester
	Wellman, Thomas G.	Clinton, Iowa
	Wellman, William E.	Rochester
	Wellner, Theodore O.	Rochester
	Wells, Arthur H.	Duluth
	Wells, Walter B.	Jackson
	Wendland, John P.	Minneapolis
	Wendt, H. P.	Thief River Falls
	Wenner, Waldemar T.	St. Cloud
	Wente, Harold A.	Rochester
†	Wentworth, Albert J.	Mankato
	Wenzel, Gilbert P.	St. Paul
†	Werner, George	Minneapolis
‡	Wesolowski, Stanley P.	Minneapolis
†	West, Catherine C.	Minneapolis
	Westby, Magnus	Madison
	Westby, Norval M.	Madison
†	Westerman, Alvin E.	Montgomery
	Westerman, Fred C.	Montgomery
	Westover, D. E.	St. Paul
	Westrup, John E.	Lanesboro
	Wetherby, Macnider	Minneapolis
	Wetzel, Earl V.	St. Paul
‡	Weyhrauch, Robert A.	Alexandria
	Wheeler, Daniel W.	Duluth
*†	Wheeler, Merritt W.	Stillwater
	Wheeler, Robert W.	Minneapolis
	White, Asher A.	Minneapolis
†	White, S. Marx	Minneapolis
	White, Willard D.	Minneapolis
	Whitesell, Lloyd A.	Minneapolis
	Whitson, Sidney A.	Albert Lea
	Whittemore, Dexter D.	Bemidji
	Widen, Wilford F.	Minneapolis
	Wikoff, Howard M.	Crookston
	Wilcox, G. Charles	Albert Lea
†	Wilder, Kenneth W.	Minneapolis
	Wilder, Robert L.	Minneapolis
†	Wilder, Russell M., Sr.	Bethesda, Md.
†	Wilder, Russell M., Jr.	Minneapolis
	Wilken, Paul A.	Minneapolis
‡	Wilkinson, George R., Jr.	Rochester
†	Wilkinson, Stella L.	St. Paul
	Will, Charles B.	Bertha
	Will, W. W.	Bertha
†	Willcutt, Clarence E.	Phoenix, Ariz.
†	Williams, Arthur B.	St. Paul
‡	Williams, Bruce F. P.	Duluth
	Williams, Charles A.	Pipestone
	Williams, Clayton K.	St. Paul
	Williams, George E.	Rochester
	Williams, Henry L., Jr.	Rochester
†	Williams, Hugh O.	Lake Crystal
	Williams, John A.	St. Paul
†	Williams, Leon A.	Minneapolis
	Williams, M. M.	Ah-gwah-ching
	Williams, Marland R.	Cannon Falls
*†	Williams, Rasmus V.	Rushford
	Williams, Richard A.	St. Paul Park
†	Williams, Robert	New York, N. Y.
	Williamson, Harold A.	Fairmont
	Wilmot, Cecil A.	Litchfield
	Wilmot, Harold E.	Litchfield
†	Wilson, Clyde E.	Blue Earth
‡	Wilson, Franklin C.	Ft. Belvoir, Va.
	Wilson, J. Allen	St. Paul
	Wilson, James V.	St. Paul
	Wilson, Louis J.	Winona
	Wilson, Robert B.	Rochester
	Wilson, Rolland H.	Winona
	Wilson, Theodore W.	Rochester
	Wilson, Viktor O.	Rochester
	Wilson, Warren E.	Northfield
†	Wiltrout, Irving G.	Oslo
	Winchell, Paul	Minneapolis
†	Winnick, Joseph B.	St. Paul
	Winter, John A.	Duluth
	Winter, Malcolm D., Jr.	Rochester
	Winther, Nora M. C.	Minneapolis
	Wipperman, Frederic F.	Minneapolis
	Wisness, Osmund A.	Comfrey
†	Witham, Carl A.	Minneapolis
	Witthaus, Melvyn E.	Mahtomedi
	Wittich, Frederick W.	Minneapolis
	Wittrock, Louis H.	Watkins
	Wohlrabe, Arthur A.	Minneapolis
	Wohlrabe, A. Cabot	Minneapolis
	Wohlrabe, Clarence F.	No. Mankato
	Wohlrabe, Edwin J.	Springfield
	Wohlrabe, John C.	St. Clair
	Wolf, Alfred H.	Minneapolis
	Wolff, Herman J.	St. Paul
	Wolff, John M.	Duluth
	Wolkoff, H. J.	St. Paul
	Wollaeger, Eric	Rochester
	Wolstan, Simon D.	Minneota
	Wolter, Frederick H.	Minneapolis
	Woltjen, Myron J.	Rushford
	Woltman, Henry Wm. F.	Rochester
†	Wood, Harry G.	Rochester
‡	Woodington, George F.	Altoona, Wis.
	Woolner, Lewis B.	Rochester
	Word, Harlan L.	St. Paul
	Workman, Warner G.	Tracy
	Woyda, William C.	Elk River
†	Wray, William E.	Campbell
	Wright, Robert R.	Austin
	Wright, Thomas D.	Minneapolis
	Wright, Wale S.	Minneapolis
	Wright, William S.	Minneapolis
‡	Wuest, John H., Jr.	Los Angeles, Cal.
	Wyatt, Oswald S.	Minneapolis
	Wynne, Herbert M. N.	Minneapolis

	Yaeger, Wilbert W.	Marshall
‡	Yamamoto, Joe	Ft. Lewis, Wash.
†	Ylitalo, William H.	Rochester
	Ylvisaker, Ragnvald S.	Minneapolis
	Yoerg, Otto W.	Minneapolis
	Young, Henry H.	Rochester
	Young, Thomas O.	Duluth
	Younger, Lewis I.	Winona
	Youngren, Everett R.	St. Paul
	Yue, Wen Y.	Oak Terrace

	Zachman, Albert H.	Melrose
	Zachman, Leo L.	St. Paul
	Zagaria, James F.	St. Paul
	Zahrendt, O. Lewis	Minneapolis
	Zarling, V. Richard	Minneapolis
	Zaworski, Leo A.	Minneapolis
‡	Zeigler, Charles M.	Pine River
	Zeller, Nicholas H.	Worthington
	Zemke, Erhart E.	Fairmont
	Zemmers, Roberts	Duluth
	Zick, Luther H.	Grand Rapids
	Ziegler, Robert G.	Welcome
	Zierold, Arthur A.	Minneapolis
	Zimmer, J. F.	Rochester
	Zimmermann, Harry B.	St. Paul
	Zinter, Ferdinand A.	Minneapolis
	Ziskin, Thomas	Minneapolis
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Original Contributions

Tetanus

A Summary of Thirty-two Cases with Special Reference to Prevention

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TETANUS, known as a clinical entity since the time of Hippocrates, continues to occur and to command a high fatality rate. Although effective active immunization is well established, the wide dissemination of the hardy spore form of the causative organism in association with its regular presence in the gastrointestinal tracts of most domestic animals makes the disease a difficult one to eradicate. Because more than one case is rarely seen by any one physician, it seems worthwhile to review certain aspects of clinical tetanus with special reference to prevention of mortality.

Cases

During the past ten years (1943-1953), thirty-two cases of tetanus were seen at the University of Minnesota Hospitals (Table I). The ages of the patients ranged from twenty months to eighty-one years. One half of the patients were sixteen years of age or younger; the mortality was 25 per cent in this group. Nine patients were over fifty years old; of these, six (67 per cent) died.

The time intervals from injury to the first symptom, in those cases where the data are available, are listed in Table II. An initial injury was apparent in thirty of the thirty-two cases. The variety of injuries, the causative mechanism, and the sites of injury are summarized in Table III.

The initial symptoms of tetanus, the events that informed the patient that he was sick, are summarized in Table II under the headings of trismus, spasm of dorsal muscles, extremity muscle spasm, and influenza-like onset. Onset with trismus was most common; nineteen (60 per cent) of the patients in the present series are included in this group, and seven of the nineteen later succumbed to the disease. These patients usually experienced difficulty in eating, but this may have been referred to by the patient as "stiffness" of the tongue or dysphagia.

TABLE I. AGES OF PATIENTS

Age	Patients	Deaths	Mortality
Under 6 years	9	2	22%
7-16 years	7	2	29%
17-50 years	7	0	—
Over 50 years	9	6	67%
Totals	32	10	31%

TABLE II. INCUBATION PERIODS

Total Group (29 Cases)	Fatal Cases (9 Cases)
Range: 3-23 days	Range: 4-13 days
Average: 9 days	Average: 8.7 days

Patients with spasm of the dorsal muscles usually noted spasm, pain, or tenderness of the back or cervical muscles which occasionally progressed to opisthotonus. Six patients are herein included; two died. The patients who presented spasm of the extremity muscles noted spasm or "stiffness" of the extremities. Four patients are in this group; one died. Patients with influenza-like onset noted generalized body aching and malaise; sometimes generalized but minimal stiffness or pain in many muscle groups was present. There were three patients with this type of onset; no deaths occurred.

It is of particular interest that several of the injuries that were followed by tetanus were grossly clean wounds or were cleansed in a manner which would seem likely to have removed contamination (Table V). It seems possible that these wounds were free of tetanus organisms at the time of the original injury but were contaminated at some later date, before healing had

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TABLE III. INJURIES FOLLOWED BY TETANUS

Cases	Instrument or Mechanism	Injured Portion of Body	Fatalities
7	Sharp instrument	Hand or finger	1
4	Nail	Foot	3
3	Sharp instrument	Foot	—
2	Wood sliver	Hand	—
2	Burn	Extremities	2
2	Unknown	—	—
1	Nail	Hand	—
1	Nail	Forehead	—
1	Barbed wire	Foot	1
1	Wood sliver	Toe	—
1	Tree branch	Leg	1
1	Bush	Leg	—
1	Broken glass	Buttocks	—
1	Dynamite	Elbow and forehead	—
1	Roadway	Scalp	1
1	Twig	Gingiva	—
1	Compound fracture	Finger	—
1	Autoexcoriation	Leg ulcer	1

taken place. The contamination of an originally clean wound, which may have occurred in these cases, rarely has been stressed as a significant feature in the etiology of tetanus.

Ten patients in this series died. Autopsies were carried out in eight. The significant autopsy findings, and the probable causes of death in those cases where benefit of autopsy was not obtained, are listed in Table VI.

Discussion

While this series of thirty-two cases of tetanus is relatively small and therefore not subject to strict statistical analysis, several features are of clinical interest. The observation has been made elsewhere that tetanus is no longer the scourge of the parturient mother, the newborn babe, and the wounded soldier.[1] The recent cases seen at the University Hospitals occurred chiefly among rural residents. The fatalities were largely in persons whose age or physical condition seems to have made them less able to withstand the respiratory complications which invariably accompany the full-blown case.

Prompt recognition of the disease, once the initial symptoms have appeared, should lead to earlier treatment and likely to better chance of cure. Early diagnosis of tetanus remains clinical in spite of some aid from the laboratory. The earliest symptom in the cases presented here was almost always related to some type of muscle spasm. The majority of the patients had spastic involvement of the jaw muscles as an initial symptom; some expressed this as pain, difficulty with eating, or dysphagia. Several patients first noted spasm of other muscles—especially those of the back and neck. A few had spasm or stiffness of the arms or legs. It is likely that trismus which cannot otherwise be readily explained—especially when associated with spasm of the back muscles—should be presumed due to tetanus.

TABLE IV. FIRST SYMPTOMS OF TETANUS

Group	Cases	Symptom
Trismus	13	Trismus
	3	Dysphagia
	2	"Stiff" or "swollen" tongue
	1	Pain in the jaw
Spasm of the dorsal muscles	2	Stiff neck
	2	Backache
	1	Spasm of back muscles
	1	Opisthotonus
Extremity muscle spasm	2	"Wooden gait"
	2	Spasm or "twitching" of the extremities
"Flu"-like onset	2	Generalized muscle aching
	1	Abdominal pain and body "stiffness"

When considering the natural history of tetanus, it has been generally assumed that those who fall victim to the disease have had the spores introduced into a wound at the time of initial injury. The suggestion has been made only infrequently that some of those who develop clinical tetanus may have had wounds that were at first free of the organism, but by one route or another the spores entered the wound later—before healing was complete.[2] Consideration of the mode of infection in wounded soldiers in World War I has led to the impression that many of the bullet wounds were originally "clean" but that contaminated soil from the fields may have gotten into the wounds before the patients reached medical aid.[3] Striking, however, is the observation by Stitts[4] that natives lacerated by the barb-like tail of the sting ray have developed tetanus—presumably after clean wounds were contaminated later with soil containing tetanus spores.

Although the exact modes of tetanus infection in the five patients presented in Table V remain problematical, they serve to emphasize that open wounds, especially if infection or tissue necrosis is present, may offer ideal ground for the growth of the tetanus organism. Debridement of wounds and eradication of other infecting bacteria continue to be measures considered necessary to destroy conditions which are favorable for the growth of the tetanus organism and the development of the disease.[5] The additional precaution of safeguarding unhealed wounds from secondary contamination with Clostridium tetani is likely important.

TABLE V. PATIENTS WITH POSSIBLE LATER-CONTAMINATION

Case Number	Age in Years	Sex	Probable Portal of Entry of Organism	Treatment of Injured Locale	Conditions Favoring Late Contamination	Injury to Symptoms
1	76	M	Postphlebitic leg ulcer	None	Worked in garden; often excoriated leg and ulcer	10 years
2	4	M	Heel, cut on bicycle spoke	Bled profusely; treated and bandaged by a physician	Up and about house and yard; wound "dirty" on admission	17 days
3	9	M	Toe, cut with a wood saw	Cleansed and sutured by a physician	Dressings often soiled as child was about the farm	15 days
4	56	M	Hand, cut with a buzz saw	Treated by a physician; 1500 units of tetanus antitoxin given	Continued most of farm duties; wound unhealed on admission	13 days
5	61	M	Fingers, cut with a circular saw	Treated by a physician; 1500 units of tetanus antitoxin given	Worked on farm after the injury; infected fingers noted on admission	21 days

TABLE VI. FATAL CASES

Age in Years	Sex	Tracheotomy	Autopsy	Cause of Death
9	M	None	Yes	Pneumonia and "respiratory failure"
7	M	Delayed	No	Pneumonia and partial tracheal obstruction
4	M	Delayed	Yes	Pneumonia
63	F	None	Yes	Aspiration pneumonia, atelectasis, and pulmonary congestion
4	F	Delayed	Yes	Pneumonia, atelectasis, and cerebral edema
77	M	Delayed	Yes	Pneumonia, atelectasis, and pulmonary congestion
65	M	Prompt*	Yes	Ruptured midcolic artery, aspiration pneumonia, and atelectasis
76	M	Prompt	Yes	Pneumonia and atelectasis
57	M	Prompt	No	Pneumonia
81	F	Delayed	Yes	Pneumonia and atelectasis

*Prompt—within four hours of hospital admission.

The introduction of tetanus antitoxin represented an advance in the prevention of clinical tetanus. Prophylactic administration of this serum to wounded soldiers in the first World War probably reduced the incidence of the disease. However, the observation that several patients in this series developed tetanus in spite of the prophylactic administration of tetanus antitoxin confirms the impression of several observers that antitoxin, at least in the common prophylactic doses of 1500 units, cannot be regularly relied upon to prevent the development of clinical tetanus.[6,7] The presence of occasional hypersensitivity to the horse serum of the antitoxin and the frequent observation that many of the patients who develop tetanus in civilian life do not seek medical attention at the time of injury, somewhat detract from the usefulness of the antitoxin in prophylaxis and further emphasize the desirability of active immunization with the toxoid.

It has been stated that active immunization with tetanus toxoid affords protection from the disease.[8] This impression is confirmed by reports in regard to World War II which indicate that of five million Navy personnel immunized with the toxoid, only four contracted the disease during the war, and of the four only two (both without record of complete immunization) died.[9] During a comparable period of time, about 2,500 deaths from tetanus were reported in the civilian population of this country.[10] In the present series of thirty-two cases, none of the patients had received the toxoid. Active immunization with tetanus toxoid should be readily available and routinely administered to those persons who are likely to sustain frequent small injuries, to those exposed to animal excreta, and to all children.[11,12]

A detailed discussion of therapy is not the purpose of this paper, but it may be noted that the problem of prevention of death from the disease is largely one of prevention of serious respiratory complications. The fatalities in the present group, like those in most series recently reported, serve to illustrate the gravity of pulmonary complications; of the ten patients in this group who expired, nine died of atelectasis, pneumonia, asphyxia, or a combination of these. The toxin causes variable degrees of central nervous system paralysis, but some of the damage may be temporary with the aid of tetanus antitoxin.[13] If the patient can be protected from severe respiratory complications his life usually can be saved. Of chief importance in treatment of the established case of tetanus is the maintenance of an adequate upper respiratory airway, but following close behind is the necessity of adequate cleansing of secretions of the tracheobronchial tree. Tracheotomy—with constant nursing care—is admirably suited for both purposes.[14,15,16] Tracheotomy is now considered to be a prerequisite for proper treatment of systemic tetanus when any respiratory difficulties are present. A number of recent papers emphasize the treatment of respiratory complications and review current concepts of therapy of severe cases of tetanus.[17,18,19]

Conclusions

Although the salient features of clinical tetanus have changed little in the 2,000 years since their first description by Hippocrates, prophylaxis and therapy have undergone a remarkable metamorphosis. Many of the features of the present series of thirty-two cases of tetanus seen during the past ten years are consistent with traditional concepts of the disease, but certain new aspects have become worthy of emphasis. The place of the battle casualty and the newborn child as victims of tetanus has been taken by the man in the barnyard and the child on the farm. Trismus remains the most frequent presenting symtom, but variation in its mode of expression occurs in some patients; a few patients apparently are more impressed with spasm of other muscles than those of the jaw.

Several cases illustrate the notable—but not entirely new—observation that a wound may be treated originally in a seemingly adequate manner but that contamination later of the unhealed wound by tetanus spores possibly occurs. It is emphasized that in addition to debridement and cleansing of wounds, protection from contamination before healing has taken place may be of importance. The usual prophylactic dose of 1500 units of tetanus antitoxin was inadequate to prevent development of the disease in several patients. It is suggested that this dose be increased to 5,000 or 10,000 units when there is a likelihood of the presence of tetanus spores.

The thirty-one per cent fatality rate in this series, although lower than several recently reported rates of 40 to 70 per cent,[20,21,22] is nonetheless high. Deaths in this group were preceded by respiratory complications. Current fatality rates, which remain high in spite of recent advances in therapy, serve to underscore the virtue of active immunization with tetanus toxoid. Routine toxoid immunization probably should include both children and adults.

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A Practical Approach to the Treatment of Tetanus

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TETANUS toxoid, used properly, has proved to be one of the most effective prophylactic agents yet developed in medicine. Part of the foundation for this declaration is my own experience acquired during the Okinawan campaign. At that time I was on the medical staff of a United States Military Government Hospital wherein between 100 to 150 nonimmunized civilians were treated for tetanus. The office of the Surgeon General of the United States Army[6] informed me that not a single case of tetanus was reported among the many thousands of members of the Armed Forces of the United States who participated in the Okinawan phase of the war. Wounds were frequent among them, and the conditions under which both they and the civilians lived were similar. Members of the Armed Forces, however, had received prophylactic injections of tetanus toxoid.

Reactions to toxoid have been reported to number only 2 per 100,000.[7] This, together with the high degree of protection and the threat of another world war, should be enough to stimulate physicians of this country to work for immunization of the entire population to tetanus. This would make unnecessary the many injections of tetanus antitoxin in horse serum which the physician is now legally obligated to administer to a wounded nonimmune person. In many cases reactions supervene, some of them serious and a few even fatal.

Alum-precipitated toxoid seems to provoke a superior antigenic response and it is recommended for basic active immunization. Fluid toxoid causes a more prompt increase in specific agglutinin (recall titer response). Thus, it is preferred as a stimulating, or so-called booster, injection, particularly when a wound arouses suspicion.[9]

Results of Treatment

The success attained in the actual treatment of tetanus falls far short of the results attained in its prophylactic treatment. Results of treatment of ten different series of patients in various parts of the world were reported in the period 1945 to 1953. Of the 960 patients concerned, 444 died of the disease or its complications; this is a mortality rate of 46.3 per cent (Table I). At the Mayo Clinic, sixty-six patients were treated for tetanus in the years 1911 to 1945 inclusive. Of these, 51.5 per cent died. From 1946 to 1954 inclusive, twenty-six patients have been treated, with a mortality rate of 26.9 per cent (table).

This reduction of the mortality rate from 51.5 per cent to 26.9 per cent may be fortuitous, but I doubt that it is. In 1946, some of us at the Mayo Clinic began to work as a tetanus team. However, it was not until about 1951 that the team began to function smoothly. Only one patient of the eight treated since 1951 has died. The team consists of internists, neurologists, orthopedists, anesthesiologists, pediatricians, plastic surgeons, clinical pathologists and bacteriologists. It is the responsibility of members of the team to be available for early diagnosis and to keep well informed concerning the most modern methods of treatment.

Fundamentals in Treatment

Tetanus is a complication of clostridial infection of a wound. The wound can be major or minor. In time of peace wounds may be trivial and often they have healed by the time the patient seeks medical attention. The infection usually is easily treated. Clinical tetanus, the result of the potent tetanus exotoxin combined with nervous tissue, on the other hand, is a formidable con-

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dition. The union has taken place once the patient has symptoms. The diagnosis, by necessity, is made on clinical grounds, for time is important; moreover, cultures are slow and are helpful in only 25 to 50 per cent of cases.

TABLE I. MORTALITY OF TETANUS

Institution or Region	Period, Inclusive Dates or War	Patients	Deaths	Mortality Rate, Per Cent
Charity Hospital,[14] New Orleans	1934-1944	352	158	45
Infants' and Children's Hospital,[12] Boston, Mass.	1924-1944	56*	24	43
U. S. Army Hospital,[4] European Theater of Oprs.	World War II	10†	4	40
Military and E.M.S. hospitals,[3] African and European Theater of Oprs.	1939-1945	103	48	46
Royal Melbourne Hospital,[1] Australia	1936-1946	21	10	47
Luzon,[5] Philippine Islands	World War II	54‡	28	52
Guy's Hospital[8]	1929-1946	10	5	50
Vanderbilt Univ. Hospital,[11] Nashville	Sept. 1925 to Dec. 1948	105	49	46
Univ. of Michigan Hospital,[10] Ann Arbor	1934-1952	81	34	42
General Hospitals,[13] Baltimore	1928-1953	167	84	50
Total		959	444	46
Mayo Clinic	1911-1945	66	34	51.5
	1946-1954	26	7	26.9
Total		92	41	44.6

*Children; †German prisoners; ‡civilians.

As yet no treatment will reverse the disease process or speed oxidation of the combined toxin. If, however, less than a lethal dose of toxin has combined with nervous tissue, the patient who has adequate treatment stands a good chance to recover. The disease is less likely to be fatal if the incubation period is longer than twelve to fourteen days. The morbidity, whether the tetanus is mild or severe, averages three to five weeks. Beginning with the tenth day after the appearance of symptoms, prognosis improves rapidly each day and, if the patient is alive beyond the fourteenth day after symptoms have appeared, he stands an excellent chance to recover without any residual. Death, when it occurs, usually results from: (1) circulatory failure; (2) asphyxia due to spasm of the glottis, diaphragm and intercostal muscles, or (3) sheer exhaustion.[2] Complicating pneumonia, cerebral edema, oversedation, or excess use of muscle-relaxing drugs along with serum reactions, have taken their toll.

Present Plan of Treatment at Mayo Clinic

The following plan of treatment is based partly on my experience in treatment of approximately seventy-five patients, some of them in the military hospital mentioned and some in the Mayo Clinic, partly on the experience of others at the clinic, and partly on experience recorded in published reports.

1. Preferably the patient is hospitalized, thus insuring close supervision and good nursing care.

2. On admission to hospital the patient is given tetanus antitoxin. The aim is to give the quantity necessary to neutralize any circulating or uncombined toxin and any that will be produced prior to elimination of the focus of infection. As yet, no method has been developed for determining the adequate amount for an individual patient. After a test for hypersensitivity has been made, 15,000 to 30,000 units are arbitrarily administered intravenously, or 10,000 to 30,000 units are given intramuscularly, either or both. The larger amounts are used when the wounds are deep, penetrating, or badly infected. Also, if symptoms are well advanced or are progressing rapidly, and therefore, it is suggested that overwhelming infection has occurred, the larger doses are used. Smaller amounts are then given for the next few days, until the focus of infection has been removed and the disease process stabilized.

Whenever tetanus antitoxin in horse serum is given, it is essential to have close at hand ampules of epinephrine, needles and syringes, along with oxygen and fluids for intravenous injection in case of anaphylactic reactions. Diphenhydramine hydrochloride (benadryl) or tripelennamine hydrochloride (pyribenzamine), in doses of 25 to 100 mg. (depending on age) every four to six hours, helps to prevent or to suppress serum reactions. These drugs also aid in sedation. Both are given by mouth.

3. The amount and kind of sedative drug used again depend on the needs of the individual. Enough is that amount that will keep the patient

reasonably comfortable and allow periods of restful sleep. Barbiturates, chloral hydrate, bromides and paraldehyde have been used to control mild or moderately severe tetanospasms. Depending on the age of the patient and the severity of the disease, tribromoethanol (avertin), 50 to 70 mg. per kilogram of body weight, has been used in a rectal retention enema; in my opinion this is the drug and the method of choice for sedative control of severe tetanospasms. Usually restful sleep for three to six hours supervenes and, on awakening, the patient frequently is sufficiently relaxed to be able to take food and fluids without too much difficulty. One or two administrations per twenty-four hours usually suffice but occasionally three injections are necessary. These enemas have been considered lifesaving in some cases at the clinic. Except for proctitis in one case, apparent side effects or reactions have been minor and few. Morphine sulfate, curare or other such respiratory depressant drugs, in my opinion, are contraindicated. Codeine and meperidine hydrochloride (demerol hydrochloride) are useful adjuncts. In addition to relieving pain, they tend to lessen the frequency with which tribromoethanol (avertin) must be used.

4. For adults, penicillin and dihydrostreptomycin in doses of 1,000,000 units and 1 to 2 gm. respectively, are as effective as any antibiotic therapy of which I know in combating Clostridium tetani and symbiotic organisms. Proportionately smaller doses are used for children. Antibiotic treatment affects the course of the disease only in helping to eradicate a focus, or foci, of infection and thus prevent production of more exotoxin. Even large doses of antibiotics cannot be entirely relied on to do this, however, particularly if wounds are deep and badly infected. Clostridia thrive in dead or dying tissue, where the blood supply is reduced or absent. Antibiotic treatment should be continued until the focus of infection has been eliminated. It is useful, too, in prevention or treatment of the pneumonia which is prone to develop in severe cases.

5. When treatment with tetanus antitoxin and antibiotics has been started, adequate débridement and other care of the wound are in order. Lethal doses of toxin often can be produced in trivial wounds. The natural tendency is to be too conservative. When amputation or extensive débridement is necessary, general anesthesia is best effected by giving thiopental sodium (pentothal sodium) intravenously, or tribromoethanol (avertin) rectally. Ether tends to be irritating to the respiratory tract, to increase laryngospasm and to predispose to pneumonia. If use of an oxidizing agent is indicated, moist potassium dichromate dressings are better, in my opinion, than hydrogen peroxide dressings, which frequently are irritating to the skin.

6. Tracheotomy should be done whenever respiration is significantly embarrassed by laryngospasm or accumulation of tracheobronchial secretions.

7. A high-caloric diet is indicated where metabolism is greatly increased. Liquid to soft foods are tolerated better than are solids. Multiple small feedings are more suitable than three large meals. A nasal tube can be used when necessary and when it does not aggravate the condition. Supplementary fluids containing glucose, proteins, electrolytes and vitamins are given parenterally when they are needed to maintain hydration, nutrition and electrolyte balance.

8. Oxygen and a portable respirator are used when indicated.

9. Urinary retention has to be relieved in some cases.

10. Cortisone, compound F (hydrocortisone) and adrenocorticotropic hormone (ACTH) are the newest preparations which apparently are useful in this condition. The action and value of hormonal therapy in tetanus have not been exactly determined. At present, the members of the team described earlier reserve use of the hormones for two groups of patients: (1) allergic patients who react to, or who are known to be hypersensitive to, tetanus antitoxin. For example: One patient had chronic asthma and hay fever. A year before his admission with tetanus, he had been given tetanus antitoxin after an automobile accident. A severe reaction followed. Intravenous administration of adrenocorticotropic hormone (ACTH) and then oral administration of cortisone made it possible to follow the usual program of administration of tetanus antitoxin.

Sheer exhaustion often is the only apparent cause of death in cases of this disease. When exhaustion becomes apparent, our team uses cortisone purely as a supportive measure in an attempt to maintain life while the patient is passing the crisis of this self-limited disease. Depending on the age, sex and condition of the patient, in the absence of absolute contraindications, 100 to 300 mg. of cortisone is given each day in divided doses, until the crisis has passed. Thereafter doses are reduced and gradually use of the preparation is discontinued. Oral administration is preferred but the substance can be given intramuscularly if necessary.

Summary

Since 1945, the mortality rate of clinical tetanus at the Mayo Clinic has fallen from 52 per cent to 27 per cent. Apparently this is attributable, not to any new treatment pariculary, unless such be the use of cortisone and adrenocorticotropic hormone (ACTH), but to the formation of a tetanus team. The team has the responsibility of aiding in early diagnosis of the disease, and of standardizing treatment so that therapeutic measures now available will be used most effectively. The present plan of treatment which the team applies is summarized in the body of the paper. One of four patients still is lost. This means that treatment of tetanus is not yet satisfactory. Although tetanus toxoid is effective in preventing the disease, and antibiotics are effective in destroying Clostridium tetani, a method which will reverse the disease process, once the exotoxin has become fixed in the nervous tissue, is lacking.

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ANEMIA IN PREGNANCY

About 80 per cent of normal patients manifest decreases in hematologic values to a variable degree during pregnancy.

A physiologic anemia does not occur in pregnancy. Minimal or moderate decreases in the hemoglobin are usually the results of an iron deficiency.

Associated with any decrease in hemoglobin there is a decrease in the serum iron and elevation of the erythrocyte protoporphyrin. These have been shown to indicate the existence of iron deficiency.

A hemoglobin of 12 grams per cent has been found to be an approximate minimal normal hemoglobin for the pregnant state. Only 20 per cent of pregnant patients not receiving supplemental iron maintained their hemoglobin above 12 grams per cent.

Approximately 80 per cent of pregnant women maintained or improved their hemoglobin values when given an iron supplement with their diet.

Every pregnant woman should receive iron during pregnancy. A minimum of ninety days has been found to constitute an adequate trial. Iron should preferably be administered late in pregnancy when the iron demands are the greatest.

Ninety per cent of pregnant women maintained or improved their hemoglobin values when given a combination of iron and cobalt (Roncovite). A significantly higher proportion of patients receiving iron and cobalt delivered with a hemoglobin above 13 grams per cent when this series was compared with the iron treated series.

No toxic manifestations associated with its use have been observed.—R. G. Holly, *Obstetrics and Gynecology,* 5:1 (April) 1955.

Tumors of the Colon and Rectum

Fundamental Considerations in Diagnosis and Treatment

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THE PURPOSE of this paper is primarily as an aid to diagnosis and treatment of benign and malignant tumors of the colon and rectum. Since the majority of these tumors will be either adenomas or carcinomas, most of it will deal with their detection and treatment. The physician should never rule out neoplasms of the colon and rectum on the basis of history alone. Many tumors present in the large bowel and rectum may be asymptomatic or present symptoms such as flecks of blood in the stool which are undetected by the patient. For this reason every complete physical examination should include a digital and also a proctoscopic examination of the terminal bowel. A carefully done digital examination should rule out neoplasms of the anal canal and distal rectum. A proctoscopic examination to 25 cm. will reveal 75 per cent of the tumors of both the rectum and the large bowel.

The commonest malignant tumor arising from the anal canal is a squamous cell carcinoma. This may arise from either the perianal skin or from the skin lining the anal canal proper. It should be suspected whenever a chronic ulcer with heaped-up or rolled-up edges is found in this region. More rarely, a basal cell carcinoma or malignant melanoma may be found in this region. The latter represents the most malignant of all colon and rectal tumors. It is rarely nonfatal. Ulcers suspected of being malignant should be widely excised and examined by a competent pathologist. If an invasive type of malignancy is detected, a radical combined abdominal-perineal resection should be done followed by a bilateral inguinal lymph node dissection and later a course of deep x-ray therapy. In cases where there is lymph node invasion the prognosis is usually very bad.

Benign tumors of the anal canal and perianal region are commonly encountered. Simple skin tags, papillomata, condylomata of various types and hypertrophied, fibrosed anal papillae are among those most frequently seen. The possibility of carcinoma in these lesions should always be kept in mind. Occasionally a low grade squamous cell carcinoma may be found in the periphery of a simple anal skin tag. Most of these tumors can be excised locally or destroyed by fulguration with a very low percentage of recurrences. Before disposing of them an adequate specimen should be sent to the pathologist for examination.

In examining the distal rectum with the index finger, tumors arising in the extra rectal structures should be carefully excluded. The prostate gland and seminal vesicles should be carefully palpated for nodules or irregularities. In the female the cervix and the base of the uterus and adnexa may be felt anteriorly to the rectum. Malignancies arising from these structures may directly invade the bowel wall.

Endometriosis may be detected in the rectovaginal septum as shotty nodes that are tender during menstrual periods. In exceptional cases endometriosis may invade the mucosa of the bowel and be indistinguishable clinically from carcinoma. Metastatic nodules from distant carcinomas may be felt anteriorly to the rectum in the peritoneal culdesac, forming the so-called Blumer's shelf. Masses palpated in the presacral areas are most often inflammatory in character but rarely teratomas, dermoids, chordomas or meningoceles may be felt. Other extrarectal nodules or masses include lipomas, fibromas, liomyomas or carcinoids. Usually these masses are found on routine examination or produce symptoms due to their bulk or in rarer instances

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produce erosion of the rectal mucosa or actually invade the lumen of the rectum.

Adenomas and carcinomas arising from the rectal mucosa will constitute the bulk of the tumors found in the colon and rectum. Adenomas may be sessile, papillary or pedunculated, single or multiple. They may measure only a few millimeters in diameter up to several centimeters. The smaller ones are more common and the surface of the polyp may be smooth, lobulated or ulcerated and may vary in color from the general appearance of the surrounding mucosa to a deep, dusky red. Occasionally a polyp may be attached high in the colon by a long pedicle and because of invagination of the bowel into the rectum or for other reasons prolapse into the rectum or even through the anal canal. This type of tumor may be difficult to demonstrate, particularly if the polyp returns high into the sigmoid colon on proctoscopic examination. Usually if the patient is asked to bear down during the examination, the polyp will once more prolapse to within range of the sigmoidoscope. Anesthesia is a detriment in attempting to visualize this type of tumor. The presence of induration, ulceration or attachment by a broad base of an epithelial tumor suggests the presence of malignancy.

If one polyp is found on sigmoidoscopic examination an air contrast barium enema should be done to rule out additional neoplasms higher in the bowel. Frequently a single polyp seen on sigmoidoscopic examination may be a satellite or sentinel polyp beyond which a frank adenocarcinoma may be growing. Approximately 20 per cent of the patients having polyps within the reach of the sigmoidoscope will have an additional one higher in the bowel. Polyps beyond the reach of the sigmoidoscope may be difficult to demonstrate with roentgenograms. A carefully done air contrast study may demonstrate tumors .5 cm. or even less in diameter. Before x-ray examinations are done the bowel must be thoroughly clean and free of fecal material. A careful air contrast study and a thorough fluoroscopic examination may have to be done repeatedly to detect the smaller tumors. A routine barium enema is usually inadequate in searching for colonic polyps.

A condition may be encountered characterized by several widely scattered polyps. These may extend at irregular intervals from the rectum cephalad to the cecum. If familial history exists with this condition it is commonly referred to as familial polyposis. Polyposis may follow chronic inflammatory disease of the colon such as chronic ulcerative colitis in which hypertrophied mucosal tags have formed a polypoid condition throughout the colon. At other times, without any hereditary or inflammatory background, several widely scattered polyps may be found throughout the colon. Carcinomatous changes are common in these cases. Multiple polyposis, regardless of the source of origin, is best treated by colectomy and ileoproctostomy. If carcinoma is found in the rectum, a combined abdominal-perineal resection must also be done and a permanent ileostomy established. Simple multiple colotomies with removal of individual polyps are not advisable unless a patient is a poor surgical risk.

Single polyps visualized from below should be biopsied either totally or by several adequate bites. The pedicle or base may then be fulgurated. If a superficial or non-invasive carcinoma is found, that is, one in which only the mucosa of the polyps is involved, thorough fulguration of the base will usually result in cure. Carcinoma which has penetrated the muscularis mucosa or muscularis should be treated by abdominal-perineal resection if in the lower two-thirds of the rectum, or occasionally by segmental resection and primary anastomosis if in the upper third of the rectum.

Biopsies and fulguration proximal to 13 cm. from the anal verge must be done with care to avoid perforation of the bowel wall. Lesions below 13 to 15 cm. can usually be fulgurated without danger of perforation except anteriorly where the peritoneal reflection in women may extend to within a couple of centimeters from the peritoneal floor. Single polyps beyond the reach of the sigmoidoscope are treated either by colotomy or segmental resection. If at the time of surgery the polyps appear to be flat, broad based and sessile, indurated or for any reason are suspected of being malignant, a segmental resection with excision of a broad wedge of mesentery is done. A small tumor on a long pedicle may be removed by simple colotomy and excision. We do not believe that segmental resection is necessary or advisable in all cases of a single polyp. However, if there is any doubt in the surgeon's mind as to the character of the tumor, segmental resection should be done. A frozen section should be made at the time of operation. While in many instances these examinations are inconclusive, there are cases in which a positive diagnosis of malignancy can be made. After the bowel is opened, a sterile sigmoidoscope should be routinely introduced to 25 cm. proximal and distal to the point of incision to rule out lesions beyond this point. The rest of the colon should be carefully palpated for additional lesions.

Frank carcinomas present in the rectum can easily be identified by their induration, rough, irregular edges and tendency to ulcerate. These are usually flat invasive lesions with a central ulceration and raised margins, but may be polypoid. The polypoid lesion may vary from a small tumor with malignant changes to large tumors completely surrounding and obstructing the bowel. Regardless of their clinical appearance they should be adequately biopsied. Preferably, the biopsy should be taken at the edge of the tumor near what appears to be the junction between the tumor and the normal bowel mucosa. Occasionally what appears to be grossly malignant may be diagnosed on microscopic examination as a simple infectious granuloma. Tumors lying in the lower two-thirds of the rectum should be resected by an abdominal-perineal resection. Tumors proximal to this in the upper third of the rectum may in some instances be removed by anterior resection and primary anastomosis. At least 8 cm. of normal bowel should be resected caudad to the tumor mass to accomplish adequate resection. Carcinoma of the sigmoid is usually removed by segmental resection, removing a wide wedge of mesentery and regional lymph nodes. A primary anastomosis is then done. Carcinoma of the descending colon and in the splenic flexure is resected by hemicolectomy and primary anastomosis. The same applies to cecal and hepatic flexure tumors. Tumors of the transverse colon are removed by wide segmental resection and removal of most of the transverse mesocolon.

A rare type of tumor which is occasionally found in various age groups is the villous papilloma. This tumor is characterized by its soft velvety appearance and many small finger-like projections. Usually it bleeds readily and secretes a large amount of mucus. These tumors are usually single and may be extensive enough to completely surround the entire bowel. About 10 per cent of these tumors are malignant and they should all be resected locally by the total biopsy method and thoroughly sectioned for evidence of malignancy. If malignancy is found it should be re-evaluated and a more radical procedure done. These tumors if benign on microscopic section are notorious for their tendency to recur locally.

Non-malignant tumors found in the colon and rectum include lipomas, fibromas, lymphomas, liomyomas and carcinoids. All of these are relatively rare. Usually they are felt as submucous tumors over which the mucosa is freely movable. They are easily removed by incising the mucosa and shelling out the tumor. They should all be subjected to microscopic scrutiny, and if any evidence of malignancy is found, a radical procedure is indicated.

Treatment of carcinoids is a matter of controversy. Up to 10 per cent of these tumors have a tendency to be invasive and metastasize. When these tumors are excised, a segment of the surrounding muscular coats of the bowel should be removed, and if on microscopic examination invasion is discovered, additional radical surgery is indicated.

Summary

1. A good digital and proctoscopic examination should be a routine part of all physical examinations.

2. Tumors suspected of lying above the reach of the sigmoidoscope should be investigated by thorough air contrast barium enema study with the x-ray and fluoroscope.

3. Most tumors lying in the rectum within the reach of the sigmoidoscope can be totally or adequately biopsied from below followed by fulguration of the base of the smaller ones. Microscopic study then should be the basis for further treatment.

4. Carcinomas lying in the lower two-thirds of the rectum are best treated by abdominal-perineal resection. Tumors lying above this area may be treated by anterior resection or segmental resection and primary anastomosis.

Topical Hydrocortisone in the Treatment of Skin Diseases

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THE PURPOSE of this report is to review a year's experience with topical application of hydrocortisone in the treatment of various skin diseases. It is well known that the steroid hormones exert a beneficial effect on many inflammatory conditions of the skin. Unfortunately, their systemic administration is attended by not inconsiderable risk, and this risk is only occasionally justified. In contrast to its effectiveness in certain eye disorders, cortisone has been largely unsuccessful when applied to the skin. However, it has become apparent that topical hydrocortisone is much more effective than cortisone, and in fact makes a considerable contribution to the treatment of at least a few troublesome skin diseases.

Table I presents the results of hydrocortisone ointment treatment of 147 cases of various dermatoses. The cases cannot be said to be unselected, but because of the relative expense of this treatment, the cases treated tended to be difficult ones, often of long duration and unsuccessfully treated with many other preparations. The results are classified as 0, ±, no or virtually no improvement; +, minor or short term improvement only; + +, considerable improvement; + + +, great but incomplete relief, or complete relief which did not persist through the observation period; and + + + +, complete "cure", lasting throughout the followup interval. It will be noted that there are relatively few + + + + results, which indicates that hydrocortisone ointment is no panacea, but this should not be interpreted as too great an indictment of the drug, because many of the + + or + + + results represent considerable therapeutic success.

The patients with atopic dermatitis presented some of the most gratifying results. Past treatment of this disorder has been disappointingly unsuccessful, and many of the patients in this group had been to a very large extent incapacitated by their illness, as well as being disfigured and uncomfortable. The patients showing + + or + + + improvement, while not completely cured by hydrocortisone, were often improved to the point of being able to resume normal activities. Several volunteered the comment that this drug gave the most relief of anything they had ever used.

TABLE I. DEGREE OF IMPROVEMENT

	No. of Cases	0	±	+	++	+++	++++
Atopic dermatitis	31	2	3	6	10	7	3
Hand and other chronic eczemas	48	3	2	12	21	7	3
Seborrheic dermatitis, including otitis externa	8	—	—	1	4	2	1
Acute dermatitis, including venenata	12	3	1	2	3	3	—
Lichen simplex chronicus	11	3	1	2	2	3	—
Nummular eczema	3	—	—	—	1	2	—
Dysidrotic eczema	5	—	1	2	1	—	1
Intertriginous dermatitis	1	1	—	—	—	—	—
Mycotic dermatitis	1	1	—	—	—	—	—
Mycotic "ID" eruptions	1	—	—	—	1	—	—
Blepharitis	3	—	—	—	1	2	—
Recalcitrant pustular eruption of palms and soles	1	1	—	—	—	—	—
Acrodermatitis continue of Hallapeau	1	—	—	1	—	—	—
Pruritus, anal, scrotal and vulvar	12	1	—	—	3	9	—
Psoriasis	3	2	1	—	—	—	—
Lichen planus	2	1	—	1	—	—	—
Lupus erythematosus	1	1	—	—	—	—	—
Rosacea	1	1	—	—	—	—	—
Darier's disease	1	—	—	1	—	—	—

An example of a good result was a patient who had been hospitalized at least twenty times in the last nine years. He has now been working for ten months without interruption, the longest time he has ever put in on the job in his whole adult life. He is by no means free of eczema, but he is able to control it to the point where he is not incapacitated by it. Another patient in the + + + group has now used the drug for fifteen months, the longest of any patient in this entire series. Before starting the use of this preparation he was required to sleep with a wire mask on his face in order to prevent dis-

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figuring himself by scratching in his sleep. His experience with compound F is interesting and presents some features common to many patients. When he first started its use, he obtained dramatic improvement. After three or four months, the drug gradually lost its effectiveness until the patient discontinued treatment. After another three or four months, during which time he tried several different remedies with poor results, he again tried hydrocortisone ointment. He has now used the drug in conjunction with a patent "softening" cream for over seven months, and remains in the best condition he has been in for twenty years. There is no present evidence of hydrocortisone ointment again losing its effectiveness. Several other patients in this group have also used hydrocortisone in conjunction with other topical remedies with apparent success, about which more will be said later.

The second group, though heterogeneous, represents some of the dermatologist's commonest and most difficult problems. It contains the chronic hand eczemas, and various chronic scaly inflammations which come under the heading of neurodermatitis, chronic contact dermatitis, or perhaps more accurately, eczema, cause undertermined. In general, the results in this group, though not spectacular, were good, considering the long history of unsuccessful previous treatment given by many of these patients. Here again, many of the patients were treated with combinations of hydrocortisone with other topical remedies such as tar, ichthyol, vioform, et cetera. The eight patients with seborrheic dermatitis did reasonably well. In latter months an ointment containing neomycin in addition to hydrocortisone was used and seemed to produce slightly better results in seborrheic dermatitis than hydrocortisone alone. It is commonly recognized that there is an element of at least secondary bacterial infection in this disease. In lichen simplex chronicus, hydrocortisone ointment was somewhat less successful than in other chronic inflammations. This is understandable, in that lichen simplex chronicus very probably is of psychic origin and no topical remedy could be expected to correct the underlying cause. It is interesting that in several instances, objective improvement in the lesions far exceeded subjective, in that the plaques lost their inflammatory character while continuing to itch.

In nummular eczema the results were good, and it is felt that hydrocortisone ointment makes a definite contribution to the treatment of this stubborn eruption. No effect was produced in an acute vesicular fungus infection of the feet, while an "id" eruption on the hands was benefited. The result in three cases of blepharitis of various causes was good.

The cases of acute dermatitis point up the weakness of this form of therapy as well as its strong points. Perhaps it would be well to discuss briefly the cases which were not helped. One was a patient with acute poison ivy eruption. Application of hydrocortisone ointment did not materially retard the development of the full-blown bullous reaction. Another patient was a florist who developed a severe dermatitis from chrysanthemums. She was unable to stop work and so continued contact with the offending allergen. Hydrocortisone application did not help her very much. Another similar patient was a farmer with a weed dermatitis on the legs. He continued to work and was not benefited by treatment.

On the other hand, those cases in which the causative substance was identified and could be avoided did much better. Illustrative cases included an acute dermatitis from contact with rubber, and another from adhesive tape. They recovered very quickly after discontinuing contact with these substances and the application of hydrocortisone. Admittedly, they would have gotten well anyway, but it seemed that their recovery time was materially shortened in comparison to similar cases treated by older methods.

The patients with pruritus ani, scroti and/or vulvae treated with hydrocortisone ointment were among the most grateful in this series. The results in these patients were strikingly good. An example is a man who had had pruritus ani since World War II. He had had a lot of treatment without much relief. He obtained immediate complete relief from his itching and gradual return of the skin to a normal appearance, and needs to apply the ointment only once every two to three days in order to remain completely symptomless. On the debit side, it must be reported that one patient who had good immediate relief noted a gradual decrease in results after five months' use, and finally discontinued using the ointment. It is interesting that, in this case, increasing the strength of the ointment from 1 to 2.5 per cent did not produce any better results. The one failure was a case of acute anal pruritus following the oral use of an antibiotic. This is the only patient with pruritus from antibiotics treated in this series, but at least one author has reported good results in similar patients.

Hydrocortisone topically may be said to be of little value in the treatment of psoriasis, lichen planus, discoid lupus erythematosus, rosacea and Darier's disease. The patient with psoriasis who was slightly helped was a patient with an acute irritative reaction in the anogenital area, such as is occasionally seen in "inverse" psoriasis. This irritation may be thought of as comparable to any other acute dermatitis. In this patient it was rapidly controlled by hydrocortisone, but the psoriasis itself was unaffected. The patient with lichen planus who was helped to some degree had a severely itching plaque over a pretibial surface. The itching was lessened considerably by hydrocortisone, and hence the irritation resulting from scratching, but the lichen planus itself was unaffected.

Contraindications

So far, no contraindications to the use of this ointment have been established. None of the patients in this series has been made worse by it. No sensitivity to topically applied hydrocortisone was observed or has been reported in the literature. The possibility of systemic effect from percutaneous absorption of hydrocortisone must be considered, but no evidence of it was observed, and it is almost mathematically impossible for patients using the amounts of drug used in this series to absorb any appreciable quantity of it. An experimental study has shown that topical application of extremely large amounts of hydrocortisone had no demonstrable effect on the circulating eosinophile count.

Another theoretical possibility is the spread of infection on the skin during the use of hydrocortisone. Actually no evidence of this was observed, though the ointment was not used in grossly infected lesions. To obviate this theoretical possibility, however, there is now on the market a combination ointment containing neomycin in addition to the hydrocortisone. This was used in several cases, and in at least two cases of seborrheic dermatitis (in which there is probably an element of infection) gave better results than the plain hydrocortisone.

In this series, combinations with other drugs were tried, including tar and ichthyol ointments and pastes, vioform ointment, et cetera. No attempt was made to compound these together, but the hydrocortisone ointment was applied first, massaged into the skin, and the other preparations then applied over it. In several instances these combinations appeared to give better results than either one of the pair used singly. This sequence was actually attempted in several instances.

There are now several brands of the ointment available. They differ only in the ointment base used, some being in a vanishing cream type base and others in a grease. Some patients expressed a preference for the grease base, others for the vanishing cream base. The grease base has the advantage of being somewhat less costly to use, in that it spreads more thinly on the surface of the skin, hence less is needed. To further confuse the issue, the ointment combination with neomycin is in yet a third type of base.

An objection to the use of this preparation which is always raised immediately is the great expense of the drug. Admittedly, it is expensive, and several patients have discontinued its use for that reason. On the other hand, the expense is less than it may appear, in that only very small quantities of ointment are necessary. The technique is to apply it in as thin a layer as possible and then massage it into the skin until practically none is left on the surface. This is necessary only twice a day, and sometimes even less often. Used in this way, the drug is pleasant to use and a 1 dram tube will last for several days or longer, even when used on relatively large areas of skin. This brings the treatment's cost down to a point where it compares not too unfavorably with that of many other medications. It is perhaps not widely enough appreciated how expensive practically all drugs are in the present age. Several patients volunteered the opinion that the cost of the ointment was well worth while in view of the benefit obtained. Certainly in the extreme cases in this series, particularly atopics, in which the patient was able to continue working and was spared the expense of hospitalization, the cost of the ointment was easily justified, even if necessary quantities cost several dollars a week.

There have already been several reductions in the cost of the ointment, and it is hoped that there will be more in the near future. Recently there has been marketed a 20-gram container which sells for roughly two and one-half times the cost of the 5-gram tube. This affords considerable saving to those patients requiring larger quantities.

An attempt was made to reduce the cost of therapy by making the ointment by grinding tablets of hydrocortisone and incorporating the powder in an ointment base. This reduces the cost of the actual material used about a third, but there is considerable labor required of the pharmacist, and there is further possibility that the resulting unsterile ointment may deteriorate rapidly. This technique is probably not justified except in large clinics where considerable quantities of the ointment are being dispensed within only a few days after preparation.

A final favorable factor in the cost of the ointment is that in almost all cases the 1 per cent ointment seemed to work about as well as the 2.5 per cent, at less than half the cost. In only exceptional instances was the 2.5 per cent definitely superior.

Summary and Conclusions

Ointments containing hydrocortisone were used in the treatment of 147 patients with a variety of inflammatory skin diseases. Results indicate that the preparation is of considerable value in chronic eczemas and in pruritus, but of little help in psoriasis, lichen planus, lupus erythematosus, et cetera.

Discussion

H. F. R. Plass, M.D.—I would like to compliment Dr. Goltz on this excellent paper and on his conservatism, both in the handling of the corticosteroid and in the interpretation of the results. This conservatism deserves to be emphasized so far as prescribing ACTH and cortisone, even when exceedingly small doses are used. A number of cases of Addisonian crisis have been reported precipitated either by surgical trauma or automobile accidents following the injection of as little as 25 mg. of hydrocortone into the knee joints. In the case of patients who have taken cortisone or ACTH, I believe it is more important that they carry identification than it is with the diabetic. This should take the form of a card in the billfold or purse, stating that these drugs have been used, so that if the individual is injured in an accident, necessary supportive measures may be taken to prevent these crises and probably death. This is more important for the ACTH or cortisone patient than it is for the diabetic, for the diabetic eventually identifies himself with a little sugar in the urine, while these patients simply go into shock and become edematous.

I. Fisher, M.D.—I agree with Dr. Goltz that there is much to be said for the use of hydrocortisone ointment in the chronic lichenfied dermatitis. This is particularly true in the case of the atopic dermatitis or in the sharply localized chronic lichenfied dermatitis. These appear to respond well to the use of hydrocortisone ointment in most instances. The ointment in itself is not a cure-all, nor does it clear all of these. It does, however, prove to be a very valuable adjunct in treatment and in many instances the only agent needed. I have not seen results quite as good as those Dr. Goltz has pointed out in some of these cases, but this may very well be a matter of individual interpretation and persistence in the use of the agent. I would certainly agree that there is a definite need for and room for the use of this preparation in the care of atopic dermatitis.

I do feel also that there is some difference in the results from 2.5 per cent hydrocortisone ointment compared to the 1 per cent and there are some instances when it does have its very definite merit and indications, particularly where the 1 per cent ointment has failed. One must admit also that the physical properties of an ointment are often as important as its specific therapeutic effect. That may explain the reason for the difference in the effect of hydrocortone ointment depending on the base in which it is incorporated. One may have to feel his way along here, using one type of base or the other in order to obtain the best results in the individual patient.

I have seen only two instances of any local irritation from hydrocortone ointment. These have not been instances of specific contact dermatitis but more apparent local irritation.

Another advantage in the use of the medication locally lies in the fact that then we may refrain from the use of such a potent agent systemically or at least to be able to cut down on the dose of this drug used systemically. It is our feeling that cortone or hydrocortone should be used very cautiously in the dermatologic patient, when it is administered systemically. There are not too many indications for its use in the acute or chronic dermatitis. We feel that this drug has been used somewhat loosely in the past and the time may come when we will have some repercussions from it. We cannot stress too strongly the need for caution in the use of this drug and for going without it whenever possible, particularly when local management will do a good job. This is especially true in the acute processes that will subside with good conservative management rather than with something as radical as ACTH, cortisone, or hydrocortone systemically.

A New Hydrocortisone for Intra-articular Use

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THE MAJOR limitation attending the intra-articular use of hydrocortisone acetate in rheumatoid arthritis is the relatively short duration of action of this drug. Although about 90 per cent of patients can expect significant local relief of symptoms within twenty-four hours following the injection of 25 to 50 mg. of hydrocortisone acetate into an actively inflamed joint, this relief usually lasts for only two weeks or less.[1,2] Occasionally the effect lasts as long as a month, and only rarely much longer.

In an attempt to obtain a more prolonged local effect from hydrocortisone acetate the author has studied several different methods of administering it.[4] Instead of the usually recommended dose of 1 to 1.5 cc. (25 to 37.5 mg.), patients were given 2 to 3 cc. (50 to 75 mg.) at one time. Although many patients obtained no greater benefit from the higher dose, in a large number local relief was prolonged by several days to a week or more. The author now makes it his regular practice to inject a minimum of 2 cc. (50 mg.) of hydrocortisone acetate into most of the larger joints.

A logical extension of this experience was the use of massive doses of hydrocortisone acetate intra-articularly. Approximately 100 injections of 125 to 250 mg. each were made into the knee joints of more than thirty patients. This study is incomplete but it appears that definite prolongation of benefit can be effected in many patients. There was no increase in adverse local reactions, and some patients showed systemic improvement for two to four days indicating probable absorption of the drug. Two patients, both of whom received three such massive injections, experienced "local remissions" in their knees. The "local remissions" have now continued for more than eighteen months despite the persistence of active synovitis in other joints.

A third method of attempting to prolong the local effect of hydrocortisone acetate involves the weekly injection, for a four to six-week period, of 1 to 2 cc. of the drug even though the joint remains asymptomatic. The majority of patients treated in this manner showed no cumulative effect. A few had relief lasting more than a month following the last injection, and one had a "local remission" of more than a year. One cannot help speculating as to the possible future replacement of surgical synovectomy by some chemical means.

Present Study

The present report concerns a less soluble ester of hydrocortisone, hydrocortisone-tertiary-butyl acetate, which was developed in the research laboratories of Merck and Co.* A preliminary report by Hollander et al[3] indicated that approximately 50 per cent of patients obtained better results with this compound than with ordinary hydrocortisone acetate, the duration of effectiveness of the new compound being two to ten times greater. Several other analogues of hydrocortisone which they tested showed no comparable superiority.

Thirty patients with rheumatoid arthritis were given, at different times and into the same joint, equivalent doses of hydrocortisone acetate and hydrocortisone-tertiary-butyl acetate. The local response was recorded in terms of the degree of improvement and its duration. The detailed data are presented in Table I. In no patient was the new compound less effective than hydrocortisone acetate. In five patients both drugs were equally effective. In the remaining twenty-five patients hydrocortisone-tertiary-butyl acetate showed some superiority. In eighteen patients this superiority was demonstrated by a longer duration of beneficial effect; in two patients by a greater degree of benefit; and in five patients by an increase in both degree and duration of local joint benefit. The average duration of relief for hydro-

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cortisone-tertiary-butyl acetate was two to three times that of hydrocortisone acetate. No adverse reactions were encountered with either drug beyond the occasional (5 to 10 per cent) mild to moderate increase in local joint symptoms for four to 12 hours following an injection.

TABLE I. RHEUMATOID ARTHRITIS

Patient	Joint	Hydrocortisone Acetate			Hydrocortisone-tertiary-butyl Acetate		
		Dose mg.	Grade of Improvement*	Total Duration† Days	Dose mg.	Grade of Improvement	Total Duration Days
1	elbow	50	III	10	50	III	42
	knee	50	III	28	50	III	28
2	both knees	50	III	4	50	III	12
	both knees	125	III	14	125	III	20
3	ankle	50	IV	24	50	IV	42
4	lt. knee	50	III	14	50	III	28
	rt. knee	75	III	14	75	III	28
5	knee	125	III	42	125	III	42
6	both knees	50	IV	14	50	IV	28
	both knees	75	IV	14	75	IV	56+
7	lt. knee	30	III	21	30	III	20
	lt. knee	50	III	23	50	III	20
8	lt. knee	25	III	10	25	III	30
	rt. knee	50	III	8	50	III	24
9	knee	25-50(×4)	I	10	50	III	56
10	rt. knee	125(×2)	IV	21	125	IV	49
	rt. knee	50	III	7	50	III	28
	lt. knee	50	III	3	50	III	21
	lt. knee	125	IV	28	125	IV	49
11	both knees	50	III	10-12	50	III	24-28
12	knee	50	III	40	50	III	42
13	knee	250	II	35	250	III	35
14	elbow	50	III	10	50	III	23
15	knee	50	II	7	50	II	14
16	knee	50(×2)	I	4	50	IV	21+
17	wrist	50(×2)	II	42-56	50	III	56
18	knee	50(×2)	I-II	7	50	III	42
19	lt. knee	50	IV	8	50	IV	22
	lt. knee	125	II	14	125	IV	21+
20	knee	125(×2)	II	2-4	125	II	21
21	knee	125(×3)	I	7-14	62.5	II	35
22	knee	125	III	21	50	III	28
23	knee	50	III	14	50	III	21
24	knee	50	III	56+	50	III	56+
25	wrist	50	III	56	50	III	56+
26	knee	75	II	21	75	II	24
27	knee	50	III	7	50	III	14+
28	rt. knee	30	II	28	50	II	30
	lt. knee	90	III	42	75	III	45
29	lt. knee	125	III	28			
		250	IV	42	50	III	56
30	rt. knee	125	III	28	125	III	42

*Grade I = 10 to 25%; II = 25 to 50%; III = 50 to 95%; IV = complete relief.
†Duration of any improvement, that is, joint returned to pre-injection state.

TABLE II. OSTEOARTHRITIS

Patient	Stage	Hydrocortisone Acetate			Hydrocortisone-tertiary-butyl Acetate		
		Dose mg.	Grade of Improvement	Duration of Improvement Days	Dose mg.	Grade of Improvement	Total Duration Days
1	chronic	125	II	4	50	III	28+
2	subacute	50	II	3	50	II	21
3	subacute	50	IV	4	50	IV	35+
4	subacute	75	II	14	75	III	14+
5	chronic	50	I	5	50	I	7

Osteoarthritis

As has been previously recorded by the author,[1] the chief usefulness of intra-articular hydrocortisone for degenerative arthritis is in the patient suffering from an acute or subacute exacerbation of his symptoms. Five patients with osteoarthritis of their knees were given injections of both hydrocortisone acetate and hydrocortisone-tertiary-butyl acetate with the results recorded in Table II. In one patient with chronic disability neither drug was particularly effective, but in the remaining four the new compound appeared to offer a significant advantage.

Shoulder Periarthritis or "Bursitis"

Due to the considerable day-by-day variation in symptoms, the high incidence of spontaneous improvement, and other factors, the evaluation of a therapeutic agent for periarthritis or so-called "bursitis" of the shoulder is beset with many difficulties. However, since the procedure is simple, safe and inexpensive, and with the belief that there is a logical rationale to its use, the author has adopted the practice of treating most of his patients with severe acute or subacute periarthritis by the localized infiltration of the injured and inflamed rotator cuff tendon with hydrocortisone acetate. Approximately 75 per cent of patients so treated have obtained 50 per cent or more relief of symptoms within twenty-four hours following a single injection. In addition to the relief of pain, the procedure has the added benefit of allowing earlier use of physical therapy for the correction of the contracted shoulder that occurs in so many patients.

Nine patients with acute or subacute periarthritis were treated with the local injection of 25 to 50 mg. of hydrocortisone-tertiary-butyl acetate. Two patients received little or no relief from the injections and the remaining seven obtained 50 per cent or more relief of symptoms. One patient who showed no relief following x-ray therapy the week before obtained a grade 2-plus response. Another patient obtained a grade 4 response within twenty-four hours whereas she had failed to benefit from x-ray therapy and two injections of hydrocortisone acetate given by her physician. From this limited group, however, it appears that the overall results with hydrocortisone-tertiary-butyl acetate are rather similar to those obtained with hydrocortisone acetate.

Summary

A comparison is made of hydrocortisone acetate and hydrocortisone-tertiary-butyl acetate when injected into the joints of thirty patients with rheumatoid arthritis. In 80 per cent of cases this new and more insoluble hydrocortisone ester demonstrated some superiority. This superiority was usually a prolongation of the local anti-inflammatory effect to two to three times that of hydrocortisone acetate.

Four of five patients with osteoarthritis of the knees showed a similar greater benefit from hydrocortisone-tertiary-butyl acetate.

Nine patients with acute or subacute periarthritis of the shoulder were given local tendon injections of hydrocortisone-tertiary-butyl acetate. Only one patient was also given hydrocortisone acetate, but when compared with previous experience with hydrocortisone acetate, the new ester appears to offer about the same degree of benefit.

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WIDOWS

The number of widows in the United States increased by 90 per cent during the years between 1920 and 1953, while the adult population of women gained only 63 per cent. In 1953, there were almost 7.5 million widows in the United States. At present, one woman in every ten in the age group from forty-five to fifty-four is a widow, and the proportion is more than one in every four at ages fifty-five to sixty-four.

The recent increase in widows is apparently chiefly in the cities. The statisticians who are responsible for these figures believe that many widows in rural areas who do not remarry soon move to the city.

The importance of these statistics lies in the fact that widows inherit considerable assets and thus have a considerable voice in the economic affairs of the country. The problem is important because of the large number of dependent children left in the care of widows, and the relationship between broken homes and delinquency is clearly established. Another question is the disproportion in life expectancy of the male population to that of the female population which is so significant as to demand special consideration.—M. Fishbein: Editorial, *Postgraduate Medicine,* 17:338 (April) 1955.

Transplantation of Duct of Wirsung in Chronic Pancreatitis

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CHRONIC relapsing pancreatitis constitutes a serious organic disease of the pancreas characterized by acute attacks separated by nearly complete remissions. Since the recognition of mild acute edematous or interstitial pancreatitis by Zoepffel[19] in 1922 and Elman[6] in 1933, and the introduction of a practical serum amylase test by Somogyi[17] in 1938, the clinical diagnosis of acute pancreatitis has been much easier and more common.

An unknown fraction of patients suffering from attacks of pancreatitis are destined to have recurrent attacks causing pancreatic dysfunction, such as diabetes mellitus, steatorrhea, creatorrhea, pseudocysts, severe abdominal pain and calcification, resulting malnutrition, anemia and drug addiction in about half the cases.[4]

Cause of Chronic Pancreatitis

Chronic relapsing pancreatitis appears to be repeated attacks of acute pancreatitis with resulting permanent changes in the pancreas. The cause of neither acute nor chronic pancreatitis is known. The most widely accepted theory is that of reflux of bile into the pancreatic duct due to impaction of a gallstone in the ampulla of Vater[14] or to spasm of the sphincter of Oddi.[1]

Other theories include (1) obstruction of the pancreatic duct from pancreatic sphincter spasm, pancreatic-duct calculi, epithelial hyperplasia, adenomas or strictures; (2) apoplexy from vascular closure or spasm; (3) trauma, including operations, and (4) infectious processes from mumps, scarlet fever or gallbladder infections.

Common-Channel Theory.—The basis for the common-channel theory rests on clinical, anatomic and cholangiographic data.

Clinical Data.—Clinical observations of the frequent association of gallstones or disease of the biliary tract with acute and chronic relapsing pancreatitis suggest either a causal relationship between them or that both diseases may be due to a common etiological agent or physiological arrangement. This association is well established.[7] Opie's common-channel theory has been the most broadly accepted explanation of this association.

Anatomic Data.—Dissection studies have revealed a common channel to be present in 4 to 70 per cent of persons. By injecting a test fluid into the common bile duct in fresh specimens after clamping the terminal 3 mm. of the duct, Hjorth[9] demonstrated a common channel in 86 per cent of 100 cases.

Since obstruction of the ampullae by stone with retrojection of the bile into the pancreatic duct was found in only 1.4 per cent of 437 cases by Guleke[8] and 4.4 per cent of 1,278 cases by Schmieden and Sebening,[15] this mechanism failed to explain the majority of the clinical cases.

Cholangiographic Data.—Widespread utilization of cholangiography in humans by Mirizzi[13] and numerous subsequent investigators had adequately demonstrated that sphincter resistance will convert the biliary-pancreatic ductal apparatus into a common channel in humans.

Howell and Bergh,[10] using pressures well within the range of the secretory pressure of the liver and pancreas and about 10 cm. of water above the patient's sphincter resistance, demonstrated the pancreatic duct in 41.5 per cent of cases. Significant was the fact that by repeated examination the pancreatic duct was visualized in fourteen of eighteen cases (77 per cent) in which clinical pancreatitis had been present and in thirteen of forty-seven cases (28 per cent) in which biliary tract disease had been present. Most important in their study, however, was the fact that elevations of serum amylase above control levels and out of the normal range occurred in both

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groups in twenty-five of twenty-seven patients in whom filling of the pancreatic duct was demonstrated. Elevations of serum amylase occurred in only one patient in whom filling of the duct was not demonstrable. Attacks of acute pancreatitis lasting a few hours, three days and five days, respectively, were induced following cholangiography. The clinical diagnosis in these cases was established by the presence of pain and a persistent elevation of serum amylase. The pressures of injection were 30, 29 and 25 cm. of water respectively.

Bergh[2] stated, "We have supposed that such irritability (of the sphincter) is due to an inflammatory lesion (choledochitis secondary to stone)." Choledochostomy for prolonged periods (several months) has usually led to disappearance of the irritability. This sphincter spasm resulting in a common channel has been considered by Mallet and associates[11,12] and by Mirizzi[13] to be due to disease in the biliary tract or pancreas and by most writers,[1,3,5,9,18] to be the cause of biliary-tract and pancreatic disease. Resolution of this difference must await further investigative work.

Surgical Treatment of Chronic Pancreatitis

The surgical attack on pancreatitis based on the common-channel theory has been indirect. In cases with associated gallstones or nonfunctioning gallbladder[4,16] cholecystectomy, choledochostomy or choledochoduodenostomy has usually been performed. Since a considerable number of patients with pancreatitis are cured by these procedures, and to permit thorough investigation of the extrahepatic biliary tract and sphincter of Oddi and determine the presence of a common channel, my associates and I have performed choledochostomy with removal of the gallbladder if it were diseased. The choledochostomies were maintained for six to nine months, and if the sphincter resistance and the cholangiogram were normal, the tube was removed. Among twelve patients so treated one patient suffered recurrent attacks of pancreatitis after removal of the tube; three patients were admitted with recurrent attacks of pancreatitis after treatment elsewhere; in these patients common channels were demonstrated by cholangiogram.

Since in all these patients future health and life were threatened by their disease and because previous therapy had failed, interruption of the common channel between the common bile duct and the duct of Wirsung was decided upon. Division and reimplantation of the duct of Wirsung were selected to avoid the possibility of stricture of the common bile duct with resultant cholangitis. Division of the sphincter choledochus was not performed, as this therapy is under trial. The demonstration of contrast medium in the pancreatic duct in six of fourteen cases by Doubilet and Mulholland[5] also suggested that a more nearly complete diversion would be advantageous.

Four patients at the Minneapolis Veterans Hospital suffering from chronic relapsing pancreatitis have had division of the duct of Wirsung, near the ampulla of Vater; in one patient the proximal end of the duct was ligated, and in three patients it was reimplanted by suture anastomosis to the duodenum distal to the papilla. A fifth patient at the University Hospital has had division of the duct of Wirsung but the pancreatic end could not be located. The end of the duct toward the ampulla was ligated and an omental patch was sutured into the operative defect in the pancreas. An external fistula resulted and he continued to have pain.

Report of Cases

Case 1.—A twenty-eight-year-old man was admitted on September 10, 1948. Closure of a perforated peptic ulcer had been done in 1945. He had noted acute upper abdominal pain on August 8, 1948, after drinking beer. On August 22, exploration resulted in the diagnosis and external drainage of a pancreatic cyst. A wound separation and a "complete" external pancreatic fistula, resulting in hypokalemia, severe dehydration and weight loss, were present on transfer to our hospital. Rehydration, high-protein and high-caloric feedings and adequate electrolyte replacement brought about closure of the fistula, complete wound healing and a weight gain of 35 pounds at the time of discharge 28 days later. On November 16, 1948, he was readmitted with a new attack of pancreatitis and choledochostomy was done. Between this time and December, 1949, he experienced light attacks of pancreatitis, the last two necessitating multiple drainage of pancreatic abscesses. Subtotal gastrectomy in March, 1949, had not helped.

On February 10, 1950, division and ligation of the duct of Wirsung were done at its junction with the common bile duct. This patient has maintained his weight, has had normal stools and is able to work as a mechanic. He has returned on numerous occasions but has had no severe attacks of pancreatitis in the four years since operation. On six occasions elevations of serum amylase have been noted. He probably has low-grade recurrent pancreatitis. He suffers from recurrent insomnia and periods of mild depression, at which time he requests barbiturates and narcotics.

Case 2.—A twenty-three-year-old man was admitted on January 20, 1949. He was a moderate alcoholic. Choledochotomy and cholecystectomy had been done elsewhere in May, 1948, for cholelithiasis. The T-tube was removed in December, 1948, after which epigastric pain with extension to the left lumbar region occurred. He was admitted for study with severe epigastric pain. The serum amylase was 1,236 Somogyi units. After three days of failure of therapy, a mass developed. Exploration showed a large bile-stained edematous pancreas. A bile-filled abscess was present behind the head of the pancreas. Common-duct and retroduodenal abscess drainage was instituted. Nine days later an abscess in the right lower quadrant was drained. The choledochostomy tube was removed on September 30, 1949. He returned on February 19, 1950, with another very severe attack which subsided with use of gastric suction, splanchnic blocks and antibiotics. Since a common channel had been demonstrated by a cholangiogram, division and implantation of the duct of Wirsung was done on April 10, 1950. When last seen on September 30, 1950, he was asymptomatic.

Case 3.—A thirty-seven-year-old man was admitted on December 21, 1948. He was a bartender and a moderate alcoholic. Since June, 1948, he had had repeated attacks of epigastric pain, nausea and vomiting. A large epigastric mass with a pronounced bruit was noted in his epigastrium. Serum amylase studies were normal. On December 28, 1948, he was explored for pancreatitis with pseudocyst formation but only a chronically enlarged pancreas was found. Choledochostomy was done. Cholangiography demonstrated a common channel and caused an exacerbation of pain and increase in serum amylase. The T-tube remained in place one year, during which time he continued to have mild attacks of pain, nausea and vomiting. Four months after removal of the T-tube he entered another hospital with an attack of severe pain. This pain recurred twenty-four hours later and subsided after ten days of conservative therapy. He was transferred to our hospital in April and on May 10, 1950, transection and reimplantation of the duct were done. His postoperative recovery was uneventful. He suffered an attack of epigastric pain eighteen months later accompanied by albuminuria, hematuria and granular casts, with normal serum amylases on two occasions. A deformed spastic duodenal bulb was found on gastrointestinal examination. No definite diagnosis was established, though the presence of glomerulonephritis was likely from analysis of the record.

Case 4.—A thirty-one-year-old man was admitted on May 7, 1950, with a history of numerous attacks of epigastric pain for 2½ years. Exploratory laparotomy and drainage of the pancreas had been done in April, 1949. In August, 1949, splanchnicectomy, vagotomy and subtotal gastrectomy had been performed. The frequency and severity of the attacks were unaffected. On December 1, 1950, transection of the pancreatic duct with reimplantation was performed. One mild attack occurred two months after operation. None have occurred since. When last studied fifteen months after operation he was eating a regular diet, maintaining his weight and had had no attacks of pain. He had soft, formed stools which floated.

Summary

Ligation of the pancreatic duct was performed on one patient who had suffered ten severe attacks of pancreatitis over a period of nineteen months, with pseudocyst formation and complete external fistula on one occasion and pyogenic abscesses on two occasions. Previous drainage of the common duct and subtotal gastrectomy had failed to control the attacks. No severe incapacitating attacks have occurred since the ligation, though several mild ones have. No serious complications or evidences of pancreatic insufficiency have occurred in the subsequent four years.

Transplantation of the duct of Wirsung was performed on three patients, all of whom had symptoms of pancreatitis uncontrolled by other procedures, including choledochostomy in two and splanchnicectomy, vagotomy and subtotal gastrectomy in one. All three patients have had control of the disease process. In one patient, pancreatic insufficiency manifested by large soft floating stools developed.

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Labyrinthine Hydrops

Ménière's Disease

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RECENTLY one of my colleagues said, "It seems to me that I am seeing an increasing number of patients presenting the complaints of dizziness and tinnitus—some with, some without the complaints of hearing impairment." He went on to say that he also felt an inadequacy about the treatment of this problem, especially for those patients with milder recurring symptoms not requiring hospital or bed care and who obviously seem to be in good general health. This led to an exchange of opinion in regard to possible factors that could explain an increase in incidence of this distressing triad of symptoms affecting the peripheral vestibular apparatus, which has long been known as Ménière's disease.

I feel sure that many physicians in specialties other than otolaryngology meet this problem very frequently and perhaps also sense an increase in its incidence. The demonstration of the histopathologic changes in the labyrinth which occur in Ménière's disease by Hallpike and Cairns[11] in 1938 served as a stimulus for greater study of this condition, and as a result the medical literature during the past decade abounds with contributions on the subject. Many studies confirm the histopathologic evidence of an endolymphatic distention of the cochlear duct without inflammatory reaction, with the result that the terms endolymphatic hydrops, hydrops of the labyrinth or labyrinthine dropsy are now commonly used to designate this disease. More and more evidence is accumulating to support the theory that an autonomic dysfunction or an intrinsic physical allergy can be the basic cause of this disorder.

Hilger[14] has shown that the vital intra-labyrinthine structures depend for the integrity of their function and structure upon end-arteries, without collateral circulation. This is true both in the cochlea and the vestibule. Disturbed vascular physiology common to all end-circulation can cause vasospastic changes in the labyrinth resulting in primary neurovascular ischemia and lymphatic hypertension to bring about this syndrome. Hilger claims spasm of branches supplying the cochlear ganglion cells results in tinnitus and hearing impairment in the frequencies involved. The frequent association of these symptoms with vertigo suggests involvement of the vestibulo-cochlear artery. Since extremes of physical and mental environment and psychomotor factors are chief causes of neurovascular disturbances, a modification in the way of our strenuous life and faulty living seems necessary in the prophylaxis and management of labyrinthine dysfunction. By this concept can we also point an accusing finger at the vasospastic effect of smoking as an additional contributing factor in recurrent ischemia of end-circulation? Do these factors then give some support and reason for our initial question of a possible increase in the incidence of peripheral vestibular and cochlear disturbances on a vasomotor basis?

Williams[25] theorizes that Ménière's disease is a form of intrinsic allergy involving the capillary loops of the stria-vascularis and that the endolymphatic distention is a localized extracellular edema produced by the spastic-atonic state of these capillaries.

Day[7] believes that in an occasional case a specific protein allergen may prove to be the causative factor and reports observations on several cases in which the symptoms could be abolished on removal of offending allergens only to recur with their reintroduction to the patient's environment. Day also believes and presents evidence that in labyrinthine hydrops the cochlea is primarily affected and the vestibular disturbances are due to secondary or overflow reactions of the vestibular mechanism. When the endolymphatic distention is limited to the ductus cochlearis, there will be no vestibular symptoms. It is probable then that distention of the utricle and saccule and membranous labyrinth producing vertigo occurs only

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following an overflow of endolymph from the distended cochlear duct. This, according to Day, may occur with the initial disturbance, or it may be delayed for months or for years. When vertigo with nausea and vomiting in violent attacks are the prevailing symptoms, he suggests that they may be so incapacitating that they overshadow and submerge the more important cochlear symptoms of hearing loss and tinnitus which may have preceded vertigo for months or years. Day reviewed case histories of 220 patients which included 175 cases with the typical symptoms of vertigo, deafness, and tinnitus, and forty-five cases diagnosed as Ménière's disease without vertigo. A positive history of allergy was obtained in forty-five of the 175 cases of typical Ménière's and in twenty-three of the forty-five cases without vertigo. Twenty-seven of the 220 had bilateral involvement.

Boies[2] presented a study of the temporal bone and inner ear in a forty-seven-year-old man who suffered a fatal sub-dural hematoma when he fell during an attack of vertigo. A diagnosis had previously been made of Ménière's disease. Histopathologic findings in this case which were unrelated to his fatal illness revealed dilatation of the cochlear duct and degenerative changes of the spiral ganglion in the basal coil of the cochlea. This case had also been studied and reported by Lindsay[18] of University of Chicago.

From these and many other valuable contributions on the subject of peripheral labyrinthine dysfunction we now have available evidence that endolymphatic hydrops exists with or without vertigo. When vertigo is present to complete the syndrome of hearing loss with fluctuating tinnitus in recurring attacks, we recognize the clinical entity of Ménière's disease. From a review by Williams[24] of the original papers of Dr. Prosper Ménière on the subject which bears his name, it is obvious that this early physician and writer had a clear concept of labyrinthine lesions causing deafness, tinnitus, and vertigo with vomiting which previously in his day were considered symptoms of cerebellar or cerebral disease. This study also clarifies a universal misintrepretation, both in textbooks and papers, that Ménière believed hemorrhage into the labyrinth was the chief cause of the group of symptoms he described. Williams has shown that Ménière was intent on demonstrating to his colleagues that vertigo, nausea and vomiting need not necessarily arise from "lesions of the cerebellum or cerebral peduncles," but that when these symptoms are accompanied by deafness, the site of the disease lies in the inner ear.

The first microscopic observations of the temporal bones of persons known to have had severe attacks of vertigo, deafness, and tinnitus during life were described by Hallpike and Cairns[11] in 1938. These findings indicated a hydrops of the labyrinth, and it is reasonable to conclude that the effect of this hydrops on the cochlear duct, utricle, and saccule is responsible for the symptomatology. Because we have definite evidence of the etiology of this hydrops, the term idiopathic has been applied to this disturbance of fluid balance in contradistinction to that which has been found in association with inflammatory disease or vascular accidents of the labyrinth or pressure from central lesions.

Furstenberg[8] in 1934 reported observations suggesting tissue sensitization to the sodium ion as the responsible factor for the labyrinthine hydrops in Ménière's disease. Relief of symptoms was affected when retention of sodium was prevented and its elimination promoted. Investigations by Sheldon and Horton[22] in 1940 gave evidence that an alteration in the permeability of the capillary walls with secondary edema of the middle ear is a likely factor responsible for the hydrops and that histamine is an important agent in affecting capillary permeability.

More recently Lempert[16] and his co-workers have reported on the histologic changes in the inner lining of the membranous walls in the endolymphatic external semicircular canals of six patients with Ménière's disease who had partial labyrinthectomy performed for the arrest of vertigo. They found this inner membranous lining of all specimens to be studded with vesiculated epithelial excrescences suggesting the formation of epithelial fluid collections. They propose the theory that an attack of Ménière's disease is due to the rupture of one or more vesicles with release of a toxic fluid into the lumen of the endolymphatic membranous labyrinth. This is described as a chronic progressive herpetic neuritis. Lempert also postulates thatMénierè's disease may be primarily a disease of the vestibular labyrinth and that the cochlear pathology and symptoms are the sequela of the vestibular disease. They conclude that the cumulative loss of hearing during the course of the disease is due to progressive damage of the organ of Corti by the repeated contamination of the endolymph by this vesicular toxic fluid.

Symptomatology

The triad of symptoms in the usual case of Ménière's disease includes vertigo, tinnitus, and hearing impairment. The most constant symptoms are tinnitus, hearing loss in one ear and usually a sense of blockage of the affected ear. The auditory symptoms may appear at the same time as the vertigo or may precede it. In occasional cases there may be attacks of vertigo for weeks or months before auditory symptoms appear. The hearing loss fluctuates in degree with periods of regression, when the noises are also relieved, until the next cycle occurs. The loss may be progressive with each cycle. In the early stages the loss affects low tones primarily up to about the 4000 frequency. In late stages the entire tone scale is involved. Loss for bone conduction hearing parallels air conduction loss, suggesting a nerve type lesion but the fluctuation in hearing has been described as an indication of conduction rather than perception loss. Lempert,[16] however, claims the permanent loss in hearing must be considered a perceptive impairment. There is usually hypersensitivity to loud sounds, a phenomenon known as recruitment, which is notably absent in deafness due to a central lesion of the eighth nerve.

The tinnitus is usually described as a low pitched roaring, buzzing or seashell noise. It is the most variable symptom of the triad and its cause has not been established.

Vertigo may often be so disturbing that the patient is not aware of hearing loss. The attacks are usually explosive or paroxysmal, without warning and unpredictable in rapidity of onset, severity and duration. It may occur at rest, or even during sleep. Attacks may last from a few seconds to several hours or even days. Repeated cycles may go on for weeks or months with only mild periods of regression; secondary symptoms of nausea and vomiting are simply indicative of the severity of the attacks of vertigo. The patient usually describes his vertigo as a turning sensation in which he feels that he himself is turning or objects about him are whirling. He may have the sensation of being suddenly thrown to one side or in one direction. During intervals between severe acute attacks many patients have a constant feeling of unsteadiness and a vague sense of pressure in one ear or in the head. During these periods of remission the deafness, tinnitus, and hypersensitivity to loud sounds may persist.

In the diagnosis it is important to exclude chronic middle ear disease, lesions of the central nervous system, vascular changes affecting the auditory nerve, and toxic labyrinthitis with perceptive deafness and varying degrees of vertigo. In the latter bilateral involvement is the rule rather than the exception and the hearing loss affects the high frequencies more than the lower tone range. Vertigo in toxic labyrinthitis is often continuous and paroxysmal attacks rarely occur.

Caloric tests of labyrinthine function in Ménière's disease, as a rule, show diminished response or normal reaction from the affected side. Response from the two labyrinths may be equal but the test may often reproduce the vertigo described by the patient. The results of the tests are not diagnostic of Ménière's disease. Crowe[4] found that in 29 per cent of a series of his cases there was no reaction when the labyrinth of the affected side was tested. Day[7] claims a hyperactive labyrinth should never be found in a case of true Ménière's disease.

A word of caution is here in order when we speak so freely of Ménière's disease in association with such a common complaint as vertigo or "dizziness". Let us not narrow our thinking into using this syndrome or complex as the catch-all or mental shelf on which we place all vertiginous disorders just because there may be an associated hearing impairment and perhaps tinnitus; or when the patient has previously had such a diagnosis made either by himself or by another physician and received ineffective treatment for a peripheral vestibular disturbance. We must remember that organic disease of the central vestibular components especially in the brain stem and adjacent structures in the early stages may be manifested only by the classical signs of Ménière's disease. An organic lesion must be suspected especially when the labyrinth does not react to the caloric vestibular test. Functional ear examination may disclose a peripheral vestibular disturbance, such as in Ménière's disease, but more careful history, x-ray studies and vestibular tests, along with complete neurological analysis, may reveal a devastating tumor or cyst of the acoustic nerve in the region of the cerebellopontive angle. Dizziness may also be present in the course of acute or chronic middle ear suppuration with labyrinthitis, cerebellar disease, hypertension and cardiovascular disease, toxic vestibular neuritis, and in many other general systemic infections.

Treatment

Both medical and surgical treatment are in current use. It should be stressed that conservative medical therapy should be instituted as early as possible in all cases suspected of Ménière's disease. If hydrops without vertigo is suspected, early treatment may retard or prevent the onset of vertigo. The longer the symptoms have persisted, the less effective is conservative medical treatment. Day[7] claimed restoration of practical hearing and relief from tinnitus in about one-half of his cases without vertigo when treatment was begun during the first year following onset of symptoms.

These patients are usually victims of tension and anxiety states. The physician must express utmost sympathetic understanding of their complaints. All deserve a careful complete physical examination, including a neurological study when any doubt exists as to the cause of the vertigo. They should be told that recurrences in symptoms are to be expected, and they must be encouraged to live a more quiet and regular life. Mild cases are benefited and often controlled by use of sedatives. Williams[25] stresses treatment of the anxiety state by discussion, explanation and relaxing physical therapy.

On the basis of vasomotor phenomenon with increase of capillary permeability resulting in disturbed water metabolism, Mygind and Dedering[19,25] recommended a low salt diet, limitation of fluids to 700 cc. daily or to a point of conscious thirst. Salt restriction promoted diuresis. They also advised laxative salts, a reducing diet for overweight patients, and a high caloric diet for the underweight. Physiotherapy was given to improve vasomotor function.

Furstenberg[8] and co-workers have recommended a sodium-free diet along with ammonium chloride in dosage of 6 to 9 gm. daily for alternating three-day periods. The course of treatment lasts six weeks. A high potassium diet has been used by Talbot and Brown[23] with favorable results. They used 6 to 10 gm. of potassium chloride in solution daily.

The use of histamine by intravenous injection has been recommended by Sheldon and Horton[22] for relief of an acute attack of vertigo. Lillie, Horton and Thornell[17] reported improvement in hearing and relief from vertigo by intravenous histamine injections daily for three to six days and sometimes longer. They have used 2.75 mg. of histamine acid phosphate in 250 cc. physiological saline solution or in 0.8 per cent potassium chloride solution. The fluid is given at a rate of 20 to 60 drops per minute over a period of about one and one-half to two hours.

The use of nicotinic acid has been recommended by Atkinson,[1] Williams[25] and others. The action is attributed to the vasodilator effect of the drug rather than that of a vitamin. Atkinson[1] claims that one group of cases responds to histamine desensitization and one group to nicotinic acid, distinguishable by an intradermal histamine test. For the nicotinic acid group he gives daily intramuscular injections of 10 to 50 mg. or oral administration of 50 to 150 mg. per day for several months. Williams[25] advocates vasodilators alone for those cases of Ménière's disease without endolymphatic hydrops (recurring attacks of vertigo without tinnitus or deafness) on the supposition that vasomotor labyrinthine ischemia alone is present. He uses nicotinate (Nicamin) subcutaneously and nicotinic acid or Roniacol orally. When symptoms of hydrops (deafness and tinnitus) are present along with vertigo, he combines the effect of vasodilators to correct the arteriolar spasm with a low sodium diet for elimination of fluid collection in the endolymphatic system. He allows 0.5 gm. of sodium per twenty-four hours. A modified Schemm diet seems to meet his requirements. This is not only a low sodium but an acid to neutral-ash diet. A moderate restriction of fluid to a total intake of 3,000 cc. is advised.

Several drugs have been used for relief of or preventive effect on vertigo. These include adrenalin, atropine, pilocarpine, estrogens and thyroid therapy, diethylstilbesterol, Banthine as ganglion blocking agent on the parasympathetics, iodides, papaverine, and tetra-ethyl ammonium chloride (Etomon) for its blocking action of autonomic impulses at the synapses in both sympathetic and parasympathetic ganglia. The latter may produce severe postural hypotension, and the drug must be given with caution in patients with hypertension.

On the basis of Hilger's[13] hypothesis that Ménière's disease is due to autonomic labyrinthine ischemia and the production of a neurovascular spastic state in the vascular bed of the labyrinth, he directs the therapy at correction of the vasospasm and the resorption of transudates. He believes this is best accomplished in an acute vasospastic crisis by a neural block rather than generalized vasodilatation alone, and the most effective method of increasing local blood supply is sympathetic denervation of the part. He proposes a combination of a neural blocking agent and a peripheral vasodilator that would increase the heart action and total blood volume as the most ideal and effective approach. For this he uses an infusion of 0.1 or 0.2 per cent procaine hydrochloride solution in 5 per cent glucose. To 1 liter of this solution he adds 0.1 mg. of adrenalin hydrochloride and 1 gm. of ascorbic acid. This is given intravenously in daily treatments of 250 to 500 cc. over a period of one and one-half to two hours. Hilger considers the beneficial effect of this therapy is due to a continuous blockade of the cervical sympathetic ganglia. No evidence of toxicity or sensitivity has been reported by him. In the writer's experience this treatment has produced favorable results in reducing the severe acute vertiginous attacks as well as restoring some degree of hearing loss in many cases presenting this syndrome.

Because of the known toxicity of streptomycin on the vestibular end-organ, this medication has been used by intramuscular injections to destroy the labyrinthine function in selected cases of severe and disabling vertigo. Hanson[12] reported good results in eleven cases and offers the hypothesis that there is a selective neurotoxic action of streptomycin on the altered (diseased) labyrinth or on the vestibular pathways in endolymphatic hydrops. He has used injections of 2 gm. daily over a period of three to five weeks and with this dosage found no significant effects on cochlear (hearing) function. Glorig[9] reported 31 per cent of patients with tuberculosis showing hearing loss when treated with dihydrostreptomycin in doses of 2 to 3 gm. daily for three months or more. It is obvious that purposeful streptomycin destruction of the labyrinth should be limited to well-selected cases which have had careful otologic and neurologic study and evaluation.

Adequate rest and regularity in habits of work and activity must be stressed in any plan of therapy. Psychotherapy and various forms of physiotherapy are of prime importance, for in many cases the onset of attacks can be traced to emotional upsets or mental and physical fatigue.

If conservative medical therapy fails to control the attacks of severe vertigo in Ménierè's disease, surgical intervention is indicated. Hearing is not improved by surgery and its effect on tinnitus is variable. Surgery should be limited to the case of unilateral involvement with severe hearing impairment. Day[5,6,7] advocates surgery when the attacks of vertigo occur without warning and are so severe that the patient falls when engaged in an occupation where a fall might endanger his life or if he lives in constant fear of impending severe attacks. Two main types of operative procedures have been advocated.

1. Partial or complete section of the eighth cranial nerve, as described by Dandy, is still practiced by some neurosurgeons. Partial section theoretically is the ideal procedure in that the remaining hearing is preserved to some degree in about 40 per cent in several reported series by Dandy and co-workers.[10] This is a major neurosurgical performance attended by some risk of intracranial complications. Tinnitus has been relieved in only about 50 per cent of cases undergoing this treatment. Day maintains that if partial section be performed with preservation of hearing, there is likely to remain a preservation also of some vertigo.

2. Several procedures attack the membranous labyrinth directly. Portmann[21] describes a destructive operation on the endolymphatic sac. Recurrence of symptoms following this procedure is common. The operation in general favor today is one employing an opening into the labyrinth by way of a mastoid approach to destroy the labyrinthine end-organ. Cawthorne[3] removes a piece of the membranous external semicircular canal or merely makes a tear in it. Day[5,6,7] opens the external semicircular canal and coagulates the vestibular contents with a weak diathermy coagulating current. Other operators have destroyed the contents by inserting a barbed dental hook into the vestibule. This is a relatively simple procedure in which there is a possibility of preserving some degree of the residual hearing. At the present time Day no longer attempts or advocates differential electrocoagulation because of his poor results when hearing was preserved and because of added possibility of incomplete destruction of the vestibular mechanism. The period of hospitalization and rehabilitation is much less with the Day operation than with the more difficult operation of total or partial nerve section.

Recently Passe and Seymour[20] have reported favorable results with a procedure consisting of removal of the stellate ganglion on the affected side, division of T_1 and T_2 and stripping or section of the vertebral artery.

Lempert[15] has also recently described a decompression of the hydrops of the endolymphatic labyrinth with degeneration of the labyrinth and organ of Corti by removal of the stapes and round window membrane.

These intralabyrinthine surgical procedures in their present stage provide relief from severe vertigo in Ménière's disease, and although they give a poor prospect for preservation of hearing, they are meeting with preference because of relative simplicity and safety from complications.

Summary

1. Abundant reports in recent years on the study of Ménière's disease reveal an almost universal conception that this common disorder of vertigo, hearing loss, and tinnitus is probably caused by an autonomic nervous system dysfunction or intrinsic allergy, producing a neurovascular spastic state of the labyrinthine circulation and resulting in endolymphatic distention or hypertension.

2. Intrinsic emotional stress related to the extremes of physical and mental environment in our strenuous life may be the chief factor in precipitating neurovascular disturbances of the labyrinth. Heredity and physical factors also have great significance. A specific protein allergen may prove to be causative factor.

3. Hydrops of the endolymphatic system may exist with or without vertigo. The effect on the cochlea with hearing loss alone may be the primary disturbance, and it may precede any disturbance of the vestibular mechanism by a period of months or years.

4. Conservative medical treatment can relieve and control the acute vertiginous episodes in the majority of cases of Ménière's disease. When therapy is instituted early, a fair prognosis in restoration of hearing loss can be anticipated. Recurrences must be expected, and the patient should be encouraged to live a more regular life with emphasis on the avoidance of stress and tension states. A sympathetic understanding of symptoms by the physician and psychotherapy play an important role in the regulation of emotional stress.

5. The possibility of an organic central nervous system lesion must always be considered when vertigo is of long standing and persists with progressive loss of hearing or when proper medical treatment fails to bring about remission of symptoms.

6. Surgical destruction of the membranous labyrinth by several methods of labyrinthotomy provides relief of severe vertigo in unilateral Ménière's disease and appears to be the treatment of choice when proper medical management has failed and hearing function is severely impaired.

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Visual Problems of Patients Who Have Unilateral Cataract Operations

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OPHTHALMOLOGISTS are often called upon to attend patients who must either consider removal, or actually undergo removal, of a cataract from only one eye, while the other eye is still capable of considerable vision. The purpose of this article is to inform doctors who do not work regularly with optical problems, about the perplexing visual difficulties of these people.

Removal of the lens from an eye radically changes its optical mechanism.[4] In the first place the eye is made very far sighted. To restore good vision after operation, in almost every case a lens of high plus power, in the range of plus 9. to plus 12. diopters must be prescribed, along with a variable amount of cylinder to correct the astigmatism resulting from the healed incision. A lens of this power is unavoidably heavy and the spectacle frames are easily bent out of adjustment. A distorted image results when the wearer looks elsewhere than exactly through the center of such a lens. In addition, the periphery of such a lens creates so marked a prismatic effect that the wearer has a ring scotoma of considerable width.

The power of accommodation is of course lost from an aphakic eye. Another important effect is the magnification of the image seen with such an eye, an image in most cases 25 to 30 per cent larger than that seen with a normal eye. The amount of aniseikonia (unequal image size) varies, depending on the distance in front of the cornea at which the glasses are worn. This large imagery is the result of the removal of the lens from within the globe and the substitution in its place of a strong plus lens in spectacles.

As may be imagined, the reactions of various patients to such a situation differ widely.

If the vision in the lensless eye is good and that in the untouched eye definitely down, the shift over to the aphakic vision will be made more or less smoothly in the course of some weeks—in general, the older the patient, the slower and more discouraging the period of relearning. If the normal eye still sees well, most patients, both young and old, will choose to see with it and will ignore, or try to ignore, the image seen with the aphakic eye. In a child this usually amounts to prompt suppression. In an older person the unnatural image is likely to be troublesome. To avoid it, the patient may close his aphakic eye, wear an occluding device such as a rippled lens, or even a patch. It may be sufficient merely to let the eye go uncorrected. In rare cases a patient is able to tolerate the marked inequality of image size and seems happy with full correction for each eye. However, on careful questioning it will usually be found that he is suppressing one of the images for the everyday, ordinary conditions of seeing.[7]

This inequality of image size may be cut down to about 10 per cent by providing the correction for the aphakic eye as a contact lens.[2,6,11] Some aphakic patients can tolerate a contact lens for a surprising number of hours. This is a clever and often quite satisfactory solution for the problem in patients who want binocular vision. The eye so corrected, however, is still unable to accommodate, and for near vision a spectacle lens with a bifocal segment is necessary in addition to the contact lens.

The best description the writer knows of, concerning aphakic vision, is in an editorial by an anonymous ophthalmologist in the *American Journal of Ophthalmology* for January, 1952.[1] In it are colorfully described the surprising magnification of images, the disturbed ability to judge distances, the field limitation and the distortion: bulging door frames, weaving pillars and walls, and so on.

Readers of this article who want to experiment with unequal image size can easily do so with the lenses in an ordinary trial case. This may be done with glasses on, or if no glasses are worn, with the trial frame.

If a trial frame is used, the cells in front of the right eye are left empty. A minus 20. D lens is put in the left back cell, or better yet the lens is held very close to the left eye. This artificially makes the left eye very far sighted. Now a plus 16. D lens is put in the farthest front cell on the left side. If glasses are worn, the same two lenses can be used, the minus lens held behind the glasses very close to the eye, the plus lens in front of the left spectacle lens. The vision of the left eye will clear, but at once it will be noted that the left eye is seeing images distinctly larger than those seen by the right eye. This inequality is of the order of about 10 per cent, and fusion of such images is readily done by many people. If more attention now is paid to the image seen with the left eye, considerable peripheral distortion will also be noted.

This arrangement of lenses is merely a Galilean system. The writer does not mean to imply that this experiment makes the phakic eye see like an aphakic eye. It does not. But it does provide an easy demonstration of rather marked aniseikonia, and of the distortion suffered in looking through the peripheral part of a thick lens.

It now becomes easier to understand the complaints of a senescent patient who is suddenly confronted with a new kind of vision through glasses two or three times as thick as any he ever wore before.[3,9] Furthermore, it is no wonder the problem is magnified when an attempt is made to team up this new vision with that of a phakic eye. If the ordinary eye affords even partly satisfactory vision, the patient will often choose to disregard the function of the lensless eye as best he can, even though the aphakic image, on Snellen testing, may be considerably sharper than he can possibly get with the other eye. Anticipating such difficulties, an eye surgeon will often decide against operating upon a unilateral cataract, but will send the patient home to wait until the vision in the better eye is such that there can be no argument about operating. It is no longer wholly a question of waiting until cataracts are ripe. It fact removal of mature cataracts has certain disadvantages that will be mentioned. But often a cataract may remain in an eye for years without causing harm, and until the better eye is reduced to correctible acuity of 20/60 or even worse, the eye surgeon may prudently advise delay.

There are certain risks to be considered in such delay. Some eye surgeons state that an eye with a mature cataract in it should have surgery if the patient is physically able.[8] The principal reasons for this opinion are:

1. One never knows just what is going on behind a cataract (possible tumor).
2. As time goes on a cataractous lens tends to grow firmer and its removal intracapsularly is more difficult.
3. Occasionally a cataractous lens may enlarge enough to cause secondary glaucoma, or breakdown products from the degenerating lens tissue may cause an irritable eye or even a disastrous inflammation.
4. Divergent strabismus of the non-seeing eye often occurs.

The first three of these complications are not frequent. Ophthalmologists have all watched, for years, numbers of aged patients with cataracts in one eye, or cataract more advanced in one eye than in the other. For one reason or another surgery may be refused or postponed, and many such old people will live out their life spans without having to submit to intraocular surgery at all. Such patients should be seen periodically to watch for complications. A cataractous eye will not become amblyopic except in a child. It may be argued that it is probably safer for a patient to undergo surgery earlier in life if he must eventually have it anyway. But it is not certain he will have to have it eventually, and the early ambulation now used after eye surgery, together with the almost universal employment of corneoscleral sutures, makes this argument less compelling.

A patient in young adult life or middle age has a different problem. The likelihood of divergent strabismus developing needs serious thought. Younger patients often need the peripheral vision on the side of the cataractous eye, or a man may have a job for which binocular vision is important. Here the cataract may be removed, and if the patient wears no aphakic correction at all, he at least gets a field on that side, though badly blurred. He may try the full correction for each eye—rarely is a patient happy with this. Or the scheme of the contact lens for the aphakic eye may be used.

The Ridley operation for cataract consists of extracapsular extraction with insertion of an acrylic lens into the eye to take the place, both optically and actually, of the original lens. It was conceived and developed to avoid the visual difficulties that patients have after ordinary cataract surgery. It is too early for the eventual fate of this operation to be known. However, many eye surgeons are voicing unfavorable opinions about it, the reason being that a large proportion of cases react to the presence of the foreign material within the eye.[5]

Lastly, unilateral cataract may be seen in a child, either from a congenital cause or as a result of any eye injury. Here the problem of amblyopia is added to the already complex situation. If the child is an infant, the amblyopia will be one of arrest; that is, the vision is not having a chance to develop and it will not subsequently develop, unless a reasonably clear image be provided for the macula by the time the patient is from one and a half to two years old. Surgery for congenital cataracts sounds simple—needling the lens and letting it absorb. But the operation has hazards and more than the procedure itself must be considered. Many infant eyes with congenital cataracts present other defects. If surgery is successful, the strong aphakic correction must be provided for the tiny patient if vision is to develop. The proportion of surgical failures is considerable, and in later years retinal detachment may occur, nullifying everything.[10] Therefore if the other eye is normal the surgeon may advise against operation.

An older child who develops a cataract after the years of his infancy may also get an amblyopia in this eye—but it will be an amblyopia of extinction, and vision can be regained if loss of the other eye should make it necessary. The cataract can be removed by needling, or by linear extraction, and the eye will probably not be used, even if corrected, unless vision in the fellow eye is poor. A contact lens for the aphakic eye would seem ideal, but Goar states he has never seen a patient under eleven years of age who would tolerate one.[8]

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TRANSPLANTATION OF DUCT OF WIRSUNG IN CHRONIC PANCREATITIS

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Giant Condyloma (Verruca) Simulating Carcinoma of the Penis

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GENITAL WARTS (condylomata acuminata, verruca acuminata) are to be found in both sexes. Condylomata are elevated, circumscribed, papillary masses that occur most commonly on moist cutaneous and mucocutaneous surfaces. They are to be seen on the genital and anal regions where the surfaces are moist and exposed to friction. Irritating discharges and secretions create an improved medium for their growth. Both the appearance, and shape of the verrucae appear to be influenced by their location as well as by the trauma that occurs associated with the location. The verrucae are usually elevated, circumscribed, papillary masses. They may resemble great masses of granulation tissue. On the male genitals, especially around the corona, one sees the more filiform type of lesion. The lesions may be isolated excrescences, or may be in small groups or even in large verrucous masses which mask normal structures. The appearance on the female genitals may be that of an enormous granulation tissue with cauliflower-like appearance. On the female genitals, the involvement is on the labia with some concentration around the entroitus. Condylomata may also occur about the face, around the mouth, the axillae, and in the periumbilical region. Extension as well as growth is limited principally to the warm, moist regions.

Like all types of warts, condylomata acuminata are autoinoculable and transmissible. The transmissibility was demonstrated long ago by Jadassohn and others. Rulison in a statistical study emphasized the higher incidence in certain families.[11] It has been shown that some patients with condylomata acuminata on the genitals have verrucae vulgaris on their hands. Recently Barret et al emphasized the reclassification of genital warts as venereal warts. They pointed out the ease and frequency of transmission from one individual to another through intercourse. Rhodes reported experimental transfer of human wart material to a monkey's prepuce.

As far back as 1919, Wile and Kingery discussed the etiology of common warts.[16] Kingery later was able to take material and reinoculate warts from the second generation in order to point out that the virus was viable and transmissible. Goodman and Greenwood[6] were able to produce verrucae vulgaris and verrucae plana from material taken from condylomata acuminata and filiform warts, thus leading to the feeling that all verrucae have a common etiology. Verrucae plana have also been demonstrated from material taken from laryngeal papillomata. The contagiousness of warts and condylomata has been definitely established by observation of accidental transmission as well as by experimental transmission carried out through inoculation of finely ground material. Wilson suggested the term genital warts rather than the term condylomata acuminata and felt that it was a better and more descriptive term.[17] He noted that the growth of condylomata increased considerably during pregnancy possibly due to the stimulating action of estrogen. Attempts have been made to incubate the verruca virus on rabbit and mouse brain but they have been unproductive of any results. On the other hand, inoculation of material in rabbit testicle produced inclusion-like bodies (eggs later inoculated from testicular material were negative). Wilson unsuccessfully attempted to inoculate chorioallantoic membrane. Felsher[3] also inoculated chorioallantoic membrane of the chick embryo with material derived from verrucae vulgaris and was unable to demonstrate any satisfactory growth. It was his opinion that no definite conclusions could be drawn concerning the possible

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From the Department of Dermatology, University of Minnesota. Henry E. Michelson, Director.

Presented at the Annual Meeting of the Southern Minnesota Medical Association, Winona, Minnesota, September 13, 1954.

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virus etiology of skin diseases by negative results in the inoculation of the chorioallantoic membrane. He felt that the chorioallantoic membrane in seven to fourteen-day-old chick embryo was unsatisfactory material for culturing dermatotropic viruses. Recently, Bivins was able to inoculate material obtained from verrucae vulgaris into chorioallantoic membrane.[2] It was reported and illustrated with material that has been carried through a Berkefeld filter producing massive proliferation and "pearl" formation on chorioallantoic membrane.

Much work has been done in demonstration of the inclusion bodies as found in warts. Lyell et al were able to demonstrate eosinophilic intranuclear and intracytoplasmic bodies in the prickle cell layers of some warts. The inclusion bodies varied considerable in size and shape. Not all of the prickle cells were involved in the process.

Blank reviewed the virus etiology of verrucae.[1] He agrees with Lipschultz that the inclusions are basophilic and intranuclear. Blank examined about 150 verrucae from sixty-five patients, six of whom had condylomata acuminata. He concluded that pathognomonic changes occur in the nucleus in such a manner that basophilic material eventually fills and distends the nuclear membrane. About half of all verrucae examined contained cells with the inclusion material. The material contained large amounts of nuclear protein, most of which appears to be desoxyribose in form.

Strauss demonstrated spherical virus-like particles which tended to aggregate in regular nests in papillomatous growths of human skin.[14] They all had a similar histologic appearance. They were intranuclear inclusion bodies with solid or vacuolated masses in the cytoplasm of the rete cells. The growths seemed to be the same in verrucae vulgaris as in verrucae plantaris. This crystalline material that was demonstrated appeared to represent the first crystalline material isolated from human growths.

The above listed information helps link condylomata with verrucae vulgaris and makes for a common demonstrable virus etiology. The different behavior of the virus at the various sites is not so readily explained, particularly when the growth becomes extensive, bizarre in character, or destructive. Usually when condylomata acuminata are seen in the male, they occur on the inner surface of the prepuce along the coronal margin or on the glans penis. The prepuce, with the accumulation of moisture and secretions under it, predisposes to the formation and localization of genital warts. They are small and usually remain confined to the named area. There is, however, one uncommon form of giant condylomata which grows extensively along the prepuce, sometimes perforating and destroying this structure. They may also grow over the glans, destroying it, producing distortion of the penis and in that manner giving the entire growth the appearance of a malignant neoplasm. The lesion is malignant locally by virture of the destruction it produces. Microscopically it appears to be benign. The uncontrolled growth and destruction produces ulceration, involves normal structures and frequently fosters secondary purulent infection. This infection adds to the foul odor produced by the growth.

The giant destructive condylomata were originally described by Buschke. Later they were discussed more extensively by Buschke and Loewenstein, both of whose names are now linked with this type of tumor.[9] Loewenstein made the first report in the American literature in 1939 when he reviewed the literature and described a case. In his description of the histology the stratum germinativum and spinosum remain clearly demarcated and show no evidence of any malignant change in spite of all the marked proliferation. This indicated definitely a benign lesion that for some reason became markedly proliferative. There is a tendency to infiltration deeper into the tissue layers of the penis. There is no sign of malignancy aside from this excessive growth. There were mitoses to be seen in epithelial layers, but there were no glandular metastases. It was Loewenstein's opinion that the growths developed in a preputial cavity which was constrictive and that the break occurred into the prepuce and then through it, perforating the structure.

Gersh reported two patients with similar destructive lesions.[4,5] In both cases the lesions occurred in individuals who had a phimotic prepuce. It was Gersh s conclusion that early complete removal with circumcision should obviate the necessity of radical procedures later on. Numerous writers have raised the question as to whether these condylomata should be considered pre-

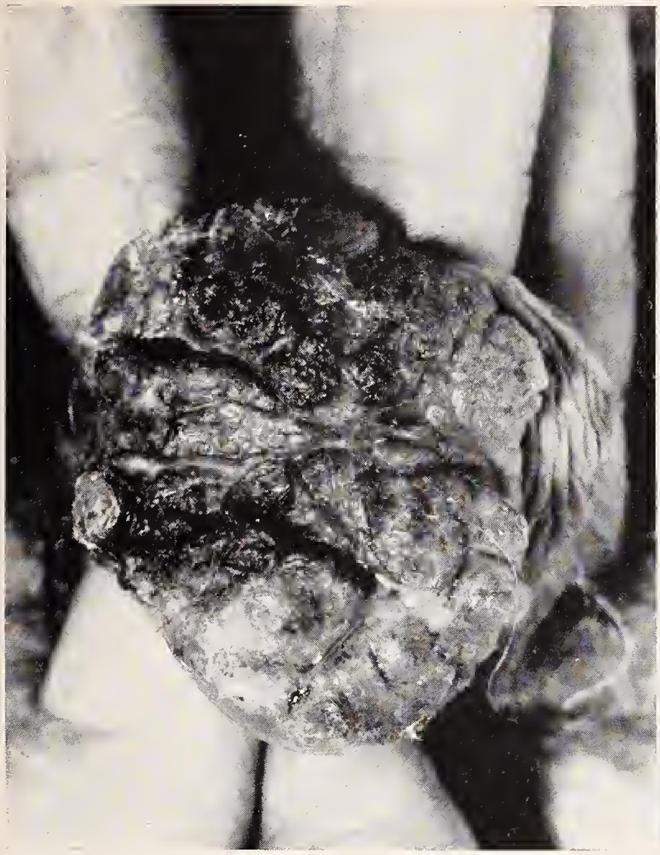

Fig. 1. Case 1, E. D. B. Large, vegetative verruca involving and destroying the glans penis. Biopsy specimen revealed benign condylomata.

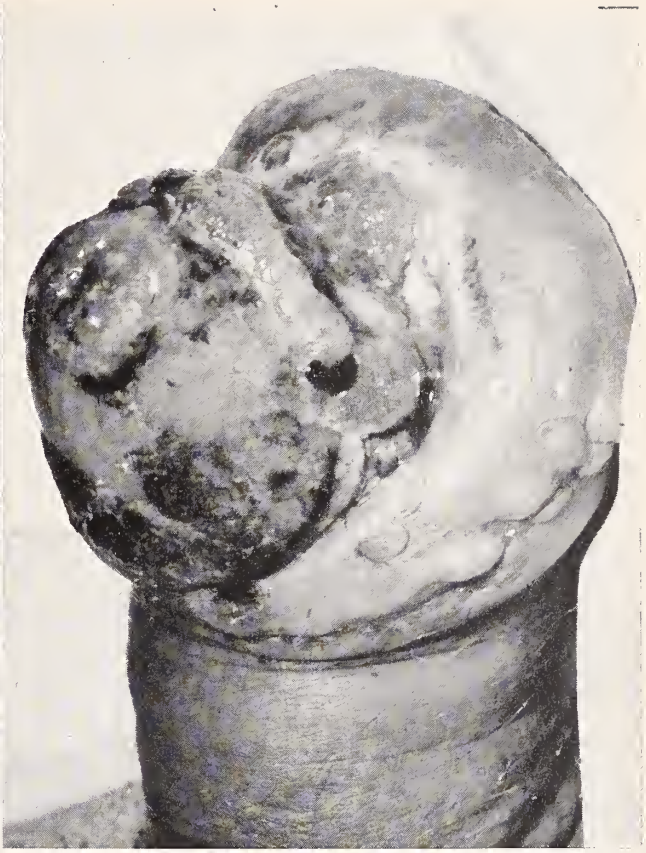

Fig. 2. Case 2, T. A. Verrucous growth seen on the glans, at the corona and involving the prepuce. Shown after drainage of abscesses and dorsal slit.

cancerous and the suggestion has been made that repeated biopsies be carried out in order to rule out this possibility. The therapy would hinge on the accurate diagnosis.

Various forms of therapy have been suggested for condylomata acuminata and particularly the ones of this type. In 1942 Kaplan[8] described the use of the resin of podophyllin in the destruction of acuminate warts. His original treatment has since been modified somewhat so that the tincture is used instead of a suspension of podophyllin in liquid petrolatum. The effectiveness of the drug has been explained by its colchicine-like action on the cell nuclei. All forms of therapy whether chemical, thermal or surgical are directed at destruction. Gersh urged surgical removal of the lesions accompanied by circumcision in order to eliminate the possible development of the giant lesions.[4,5] When the lesions are large and are destructive, partial or complete penile amputation becomes necessary.

It appears to be Gersh's conviction that the presence of a prepuce and the accompanying moisture beneath it, predisposes to the development as well as the recurrence of condylomata acuminata. For that reason he believes circumcision would prevent recurrence or the development of giant destructive condylomata. In reviewing his results of therapy with podophyllin in tincture of benzoin, Barrett reported twenty-eight patients who were retreated with recurrent verrucae from one to three times before complete clearing occurred. These patients were not circumcised. Thirteen other patients were treated with 20 per cent podophyllin in tincture of benzoin, followed by circumcision. There were no recurrences in this group.

The following cases are presented to illustrate further the questions concerning the proper approach from the standpoint of therapy and accurate diagnosis. The cases also demonstrate that the destruction may progress over a long period of time with tissue loss but without a threat to the individual's life expectancy.

Case Reports

Case 1.—E. D. B., a white man, aged fifty, first noted a small papule on the coronal sulcus in 1937. The papule grew, became painful and spread to the surrounding area on the glans penis. In 1938 he was circumcised and the warts were desiccated. Desiccation was carried out at irregular intervals from 1938 to 1942. At the time he enlisted in the Navy in October, 1942, the penis looked normal except for a small area of discoloration where warts had formerly been. Patient was discharged in October, 1945. A small asymptomatic lump had developed at that time. In 1946 new warts developed and a biopsy was taken at the University of Minnesota. Since then the patient had repeated desiccation of lesions followed with podophyllin tincture applications at irregular intervals. The patient complained of a great deal of pain from podophyllin and the desiccation. From 1946 to February, 1952, he received the treatment at intervals. With the year 1953 the lesion had again grown to larger proportions. A biopsy specimen was taken in July, 1953.

Report of biopsy tissue taken from glans penis: *Gross:* The specimen is a triangular piece of tissue 8 by 7 by 7 mm. and approximately 3 mm. in thickness. It appears to be necrotic and is mottled red and brown in color. *Microscopic:* There is hyperkeratosis and parakeratosis to a marked degree. There are numerous granular basophilic areas that appear to be calcium deposit. There is a thin band of lymphocytes present. There is a thickened squamous epithelium that has blunt sharply demarcated lower surface. There is some acanthosis of the adjacent epidermis. There is no evidence of malignancy present. *Diagnosis:* Benign condylomata of the glans penis.

The patient was admitted to the United States Veterans Hospital on July 9, 1953. His chief complaint was shortness of breath for six weeks. There was a past history of rheumatic fever in 1912 at age 10. He had been hospitalized for heart failure in February, 1952 and March, 1953. The verrucous lesion had grown and spread to the surrounding area on the head of the penis (Fig. 1). It was quite painful.

E. D. B. was readmitted for the last time on September 21, 1953. He was comatose at that time and expired on September 28, 1953. At the time of death there was a large cauliflower-like vegatative lesion involving the ventral surface of the glans penis and measured 3 by 3 by 1 cm. Autopsy examination revealed no evidence of carcinoma or metastases.

Case 2.—T. A., a forty-two-year-old white man, first consulted a physician on July 1, 1949, concerning a wart which appeared on the glans penis. Because of the possibility of cancer, the wart was excised widely. The pathological report on the excised material was that of chronic inflammatory tissue. Since that time the lesions had grown considerably. The patient had no other symptoms except some increasing fatigue and weakness during the past two months. The glans penis was markedly enlarged with a fungating growth which appeared localized in its greatert portion to the underside of the glans penis. The end of the penis was enlarged about three times normal size. There were no areas of frank ulceration. A great deal of secondary infection was present. An urethral fistula was present just behind the frenulum which appeared through the center of the fungating growth, on the ventral portion of the penis. A few shotty nodes were present bilaterally in the inguinal region but these were freely movable and none were larger than pea size.

On admission the clinical diagnosis of carcinoma of the penis was made. A punch biopsy was performed September 1, 1949, and reported as consistent with the diagnosis of chronic inflammation. Because there was some question of the type and source of this biopsy, on September 4, 1949, the patient was taken to surgery, and two sections were excised from the edge of the lesion near the glans penis. Both of these sections showed no carcinoma to be present and were consistent with a diagnosis of chronic inflammatory tissue and benign papilloma. At the time of surgery approximately 30 cc. of pus was aspirated from one abscess cavity on the ventral portion of the shaft of the penis and multiple fistulae were noted all through the glans. These cavities were opened and the fistulous tracts probed and drained (Fig. 2). Under treatment the swelling and edema of the glans gradually subsided. However, another area of the fluctuation developed near the glans. This was opened and drained within the next few days. Biopsy specimen of the lesion was obtained and reported.

Report of biopsy specimen from penis, August 4, 1949: This portion of tumor shows only the appearance of a benign papilloma. There is no definite evidence of malignancy from the sections. *Diagnosis:* Papilloma.

On August 16, 1949, multiple abscesses about the glans were treated and the superficial papillomas were fulgurated. He received a total of 3100 roentgens to the penis from August 12 to September 12, 1949. The lesions seem to regress somewhat under the irradiation therapy. On September 16, 1949, an external meatotomy was done. A new urinary fistula developed on the ventral lateral surface of the glans penis. On November 3, 1949, a redundant portion was excised down to the urethra ventrally. In December, 1949, following this he was given a course of bismuth subsalicylate with no appreciable effect in the course of the lesions.

Report of tissue removed from penis with biopsy punch, January 10, 1950: Examination of the portion reveals it to be covered by a stratified squamous epithelium which shows a marked acanthosis and a papillary irregularity of the outer surface. Examination of the epithelium reveals considerable dyskeratosis. No good evidence of invasion is seen but the dyskeratotis change in the epithelium suggests that this is possibly changing into a malignant process. This is not a convincing carcinoma but certainly represents a precancerous growth. *Diagnosis:* Papilloma of penis showing questionable malignant change.

Penis was amputated on February 2, 1950. Lesion healed well. The patient is living and well to date.

Discussion

Two cases of benign destructive giant condylomata have been presented. In one case a dorsal slit was performed during the course of treatment without any change in the progress of the lesion. The other case persisted and recurred over a period of sixteen years after the first surgical removal accompanied by circumcision. At one time there were only small pigmented scars and no warts. Recurrence developed nevertheless. It is the writer's opinion that persistence and recurrence of this type of wart is similar to that seen in other types of warts. Some patients have a peculiar susceptibility to warts and respond poorly to all forms of therapy. In other patients almost any form of therapy will be effective. These are the factors which confuse the evaluation of treatment results in patients with warts.

The giant (carcinoma-like) condyloma is a special and unusual lesion. Good hygiene, circumcision and early therapy will help prevent their development. However, individual patient susceptibility may play a role in the development of such a lesion.

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PRESENT STATUS OF MULTIPLE SCLEROSIS

Forty of the world's leading research investigators in the field of multiple sclerosis met in the Conrad Hilton Hotel, Chicago, on June 12, for the Eighteenth Session of the Medical Advisory Board Meeting of the National Multiple Sclerosis Society.

International guests of the Medical Advisory Board were Dr. Douglas McAlpine of the Middlesex Hospital, London, England, and Dr. R. S. Allison of the Royal Victoria Hospital, Belfast, Ireland. They addressed the meeting, describing the multiple sclerosis problem in their respective countries.

Seventeen research applications for financial support from the National Multiple Sclerosis Society were reviewed; ten of these represented proposed projects, the remaining seven being applications from investigators already being sponsored by the Society.

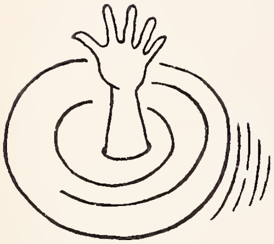
Fifteen currently sponsored investigations were discussed and analyzed by the medical board. Among them were the report of Dr. Harry M. Zimmerman, of the Montefiore Hospital, New York City, who has established a Central Pathology Registry in Multiple Sclerosis. The studies presently being conducted at the Mount Sinai Hospital in New York City under the direction of Dr. Harry Sobotka were also reviewed. They seek to learn what role, if any, vitamin substances play in the cerebrospinal fluid, and whether such changes, if they exist, can be related to a multiple sclerosis patient's problems.

Dr. Hans H. Reese, Professor of Neuropsychiatry, University of Wisconsin Medical School, and Chairman of the Medical Advisory Board, and Dr. Augustus S. Rose, Professor of Medicine in Neurology, University of California, Chairman-elect, presided. A summary report was delivered by Dr. Harold R. Wainerdi, Medical Director of the National Multiple Sclerosis Society.

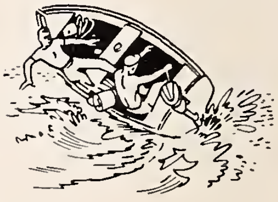
The Society has supported forty-eight research projects, including five research clinics, at a total cost of $792,414, during the nine years since its founding. During that time, an average of 41 per cent of all funds has been spent on research programs. The only national voluntary health agency dedicated to combating multiple sclerosis and allied neurological diseases through its programs of research, rehabilitation, patient service and clinics, the Society is presently seeking a minimum of $2,000,000 to meet these needs.

Water Safety for Minnesotans

JOHN R. HARTMANN
Minneapolis, Minnesota



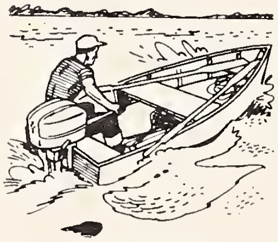
Of 6,700 drownings in the United States in 1954, there were 111 in Minnesota. In 1953, Minnesota numbered 156 drownings. Ninety million Americans use our swimming facilities throughout the United States. It has been conservatively estimated that the above number of people swim 2,000,000,000 times per year.



Avoid sharp, sudden turns at high speeds, and save yourself a ducking!

The Problem

If statistics are correct, 90 per cent of the millions who use our water facilities for recreation, or other means, swim very poorly or not at all; 10 per cent swim very well or reasonably well, and only a small percentage have the ability to assist others in need.



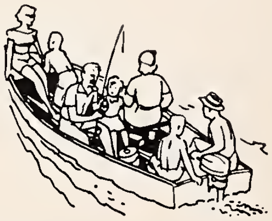
Overpowered boats are extremely tricky to handle. Match motor to boat.

Millions of people use the water as a means of transportation for business or pleasure. The majority of these people do not know the limitations of small crafts or have the ability to handle them safely. At one time, not too many people were in a position to purchase small craft, but today boats of all kinds are within easy reach of the average wage earner, and are operated by people at the risk of their own lives and the lives of others. Leading manufacturers stress safety in the construction of small craft.

Pre-season and post-season drownings have caused a large number of fatalities due to subnormal temperatures of the water. Very few swimmers can survive more than a very few minutes in subnormal water. With water safety education people need not die from drowning.

The Reason

People are handicapped in the water by habits they have acquired in their movements on land in an upright position. This position has been well established in a person's mind, and he will fight to assume it; therefore, little or no progress will be made if he is accidentally thrown in the water.



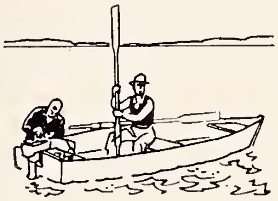
Overloaded boats swamp easily. Don't take the whole crowd at once—take turns!

There are a great number of people who believe that if one is thrown into water he instinctively swims for shore. Animals are born with this ability because movements are horizontal. People have to acquire this position under a competent instructor who teaches safety, how to avoid panic, ease of movements and how to conserve energy.

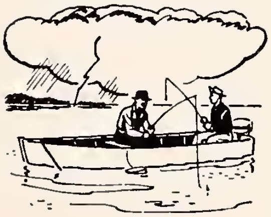
Mr. Hartmann is director of First Aid and Water Safety Services, Minneapolis and Hennepin County Chapter of The American National Red Cross.

Conditioning

To swim safely, for even a short distance, one must be in good condition and in order to have sufficient reserve stamina one must swim at least once or twice each week. Ninety per cent of the 90,000,000 people who swim are poor swimmers as they swim not more than seven or eight times a year and are therefore in poor condition. This also applies to other sports. Spur-of-the-moment sportsmen may find themselves in a desperate situation of exhaustion because of their poor physical condition. Regardless of all the warnings publicized by the press, radio, TV, posters and educational organizations, there are people who believe that a warning is not for them. Let's not fool ourselves—warnings are for *every one of us* if we want protection in and around water.



Stick with your boat in any kind of distress—motor failure or capsizing.



Head for shore the moment a storm comes up. Dawdling is risky.

Progress

Tremendous strides have been made since 1902 when the great Commodore Longfellow of the American National Red Cross pioneered the water safety program. One of his famous quotations was *"You can't think of a better sport to save your life."* Water fatalities have been reduced by 50 per cent through safety education. This was made possible by the work of leading organizations such as the YMCA, YWCA, Boy Scouts, schools, colleges, park boards, beaches, state recreational departments, American Red Cross and numerous other organizations that have not been mentioned. This reduction in fatalities would never have been made possible without all organizations co-operating. The American Red Cross alone issues 891,200 certificates in water safety annually, and has 42,000 authorized volunteer Water Safety Instructors throughout the United States.

ARTIFICIAL RESPIRATION

In the new back pressure—arm lift method, the victim is placed prone with the elbows bent and with one hand on the other. The cheek is placed on the hand, with the face turned slightly to one side. The operator kneels on one knee at the head of the victim. To start, the operator places his hands on the victim's back so that the thumbs just touch and the heels of the hands are just below a line running between the armpits.

He then rocks forward slowly, keeping the elbows straight, until his arms are approximately vertical, exerting steady pressure upon the chest.

Then he rocks backward, slowly sliding his hands to the victim's arms just above the elbows. Continuing to rock backward, he raises the arms until resistance and tension are felt at the victim's shoulder. Then he drops the arms and thus completes a full cycle. The cycles are repeated twelve times a minute, the expansion and compression phases being of equal length, and the release periods of minimum duration.

Caution: Never administer this method to a young child or an expectant mother. Artificial respiration is taught in Red Cross first-aid classes. It is important to give it properly. The best way to learn how to do it correctly is to take a course from a volunteer Red Cross first-aid instructor.

SAFETY SWIMMING RULES

1. Don't swim alone.
2. Wait an hour after meals before swimming.
3. Don't swim if tired or overheated.
4. Swim close to shore.
5. Don't ignore muscle cramps. Leave water immediately.
6. Don't swim from boats in deep water.
7. Be wary of river currents.
8. If your boat capsizes, stay with it.
9. Never stand or change positions in boats.
10. Don't overload boats.
11. Select a safe swimming place, preferably lifeguard-manned.
12. DON'T TAKE CHANCES AND—know how to save a person from drowning and how to apply artificial respiration by completing a Red Cross lifesaving course.

Common sense, you say? Yes, but three out of four drownings occur within 60 feet of shore—a stone's throw from safety.

Laboratory Aids

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The Minnesota Society
of Clinical Pathologists
George G. Stilwell, Editor

THE DIAGNOSIS OF PANCREATITIS BY LABORATORY METHODS

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A correct diagnosis of pancreatitis depends on the attending physician's being suspicious that the condition is present and the use of laboratory tests to substantiate such a diagnosis.

The diagnosis of pancreatitis is often missed because the condition is not considered in the differential diagnosis. It has been aptly recommended that a determination of serum amylase should be done routinely on the admission of any patient with acute abdominal disease, just as routine leukocyte counts are done.[1]

Pancreatitis is classified most simply by dividing it into two main categories, namely (1) acute pancreatitis and (2) chronic pancreatitis.

Acute Pancreatitis

The most important diagnostic laboratory test in this condition is a determination of the value for serum amylase done as quickly as possible in the course of the disease.[2] Serum amylase becomes increased shortly after the beginning of the clinical condition and remains increased for twenty-four to forty-eight hours. It may remain increased for a longer period but often it does not. If this test is done routinely on admission of all patients suffering from acute abdominal conditions, the diagnosis would be established in the great majority of patients who subsequently prove to be diagnostic problems. It must be emphasized, however, that not all patients who have acute pancreatitis will show an increase in serum amylase. When the disease is most severe and extensive destruction of the pancreas is present, the necrotic tissue secretes little or no enzyme, and as a result the blood levels are not increased. It has not been shown, however, that patients with such severe lesions do not routinely have increased serum amylase early in the course of the disease.

Other laboratory tests are available in the diagnosis of acute pancreatitis, the principal one of which is the determination of the level of lipase in serum and urine. Some authors consider that this test is helpful later in the course of the disease, when levels of serum amylase have become normal. This procedure is used by relatively few laboratories.[3,4] The results are not available for twenty-four hours because of the prolonged incubation period that is necessary. This delay is critical when one is trying to decide on the necessity of an emergency laparotomy.

Collateral laboratory evidence of acute pancreatitis consists of a decrease in blood calcium, which usually does not occur for three to four days after the attack, and transient hyperglycemia (not glycosuria) which occurs in 5 to 10 per cent of cases. Neither of the latter two tests is of great diagnostic value because the changes occur in relatively few cases of acute pancreatitis. When the blood calcium decreases to less than 7 mg. per 100 ml. in pancreatitis, the patient has a poor prognosis for recovery. Other tests, such as the antithrombin titer,[5] have been described but these have not been established as yet as reliable procedures.

It is apparent, therefore, that the laboratory diagnosis of acute pancreatitis relies almost completely on the demonstration of increased values for serum amylase very early in the disease. It must be remembered that an increase in serum amylase may occur in nonpancreatic conditions, such as perforated peptic ulcer, peritonitis, intestinal obstruction and dissecting aneurysm; the use of opiates also may lead to such increases.

This is the twenty-eighth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

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However, the values under such conditions almost always are less than 300 Somogyi units.

Chronic Pancreatitis

Under this heading are included instances in which the disease is of a prolonged nature, in the form of either recurrent attacks or persistent damage following an initial acute attack. Determinations of serum amylase are of much less value in the chronic form than in acute pancreatitis. Increased values commonly are found during the acute phases of recurrent attacks. Obviously, in those cases in which extensive destruction of pancreatic tissue has occurred, insufficient tissue remains to form amylase, which is reflected by normal values for this enzyme. Faulty metabolism of carbohydrate, protein and fat is present as the result of extensive destruction of pancreatic tissue. It is in these cases, therefore, that examination of the stool for an increase in total fat, undigested fat and fatty acids is helpful. Increased values are not diagnostic in themselves, since similar findings occur with overactive peristalsis when the transit time through the intestine is decreased.

The secretin test of pancreatic function[6] has been advocated in the diagnosis of chronic pancreatitis. In this test, following a twelve-hour fast, the duodenum is intubated and a dose of 1 unit of secretin per kilogram of body weight is given intravenously. The duodenal contents are collected at twenty-minute intervals for eighty minutes. A reduction of the volume of pancreatic secretion with maintenance of bicarbonate and enzymatic secretion is said to favor tumor, while secretion of normal volumes with a diminished bicarbonate response is said to favor chronic inflammation or fibrocystic disease.[4]

Summary

The laboratory is of great help in the diagnosis of pancreatitis, especially in the milder forms of this disease. It has been increasingly apparent that the disease is much commoner than heretofore suspected. However, as in most other diseases, results of laboratory tests cannot be relied on as a substitute for good clinical judgment. Increased values for serum amylase are extremely helpful as an aid to diagnosis in pancreatitis, but the values are not always increased, especially in extremely severe disease when laboratory help is most needed. Furthermore, the time during which the laboratory can be of greatest aid in cases of acute pancreatitis is limited to the first twenty-four to seventy-two hours of the attack. The laboratory is of less help in the diagnosis of chronic forms of pancreatitis. In the latter condition, clinical judgment, roentgenologic examination and laboratory tests all contribute a share to the diagnosis.

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LABYRINTHINE HYDROPS

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Current Concepts

IATROGENIC HEART DISEASE

CHARLES N. HENSEL, M.D.
Saint Paul, Minnesota

Typical Case History

Mrs. M. M., aged sixty, married, a social worker, stated on her first visit, May 8, 1945, "I have a heart condition and I want to know what that condition is."

She said that her heart skips beats, but mostly when she is in bed at night. She stated that some months ago, she was awakened from sleep about 5 a.m. with a severe pain in her left chest, located half way between the nipple and the breast bone, that she broke out in a clammy sweat and expected death at any moment.

The doctor arrived in a short time and gave her amyl-nitrite to inhale and the chest pain which already had begun to lessen, completely disappeared.

Later that day she was taken to the hospital, where her sedimentation rate was reported as 6 mm. and her electrocardiogram as "not diagnostic"; however, she was kept in bed, in the hospital, for thirty-three days.

While there, her blood pressure, which was recorded as 184/94 four months before the attack, was reported as 146/84. She was told that the second electrocardiogram showed very little change; however, she was warned that she had a serious heart condition and was advised to be careful.

She stated that since leaving the hospital she has worked short hours and done no house work.

Heart Symptoms.—She stated that she had an occasional "flick" of pain in the left chest in the daytime, but that at night, when she gets into bed, she is conscious of her heart working slowly and hard and that after a few minutes she trembles all over and even her teeth chatter. Then she gets warm all over.

Such spells may come every night for three to four weeks and then be absent for several nights. She stated that during the daytime, she is not conscious of her heart. She had raised four children and had always been healthy.

The physical examination revealed a ruddy cheeked, healthy appearing woman with a slightly enlarged thyroid gland containing several nodules, some hard and some elastic. The throat showed no abnormalities. The lungs were clear.

The heart was not enlarged, the action was brisk and forceful, the rate was 80 beats per minute and the rhythm was regular. The blood pressure was 176/96.

The abdomen was normal. The knee jerks were present and equal. There was no edema.

X-rays showed a normal heart, the electrocardiogram was normal, the blood count and blood chemistry and urine were all normal.

The basal metabolism was a minus 6 per cent and pulse rate during the test was 60-58-56 beats per minute.

She was told that her heart was normal and that there was no evidence of coronary disease, but that quite obviously, she had been badly frightened by the belief that she had serious heart disease and that fearful thoughts could make her heart misbehave.

She was told that since she did not have heart disease, she needed no medicines and needed no restrictions on her activities and should go home and lead a normal life.

A week later she reported that she was now sleeping well at night, that she no longer had chest pain and was not bothered when getting into bed at night. Her blood pressure was 124/80 and her heart rate was 72 beats per minute and regular.

Three years later, she reported that there had been a number of sudden deaths in her town and she had again become concerned about her heart. She was belching, there was gas rumbling through her bowels, and she felt occasional pain in the left chest.

On examination, recumbent, her blood pressure was 190/100, ten minutes later 176/90 and after a further rest 156/80. Her heart action was strong and regular. X-rays and an electrocardiogram again gave normal findings.

Nine years after her first visit, March 11, 1954, she reports that she has been living a busv and an active life, and that she nursed her husband through a "stroke" five years ago.

As to her heart, she told me that occasionally. at night, if she thinks too much, she gets an inner trembling and at such times she can feel her heart beating against her ribs, but that this no longer frightens her. She stated that she is not conscious of her heart in the daytime when she is busy.

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Editorials

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

MANAGEMENT OF THE FEDERAL DEBT

The objective of monetary policy, as determined by the Federal Reserve System, of fiscal policy, as determined by the legislative branch of the government, and of debt management, as determined by the Treasury Department, is economic stability through the maintenance of high employment, rising production and steady prices. The Research and Policy Committee of the Committee for Economic Development has recently released an interesting and thought provoking statement on *Managing the Federal Debt.*

The importance of debt management and its impact upon the economy has come sharply into focus since the war. In 1929, the Federal debt constituted only 8 per cent of all outstanding public and private debt, while today the Federal debt accounts for approximately 36 per cent of the total debt. The Federal debt held by the public at the end of 1953, excluding that part owned by the Federal Reserve Banks and various Federal Government accounts, aggregated $226.9 billion. The importance of the Federal debt upon the economy should not be measured in absolute size, but rather as a ratio of the size of the debt to the size of the national income. Since the end of the war this ratio has declined from 127 per cent to 74 per cent; the greater part of this decline having resulted from the increase in national income.

In deciding what is the "proper composition" of the Federal debt at any given time in order to minimize the effects of inflation and deflation upon the economy, five factors must be taken into consideration:

1. Too large a proportion of short-term debt requires frequent borrowing by the Treasury and may handicap the Federal Reserve in curbing inflationary tendencies.

2. On the other hand, too small a proportion of short-term Federal debt may tend to accentuate deflationary tendencies by restricting bank lending and private spending.

3. If the Treasury were to shift from short-term to long-term debt, some private businesses might be induced to shift to short-term borrowing. Such a shift in substantial proportions would make business more vulnerable to a recession.

4. Changing economic conditions are reflected in substantial fluctuations in the market value of outstanding long-term Federal securities. Such fluctuations in market prices tend to have a stabilizing influence upon the economy.

5. A wide distribution of the Federal debt among maturities and holders is an effective method of transmitting credit ease or tightness to other segments of the capital market.

In order to attain a greater degree of economic stability, the CED recommends that the debt structure be modified in four respects:

1. "The amount of very short-term marketable debt, due within one or two years, should be smaller."

2. "Most of the increase to take the place of the reduced volume of short-term marketable debt should be in the marketable debt of intermediate term, say up to ten years."

3. "The long-term marketable debt oustanding should be moderately larger."

4. "A larger part of the total debt should be in the form of savings bonds."

Edward B. Chapin
Assistant Vice President
First National Bank of Saint Paul

THE DIABETIC PATIENT AND HIS CHEST X-RAY

It is recognized that patients with uncontrolled diabetes mellitus are predisposed to certain diseases. X-ray examination of the chest may be of assistance in finding some of these conditions. In this category belong pulmonary tuberculosis, lung cancer, lung abscess and pulmonary congestion. Roentgenograms of the chest are preferable to fluoroscopic examination. In any event, more time should be spent with the inspection of the right lung than of the left lung. Normally, from 53 to 56 per cent of the air inhaled goes to the right lung. Consequently, proportionately greater number of pathogenic microorganisms and larger amounts of carcinogens are deposited in the right lung than in the left lung. Primary tuberculosis, primary bronchogenic cancer and lung abscess are found with a correspondingly higher frequency on the right side. Patients with diabetes mellitus should have a roentgenogram of the chest every six months.

The highest incidence of pulmonary tuberculosis is observed in young adult men. Both in primary and in reinfection forms of this disease predominantly exudative lesions are common. Concurrent enlargement of the hilar lymph nodes is frequent in primary tuberculosis. One should look for configurations suggestive of cavity. Also, one may see a radial linear shadow connecting the hilus to the disease focus. It represents lymphangitis. Bronchopneumonia or lobar pneumonia of tuberculous origin may be noted. It is well to keep in mind, however, that in a great many instances the tuberculous process appears as a localized conglomeration of small nodules. Rarely, one may find a round, "coin," shadow of 1 to 3 cm. in diameter. When such circular shadows are multiple, they are suggestive of metastatic tumors. Miliary nodules or larger, coarse nodules widely scattered in both lung fields are also encountered. The apex of the lung may be free of disease. Predilectional site for early reinfection type of tuberculosis is the superior retroradicular region. Early pleural tuberculosis is uncommon. Occasionally, fibrinous pleurisy or tuberculous pleurisy with effusion may complicate specific lung involvement.

The increased incidence of primary bronchogenic carcinoma in patients with diabetes is attributable to the following causes.

1. The tissues of these patients are older than their chronologic age. For reasons unknown, senescence is the period when epithelial cells become anarchistic and develop a tendency to uncontrollable proliferation and spread.

2. Cancer cells are "glycophilic." They thrive on dextrose. This has been demonstrated in tissue cultures. Obviously, hyperglycemia represents a favorable environment for cancer growth.

3. Hepatic dysfunction in uncontrolled diabetes results in hypovitaminosis A. It is known that lack of vitamin A causes hyperkeratotic metaplasia of the bronchial mucosa. Such changes may be looked upon as the premonitory phase of lung cancer.

The high incidence of lung abscess is explained on the basis of immunologic inferiority (cellular and humoral) of the body. Also, aspiration of infected material from the oropharynx and mouth during coma is instrumental in the causation of lung abscess.

Heart failure resulting from vascular degenerative changes may lead to pulmonary congestion and edema.

In closing, it is well to emphasize a few pertinent points.

1. Roentgenogram of the lung is only one of the sources of diagnostic information. X-ray pictures represent shadows which do not reveal the nature of the disease casting the shadow.

2. X-ray shadows must be interpreted in the light of other diagnostic data and findings which are procured by history-taking, physical and laboratory examinations.

3. It does not detract an iota from the dignity of the medical profession to admit to the limitations of x-ray examination. On the contrary, cognizance of these limitations adds to the profession's intellectual integrity and to the doctor's diagnostic competence.

Andrew L. Banyai, M.D.

HAROFE HAIVRI

We recently received a copy of *Harofé Haivri.* It evoked thoughts, memories, and sensations—all pleasant—which we shall discuss in part and circumstantially (if we were poetic, this could be classed as imaginative rather than circumstantial).

First of all, we owe Dr. Moses Einhorn, the editor of the bilingual *Hebrew Medical Journal (Harofé Haivri),* our thanks.

Next, we are reminded by the Hebrew letters and the pagination from back to front, of Dr. Matas. *That* reminiscence goes like this: A friend of ours interned in New Orleans about twenty-five years ago specifically to be able to work under Dr. Matas. Even then, Dr. Matas was a venerable old man of medicine. In the course of making rounds one day, the staff trooped into the private room of a little, old Hebrew lady. The only means of communication between her and Dr. Matas was Hebrew, with which the linguistically accomplished doctor struggled somewhat haltingly. Finally the staff filed out of the room again and Dr. Matas, already past the age at which most men retire from work, and surely from any pretense of learning anything new, said, with only a minor note of irritation, "Well, well, now I'll have to brush up on my Hebrew." We don't have any Hebrew to brush up on, so we are still trying to figure out how come this journal was sent to us. The accompanying letter gave us no sales talk; it didn't even mention a subscription price, as a matter of fact.

A classmate of ours used to point out that we should be smart enough to call on his medical fraternity if our class needed any of its financial affairs managed. We are also convinced that we might well call on the same Hebrew fraternity's scholars for well-written medical papers. We base this judgment on English papers in this issue of the *Hebrew Medical Journal,* as well as on articles from other journals.

We enjoyed particularly the series of short articles on Maimonides, written in honor of his 750th anniversary. And the section on Medicine and Religion consists of an article by an Irishman, I. Jakobovits (this copy of the journal arrived on St. Patrick's Day, and, lest you misapprehend, we point out that this Irishman is not only from Ireland, but is the Chief Rabbi of Ireland) entitled *Sterilisation and Eugenics in Jewish Law* that is a readable, scholarly discussion of this moral-socio-economic topic.

Because at least some of you will have a sufficiently inquiring mind to want to pursue these reveries to their source, we tell you that Dr. Moses Einhorn's address is listed as: 983 Park Ave., New York 28, N. Y.

H.G.M.

FLEXIBLE FARM PRICES

Under our heritage of American competitive enterprise systems, the prices paid to farmers for the products they produce should be consistent with the consumptive demand for that product. The voters in the market place have helped for years to determine generally what farm prices should be and what the farmers should produce. Farm prices should have a relatively free interplay of the market in response to demand and supply level.

In 1938, the Congress of the United States passed the Commodity Credit Loan Act which has been highly abused as far as its original purpose is concerned. In the later depression years, the farmers that were left were in rather tough financial shape and the Commodity Credit Loan program was simply a government guaranteed loan program which would enable the farmer to hold his produce until the market could firm up and he could get a better price. In this way, the farmer would get his money from the market place—not government. The government only acted as a loan guaranteer to the banks or elevators that were actually loaning the money.

The same act established the "old parity formulas" which was a simple mathematical calculation of what a unit of a farm product was worth. This "true value" or "100 per cent of parity" value of each crop bushel was calculated from farmers' costs. As farmers' cost rises, the parity or "true value" calculated price should also rise proportionately. The same act provided that these loans to farmers should be set up on a graduated or "flexible" scale basis.

The annual loan guarantee was to be set on each basic farm product each year commensurate with the *supply needs.* If the supply produced was larger than the combined total of (1) domestic need, (2) export need and (3) normal reserve as provided for by law then we would have a "surplus" and supports should drop on a "flexible" scale of one (1) point support drop for each 2 per cent additional surplus produced and not sold. This was to increase sales, discourage production of that commodity and keep the government from having to buy and store and compete for a later market against the farmer.

In 1938, the support level ranged from 75 to 52 per cent of parity depending on supply. Congress later amended the act to provide for price supports on basic crops at 90 per cent of parity or "true value" for the duration of the war and two years after in order to stabilize farm income.

In some crops, wheat as an example, the "support level price" was high enough to be a profitable price and as a result, we have now over a two years supply of wheat on hand, mainly in the hands of the government competing for current markets. At the same time the farmers are under government enforced acreage cuts, less than "true value" prices and are really getting hurt.

This is a perfect example of government bungling of a private enterprise business.

The politicians bought farm votes with "high rigid price supports" and in turn they took away the farmer's market, his right to fully produce and his opportunity for management decision.

Farm Bureau, the voice of agriculture, along with the help of others including independent and professional businessmen is determined to break the ever tightening band of government controls and "political paternalism" that can only lead to the downfall of our free enterprise system.

J. Delbert Wells
Secretary, Minnesota Farm Bureau Federation

GENERAL PRACTICE RESIDENCY

There has been a growing trend recently for hospitals to provide residencies designed for general practice. This type of residency has long been needed to fill a need for the development of further training to the men who desire to specialize in general practice. Men who select such residencies do so in order to round out their previous medical training. This program provides them with an opportunity to learn the nature of general practice. It further enables them to come in contact with men who are engaged in family practice and to learn from them the art, as well as the science, of medicine. A well-developed general practice residency gives the participant an opportunity to see and assist in the practice of medicine on the community level. It also enables the participant to gain some insight as to the problems and the nature of such a practice.

The general practice resident has an opportunity to see the application of medicine to the individual as a whole, rather than the specialized approach to the patient in which an organ, or group of organs, is studied and treated. During this residency, however, the physician is trained not only by men who are engaged in general practice but he also is trained by specialists in the various fields of medicine. An association of the general practitioner with the specialists in this training program has proven to be of invaluable assistance to the student physician. This type of residency should be further developed, and more opportunities should be available for men who are to engage in general practice so that they may have these residencies available to them. The quality of such a residency, and the benefit that accrues to the physician who engages in such a residency is immeasurable and will reflect itself on the medical care he renders to the families in his community.

A. E. Ritt, M.D.

DROWNINGS—A KILLER OF MINNESOTANS

With summer upon us, the problem of drowning in one of our ten thousand lakes (to say nothing of our many rivers, brooks, sloughs, wells and cisterns) is with us for several months ahead. Have you given your safety vaccination shots for water hazards? Have you spoken to your young mothers about children's supervision at the beach, life preservers when in boats, swimming safety rules?

Drowning is one of the leading "killers" of Minnesotans! Do peruse Mr. Hartman's comments in this issue on "Water Safety for Minnesotans."

Tague C. Chisholm, M.D.

THE SCIENCE REPORTER

With Americans demanding more and more information about all types of scientific developments and medical progress, the role of the science reporter gains in importance daily.

It's time to face it—the science writer is a permanent fixture on American newspapers.

But as he gains in importance, he also must assume greater responsibilities, not only to his readers but also to the medical man, the physicist or the biochemist with whom he deals.

The wariness with which both the scientist and the science reporter often regard each other must be supplanted with an atmosphere of trust and cooperation before both can fulfill their joint obligations to an information-eager public.

Problems voiced last summer, for example, during a panel discussion at the University of Michigan must be solved, and that can be done only through joint efforts on the part of both journalists and men of science.

Dr. Allen V. Astin, director of the Bureau of Standards, criticized the press for generalization, extending the bounds of a scientist's work —bounds that are carefully set.

On the other hand, Paul Block, Jr., publisher of the *Toledo Blade*, criticized scientists for an unwillingness to interpret the effects or the impact of their work and leaving that job to the reporter.

Both agreed that the scientist and the newsman have similar goals: to collect the facts and report them accurately. The big difference lies in the emphasis each places on those goals.

The scientist often is as much interested in the methods used to arrive at a specific goal as he is in the results themselves.

The reporter, by the very nature of his work, is primarily interested in the results of the scientist's labors and uses only those facts concerning methods that he feels are necessary to explain the procedures to his readers.

But it is not necessary to recount the arguments pro and con here. Suffice to say that only by learning more about the other's problems can we hope to eliminate any causes of friction that may exist between the two groups.

Who is the science reporter? What are his qualifications, his background?

Since few newspapers have an editorial budget large enough to include a full-time science writer—one who does nothing but feel the medical or scientific pulse of his community daily—most science reporters at present are of the "part-time" variety.

Today, they may interview a surgeon about a new operative technique. Tomorrow, or perhaps only an hour later, they may be covering a three-alarm fire.

Some men handling science news do so because their "beats" bring them into contact with researchers. The reporter covering the campus of a large college or university is an excellent example.

The average science reporter may or may not have had any formal education in the advanced sciences but he generally is a college graduate and has to be a competent newsman in order to hold down the job for any length of time. If he isn't competent, his scientific news sources dry up and he is no longer of any use to his paper.

He can get involved in science reporting any number of ways. He may have volunteered for the job primarily because of intense interest and as a result, what he may lack in educational resources he makes up for in enthusiasm.

Of course this educational lack or unfamiliarity with terminology can hinder him in relations with some scientists. Often he is forced to ask questions that seem extremely basic to the medical man or researcher (better that than have him attempt to bluff his way through without completely understanding the material discussed). It must also be remembered that not even the most experienced science reporter is familiar with all fields of science.

A good science reporter (as any good general reporter) is accurate.

Here, too, arises the question whether a science reporter should have his articles checked by the "expert" before publication. Most good reporters, I believe, do recheck their material when it is of a highly technical or specialized nature. The reporter is not any happier with major errors than is the source of his story. Such checking, however, generally is limited to errors of fact and not to style or method of presentation. (Suggestions to change a writer's style are akin to a layman's suggesting to a surgeon that he change the type of incision he uses for appendectomies.)

The good science reporter can and will keep a confidence.

A recent complaint registered by a man on a science run was that scientists refuse to keep him informed of the progress of their projects.

"I have no intention of printing anything until they say the word," the reporter explained. "I just want to follow the thing along so that when the story is ready for publication I'll know the background and have some basic knowledge of the work."

A science reporter should not attempt to sensationalize news but, as one writer pointed out, "When a subject like the development of cortisone is involved, how can you avoid it?" In the same regard, the reporter should remember that not all medical or scientific developments have immediate practical application.

Many critics of the present crop of science reporters deplore the fact that there are not enough fully qualified men in the field.

To such critics must be put the question, "Whose responsibility is it to see that more qualified writers are developed? The newspapers? Journalism schools? The scientists?"

Actually, all three have a deep responsibility in this regard.

Journalism schools should be encouraged to train more men in medical and other scientific fields of reporting. At present there are few schools that have curriculums designed to fill that need.

Newspapers should be encouraged to make a place on their staffs, whenever possible, for a full time science writer since the man who is a

(Continued on Page 441)

President's Letter

LEGISLATIVE REVIEW

The Minnesota Legislature of 1955 considered and acted upon many items which are of interest to the medical profession of the state. Below is a summary of some of the most important items and the action that was taken:

Senate File 1174—This would have limited the state's share of the cost of medical, hospital, nursing home, and dental care, including drugs and medicines, to $25.00 per month for all cases of recipients of old age assistance. The chairman of the Senate Committee on Public Welfare worked vigorously for the passage of the bill, notwithstanding the united opposition of the medical profession, the hospitals, druggists, county commissions, county welfare boards and others. The proposal was overwhelmingly defeated in the House of Representatives. The sharing of such costs presently are on a 50-50 basis between state and county. In the future, the sharing will be state, 45 per cent, and county, 55 per cent.

The Legislature continued for two years more the appropriation of $75,000 per year for nurse training scholarships. Maximum scholarship is $600 for one studying to be a registered nurse; a maximum of $300 for one studying to be a practical nurse. Basis for the award is need and ability.

The Legislature again refused to grant full medical and surgical privileges to osteopaths. The bill also would have added two osteopaths to the State Board of Medical Examiners. The bill was defeated in the Senate.

For a second time, the Legislature refused to create an examining and licensing board for psychiatric aides. The bill was supported by some of the psychiatric aides at the state hospitals. There is serious doubt as to the need for such legislation.

The Legislature increased the maximum compensation of the administrators of state hospitals to $12,250 per year, plus complete maintenance.

The Legislature appropriated $400,000 toward the construction and equipment of a medical, dental, biological library building at the University of Minnesota. The University was authorized to accept any further sum for such purpose that may be made available by the Congress of the United States. The original request of the University for such a building was $875,000.

The Legislature appropriated $65,000 for mental health research in the fiscal year 1956 and $85,000 for fiscal year 1957. Also, $75,000 for the mental health training program in fiscal 1956 and $98,600 in fiscal 1957, of which $30,000 per year may be used for the employment of additional psychiatrists.

The Legislature again refused to authorize the corporate practice of medicine by consumer controlled groups and cooperatives. The bills did not provide for free choice of physician nor did they require any financial guaranty of the organizers.

On and after January 1. 1963, the requirement of high school education as a prerequisite to taking the Minnesota Basic Science examination is increased to one year of approved college education; after January 1, 1965, the requirement will be two years of approved college. The change will affect some chiropractic applicants.

It can be seen by the above that the Legislature this year considered a great many important measures which affect the medical and allied professions in this state, and that this large volume of legislation on medical and allied matters shows the increasing interest our legislators have in scientific and medical economic proposals.

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

AMA OFFICIALS HEAR POLIO REPORT

When the safety and effectiveness of the Salk polio vaccine were announced on April 12, a simultaneous scientific meeting was begun. Attending the scientific session on behalf of the American Medical Association was Board of Trustees chairman, Dr. Dwight H. Murray; President-elect, Dr. Elmer Hess; Assistant Secretary, Ernest B. Howard; and *AMA Journal* editor, Austin Smith.

The American Medical Association *Secretary's Letter* reports:

> "When the National Foundation for Infantile Paralysis announced that the vaccine works and is 80 to 90 per cent effective in preventing paralytic polio, the biggest medical news story of the century unfolded amid the fanfare and drama typical of a Hollywood premiere. More than 300 press, radio, and television people were on hand to cover the event."

Reactions were varied to the dramatic announcement. Said Dr. Murray: "The American people can be assured that the thousands of practicing physicians in cities, towns, and hamlets will give their wholehearted co-operation to get the vaccination programs under way as effectively and safely as is humanly possible." He urged parents to have patience and not rush to doctors' offices immediately because probably most parents already have immunity to the disease. "Give children priority," he added.

However, although the Ann Arbor event was a glorious day for American medicine, many politicians moved in with some "corny" statements and sickening actions, noted in the Secretary's Letter.

The *Letter* quotes a *Chicago Tribune* editorial:

> "No sooner had the worth of the Salk vaccine been scientifically affirmed than the Oregon maverick (Senator Morse) tried to get into the act. He introduced a bill in the senate for a five-man bureaucratic commission to take control of the vaccine out of the hands of the people who discoverd it and are supplying it, out of the hands of the medical profession which will administer it, and supply it to the nation as a blessing of the government."

The *Tribune* editorial goes on:

> "Government had nothing to do with discovering the vaccine. . . . You could lock Jaybird up in a laboratory, give him all the apparatus and materials needed, plus the most minute instructions from the discoverer, and he couldn't turn out a single usable portion of the vaccine between now and the time the voters of Oregon will have a chance to catch up with him. But now that the job has been done by others, he thinks he is just the man to take over. . . . Does he maintain that the physicians of the United States will be parties to extortion? Apparently he does, for this is Jaybird's song: 'You can't trust commercial interests. You can't trust doctors. If you want a polio shot, see your precinct captain.'"

Minnesota Program Outlined

Considerable confusion over the administration of the Salk polio vaccine in Minnesota has existed, as it has in most states. However, as of May 15, the situation was as follows:

The Council of the Minnesota State Medical Association approved the following priority schedule for vaccinations in private practice, to be administered after the first and second graders have been vaccinated under the program of the health department: (1) pregnant women, because they are most susceptible to poliomyelitis; (2) children in age groups fanning out from the six-to seven-year group, until all children from one through twelve have been vaccinated: five to eight, then four to nine, then two to eleven and finally all from one to twelve.

The Council also went on record against any "first come-first served policy" in the doctor's office. Also, until the supply is increased, the Council decided that the vaccine should be withheld from babies under one year of age.

The Minnesota Department of Health has issued several summaries of recommendations on the actual administration of the vaccine, also. Plans were to vaccinate all pupils of the first and second grades, using vaccine furnished by the National Foundation for Infantile Paralysis, thus covering some 133,000 children in the state. However, since the recalling of certain supplies of the vaccine by the federal government, the entire program has been held up pending final approval of batches already received, and approval of future batches to be received for the program. This approval, or a statement on the entire program as recommended by the federal government, was not yet available at the above-mentioned date.

CHARGES MADE AGAINST MORE HEALTH INSURANCE FIRMS

Five additional insurance companies were charged by the Federal Trade Commission with misrepresenting the benefits of the health and accident policies. The new charges bring to twenty-eight the number of companies against which the FTC has filed complaints. Two have agreed not to engage in the challenged advertising practices.

According to a recent story in the *Washington Post and Times-Herald,* "The five firms collect premiums of 35 million dollars a year in the billion-dollar field of individual policies on health, accident and hospitalization. All five were charged with misrepresenting in advertising the extent and length of coverage."

The new firms charged were: Educators Mutual Insurance Co. of Lancaster, Pa.; Beneficial Standard Life Insurance Co. of Los Angeles; Fireman's Fund Indemnity Co. of San Francisco; National Casualty Co. of Detroit, and Federal Life and Casualty Co. of Battle Creek, Michigan.

FTC Authority Upheld

Along this same line, a hearing examiner has ruled that the Federal Trade Commission has authority to police the interstate advertising of health and accident insurance companies. The ruling came as a result of two previously charged companies challenging the commission's jurisdiction over such advertising. Both of these companies asked dismissal of the FTC complaints on the grounds that the companies were adequately supervised by the states that licensed them.

However, according to a *New York Times* story recently: "Hearing Examiner Loren H. Laughlin held that Congress 'clearly intended' for the F.T.C. to police the interstate mail-order business of the companies. The two companies can appeal, if they choose to do so, and get a ruling from the commission itself."

INSURANCE COMPANY OFFERS CANCER RIDER

The Continental Casualty company now has on the market and for the first time cancer benefits up to $5,000 by rider to the accident and health group insurance plans covering 100 or more employes and to be sold through the company's general group division in all states except Maryland. According to the monthly house organ of the company, another new rider to group policies embraces ten dread diseases. These benefits, up to $5,000, may be sold with any basic general group plan.

According to the publication, "The cancer rider will cover for the disease when cancer is first diagnosed while the insurance is in force. Expenses taken care of will be those incurred within two years of the first treatment, beginning with diagnosis. They are payable in lieu of benefits that would otherwise be paid under the base plan. Employes' monthly premium for the cancer benefits will be 55 cents. The family rate for employe and eligible dependents will be $1.17 per month. It is emphasized that at the present time the benefits are available only to groups of 100 or more employes. They include hospital room and board, necessary services; physicians', surgical and nurses' fees."

The company also reports that the dread diseases covered for $5,000 aggregate include polio, scarlet fever, diphtheria, spinal meningitis, encephalitis, rabies, tetanus, tularemia, typhoid or leukemia, symptoms of which first appear while the insurance is in force.

GROUP CLINIC REGULATION UPHELD

A 1953 act of the California legislature regulating private out-patient clinics operated by groups of physicians was declared to be constitutional recently. An article in the San Francisco *Chronicle* noted that Attorney General Edmund G. Brown made the ruling, which was requested by Dr. Malcolm Merrill, director of the California State Department of Public Health.

The chief effect of the act is to prevent formation of medical groups under names that do not reveal the names of the physicians involved, as in the Permanente Medical Group, the Palo Alto Clinic and San Mateo Clinic, members of Brown's staff said. According to the news story: "Groups formed after January 1, 1954, are required to use the last name of at least one of the physicians involved, followed by the words 'Medical Group.'"

Success Undermined?

Apparently, some of the clinics and groups involved feel that the regulating law undermines their success. The story goes on to say: "Although the law does not affect actual operation of the groups, it was pointed out that some clinics believe a large part of their success is based on public familiarity with the established names. The law provides that any clinic established before 1954 can continue to operate under the established name unless there is a change in the partnership, in which case a new license must be acquired and the new regulation followed.

The article quotes Mr. Harry Fledderman, attorney for the sixty-member Permanente Health Group, who said:

> "It would be considered a serious matter if the group has to change its name. The Permanente partnership contracts to provide medical care in Kaiser Foundation Hospitals and also operates several out-patient clinics separated from the hospitals. The group sometimes takes in or loses several members of the partnership yearly, which may force a name change."

The article further states:

> "The law provides exemptions for out-patient clinics operated as integral parts of hospitals. The Legislature, when it passed the law, apparently felt that operation of clinics without including at least one name of a physician involved bordered on practicing under a fictitious name, members of Brown's staff said. There are 195 out-patient clinics in California that will be affected. Charity, teaching and research and employer clinics are exempt from the regulation."

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

LICENSE OF MINNESOTA PHYSICIAN SUSPENDED FOR FIVE YEARS

In the Matter of the Revocation of the License of Kenneth L. Kelsey, M.D.

The Minnesota State Board of Medical Examiners on May 6, 1955, suspended for a period of five years the medical license held by Dr. Kenneth L. Kelsey, an Aurora, Missouri, physician who intended to open an office for the practice of medicine at Annandale, Minnesota. When Dr. Kelsey came to Minnesota in September, 1954, and attempted to pay up the arrears in his registration fees in reference to his Minnesota medical license, an investigation disclosed that he was personally using large amounts of barbiturate drugs, especially nembutal. A citation for the revocation of Dr. Kelsey's license was issued in which he was charged with habitual indulgence in the use of barbiturate drugs, and also with furnishing, prescribing and administering narcotic drugs to his wife, not in the bona fide practice of medicine.

At the time Dr. Kelsey appeared before the Board on February 4, 1955, in response to the citation, both he and Mrs. Kelsey had been hospitalized since the first part of October, 1954. Dr. Kelsey admitted the truth of the charges contained in the citation and, after he gave his promise to the Medical Board that he would refrain from applying for a narcotic tax stamp, the Board took Dr. Kelsey's case under consideration, the decision to be held in abeyance until it could be determined what could be worked out that would be beneficial to Dr. Kelsey and his wife. However, when Dr. Kelsey's case was again taken up by the Medical Board on May 6, 1955, it appeared that Dr. Kelsey had left the hospital on his own request in the latter part of February and had subsequently gone back to Missouri.

Dr. Kelsey was born in Saint Paul, Minnesota, on February 25, 1911. He graduated from the University of Minnesota with an M.D. degree in 1934 and was licensed to practice medicine in Minnesota in the same year. Before coming to Minnesota in the fall of 1954, Dr. Kelsey had practiced medicine at Aurora, Missouri, since 1936.

THE SCIENCE REPORTER

(Continued from Page 437)

part-timer is admittedly less qualified to develop science news adequately.

But a great deal of the responsibility also falls on the shoulders of the scientists, themselves. They should be willing to support any programs designed to get information about scientific progress to the public with speed, completeness, and accuracy.

The attitude that any medical man or researcher who is mentioned in the press is automatically a "publicity hound" or a violator of professional ethics hinders any such programs.

The problems that face the science reporter and the scientist in getting news to the public are vast and will not be solved quickly. But they will never be resolved until all interested parties are willing to lend a hand.

John R. Finnegan
Saint Paul Pioneer Press and Dispatch

The Dean's Page

THE FAMILY PHYSICIAN

In a recent issue of *The Chronicle* of Plymouth Congregational Church, Minneapolis, of which Mrs. Diehl and I are members, our minister, Dr. Howard J. Conn, published such a splendid statement of the spirit and service rendered by the family physician that I requested his permission to reprint it as follows:

> "I always think of Jesus as the family physician of the human race. Religion to Him existed for the purpose of action; it was valuable solely for the service of mankind."
>
> So wrote Sir Wilfred Grenfell, the great Labrador missionary doctor back in 1911.
>
> Think of the role of the family physician, and realize how strikingly Jesus Christ can play a similar part in our lives. The doctor seeks to
>
> —diagnose our ills;
> —appreciate the family background;
> —read the hidden thoughts of the patient;
> —understand the universal rules of health;
> —prescribe a course of action;
> —see the long-range possibilities as well as the immediate cure;
> —encourage us to co-operate;
> —love us in every situation;
> —draw forth the best in us;
> —blend medical science, divine power and human faith into a healing channel.
>
> What the dedicated doctor seeks to do for his patient, Jesus can do for the moral and spiritual well-being of the whole human race.
>
> But just as the doctor must be called before he can assist, so Christ must be welcomed into the heart. Lent is a period when we especially do this.
>
> God be praised for the family physician!
>
> God be praised for Jesus Christ, the family physician of the human race!
>
> God be praised when we invite Him in, and entrust our lives to His guidance!"

The Medical School of the University of Minnesota is striving to select and train young men and women to be "dedicated doctors" and "family physicians" of the type described by Dr. Conn. Incidentally, Dr. Conn is substituting for Dr. Ralph Sockman on the National Radio Pulpit—NBC— 9 a.m. Sunday mornings, C.S.T.

HAROLD S. DIEHL, M.D.
Dean of Medical Sciences
University of Minnesota

Public Health

PROGRAM FOR PROFESSIONAL PERSONNEL IN NATIONAL EMERGENCIES

The Commissioned Reserve of the Public Health Service is being expanded and reorganized for service in grave national emergencies as the result of a delegation by the Federal Civil Defense Administration to the Department of Health, Education and Welfare which was approved by the President on July 14, 1954. Commissioning of officers in the emergency reserve is under way.

Function.—There are three main groupings of professional personnel in the Commissioned Corps of the Public Health Service—Regular Corps officers, Reserve Corps officers on active duty and Reserve Corps officers in inactive status. It is the latter group that is being expanded and reorganized under the Service's emergency program.

Reserve Officers in inactive status, now and in the future, may apply for active duty and will be considered for available assignments. But no officer of the emergency reserve is subject to mandatory duty except in the event of a national emergency publicly recognized as requiring such action. Moreover, even in an emergency, an officer of the emergency reserve already performing important health duties would be assigned to active duty in another area only if the need in that area were so serious as to clearly require his services there.

Size.—The number of reserve officers to be appointed under the present program is limited. Ultimately the reserve is expected to be built up to approximately 5,000 officers—physicians, dentists, sanitary engineers, nurses and persons in allied health professions. At the present time, the program provides for 2,000 officers. The principal need at this stage is for physicians.

Eligibility.—To be eligible for appointment to the Commissioned Reserve, a person must be an American citizen at least twenty-one years old and must have a professional degree. A person need not have had experience in the public health field to be eligible. Grades in the Commissioned Reserve correspond to the grades in the regular Commissioned Corps of the Public Health Service and to grades in the Army, Navy, and Air Force. The grade to which an officer is appointed is determined by his years of education, training and experience.

Duration of Appointments.—All Reserve Corps commissions in the Public Health Service are for periods of five years. They are made by the Secretary of Health, Education, and Welfare, and are renewable at the end of each five-year period. If the officers is reappointed, his grade is determined by the level of his training and experience at that time. An officer can resign his commission at any time.

Training Program.—The Service will make every effort to train officers of the Commissioned Reserve in present-day emergency health problems, particularly those associated with atomic, biological and chemical warfare. Insofar as funds permit, officers of the Commissioned Reserve will be given opportunities to go on active duty for limited periods for training purposes.

The Service also will offer opportunities for training not involving active duty. As the reserve is built up to emergency strength, a variety of training methods will be employed. The aim is that every officer of the reserve will receive some basic training in emergency health problems. Intensive, specialized training is planned for a limited number of reserve officers.

Appointments.—The first step toward obtaining a commission as a reserve officer of the Public Health Service is to submit an application on a form provided by the Service.

Filling out this application obligates neither the applicant nor the Service. It does, however, provide the Service with the necessary information on which to determine whether the applicant is eligible and, if eligible, to what grade he should be appointed. If the applicant is judged eligible, he will be informed of the next step.

General Considerations.—No one can know in advance what he will be doing if a grave national emergency develops. But one thing is certain: Everyone with training and experience in health professions will be needed in a dozen places at once.

Obviously, we must anticipate as many of the problems as we can, get ready to deal with them in the most realistic way possible and stay ready.

In any successful attack, treatment and care of the injured will be a monumental task, but it will be only part of the problem. Those who escape direct injury will be the human resources we have left. Their health will have to be safeguarded at all costs.

Even in emergencies resulting from natural causes, health personnel are often spread too thin or are not readily available.

In an attack, these problems would breed and multiply—not in days and weeks but in hours and minutes.

There must be provision for physicians, en-

(Continued on Page 450)

Reports and Announcements

MEDICAL MEETINGS

National

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

International

International College of Surgeons, Cape Cod regional meeting, Chatham Bars Inn, Chatham, Massachusetts, July 1-4, 1955.

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

Applications for certification for the 1956 Part I examinations are now being accepted by the American Board of Obstetrics and Gynecology. Candidates are urged to make application as early as possible—before October 1, 1955.

Information can be obtained from the secretary of the board, Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, Ohio.

MINNESOTA SYMPOSIUM ON ARTERIOSCLEROSIS

The Minnesota Symposium on Arteriosclerosis, sponsored by the University of Minnesota and the Minnesota Heart Association, will be held at the Mayo Memorial, University of Minnesota Medical School, Minneapolis, on September 7, 8 and 9, 1955.

Five general sessions and a half-day of clinic and laboratory visits will be devoted to five major topics: "The Challenge of Arteriosclerosis," "Atherosclerosis," "Peripheral and Cerebral Arteriosclerosis," "The Internist and Some Tools," and "The Surgical Treatment of Arteriosclerosis."

In addition to Minnesota speakers, guest lecturers will include representatives from other U. S. medical centers and from South Africa, Sweden, Canada, Scotland, Italy and Germany. Principal speaker at a dinner meeting at the Minneapolis Club on September 7 will be Dr. Paul Dudley White, Boston, who will discuss "The Coronaries Through the Ages."

General chairman of the symposium is Dr. Ancel Keys of the University of Minnesota. For further information, reservations, housing, et cetera, write Dr. Robert B. Howard, Mayo Memorial, University of Minnesota, Minneapolis 14, Minnesota.

ASSEMBLY IN OTOLARYNGOLOGY

The Department of Otolaryngology, University of Illinois College of Medicine, announces its annual Assembly in Otolaryngology from September 19 through October 1. This assembly will consist of two parts.

Part 1, September 19-24, will be devoted to surgical anatomy of the head and neck, fundamental principles of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Dr. Maurice F. Snitman. Part 2, September 26-October 1, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Dr. Emanuel M. Skolnik.

Registration is optional for one or both weeks.

WORLD MEDICAL ASSOCIATION JOURNALISM MEETING

The sixth medical journalism meeting, sponsored by the World Medical Association, will be held in connection with the association's ninth General Assembly in Vienna, Austria, September 20 to 26, 1955. September 23 will be devoted to the medical journalism program, at which the main topic will be "Problems of Teutonic Medical Publications."

Editors of medical journals are invited to attend. A complete program will be sent on request. For further information, write the World Medical Association, 345 East 46th Street, New York 17, N. Y.

INDUSTRIAL MEDICINE COURSE

A full-time eight-week comprehensive course in industrial medicine for physicians will be given in the Post-Graduate Medical School of New York University-Bellevue Medical Center commencing Monday, September 26, 1955.

Among the subjects being offered to physicians are: organization, administration and economics of an industrial medical department, the practice of preventive and constructive medicine in industry, the clinical aspects of occupational diseases, industrial injuries and the elements of safety programs, toxicology and industrial hygiene for the physician. Opportunities will be provided for attendance at medical, surgical and clinical pathological conferences during the course.

Applications should be sent to the Dean, NYU Post-Graduate Medical School, New York 16, N. Y. Tuition is $250.

IATROGENIC HEART DISEASE

(Continued from Page 442)

On examination, her heart action was quiet, easy and regular at 80 beats per minute and her blood pressure, which was 170 on first reclining, settled down to 136/70.

Summary

This case history is typical of a woman who was badly frightened when the doctor told her she had serious heart disease and advised her to be careful.

Even though no serious heart disease was found, she continued to be fearful and needed repeated examinations and reassurance to keep her going.

Woman's Auxiliary

RAMSEY AUXILIARY ELECTS OFFICERS

Mrs. L. T. Simons

The regular luncheon meeting of the Woman's Auxiliary to the Ramsey County Medical Society was held on April 25, 1955, in the Commodore Hotel. Members accepted the slate of officers presented by the nominating committee, of which Mrs. Harvey O. Beek was chairman. Officers for the coming year are:

President	Mrs. H. O. Peterson
President-Elect	Mrs. C. W. Froats
First Vice President	Mrs. Rodney F. Sturley
Second Vice President	Mrs. H. J. Wolff
Third Vice President	Mrs. A. E. Ritt
Recording Secretary	Mrs. J. P. Medelman
Corresponding Secretary	Mrs. S. M. Loken
Treasurer	Mrs. O. I. Sohlberg

Mrs. W. P. Gardner, retiring president, called a board meeting on May 2. Committee reports were given.

Mrs. H. O. Peterson entertained eighteen new and prospective auxiliary members at a get-acquainted coffee party in her home on May 8. Interests and hobbies were discussed.

The annual luncheon meeting was held on May 16 at the University Club. Mrs. W. P. Gardner announced the allocation of funds as approved by the members:

One gift of $500 to the American Medical Education Foundation, designated for the University of Minnesota.

Two gifts of $100 each to University of Minnesota medical students, subject to approval of the Dean of the Medical School.

One gift of $150 to the Hallie Q. Brown settlement house, $150 to the Neighborhood Settlement house to be used to send children to camp.

Mrs. Gardner introduced the new officers to the group and then presented the flower-bedecked gavel to Mrs. H. O. Peterson, who accepted her new responsibility very graciously. The auxiliary members gave a rising vote of thanks to Mrs. Gardner for her fine work during the past year.

Mrs. S. M. Loken, program chairman, presented Mr. Otto Dallman, sculptor, who gave a very interesting demonstration using plaster, granite and clay as media of expression.

In Memoriam

JOHN D. B. GALLOWAY

Dr. John D. B. Galloway, orthopedic surgeon of Minneapolis, died May 4, 1955. He was forty-seven years old.

Dr. Galloway was born in Lewistown, Pa. He attended Cornell University and received his medical degree from Temple University. In 1938, he received a degree of M.S. in orthopedic surgery at the University of Minnesota, having entered the Mayo Foundation as a fellow in orthopedic surgery in 1936.

From 1942 to 1946 Dr. Galloway served in the army. He was chief surgeon at Shriners' Hospital for Crippled Children and was a member of the staffs at Swedish and Lutheran Deaconess Hospitals and of the visiting staff at St. Barnabas hospital. He was attending orthopedic surgeon at General Hospital, Jones-Harrison home and Ebenezer home. He was a clinical assistant in orthopedic surgery at the University of Minnesota.

Dr. Galloway was a member of the American Academy of Orthopedic Surgeons, American College of Surgeons, American Medical Association, Minnesota State Medical Association, Hennepin County Medical Society, Clinical Orthopedic Society, International College of Surgeons and Minnesota Dakota Orthopedic Club. He was a diplomate of the American Board of Orthopedic Surgery.

Dr. Galloway was also a member of Sigma Chi and of St. Luke's Lutheran Church in St. Louis Park.

He is survived by his wife, Gertrude; his daughter, Jane, and his mother, Mrs. Willa B. Galloway, Chester, Pennsylvania.

WILLIAM ANTHONY MILLER

Dr. William A. Miller, born in Appleton, Wisconsin, on July 30, 1872, died in New York Mills, Minnesota, June 1, 1955, after nearly sixty years in the medical profession.

Dr. Miller graduated from the Kansas City Medical College in 1896. He then went to New York Mills and became an active member in many community activities. In February, 1953, a banquet was held by the Lions in New York Mills to honor Dr. Miller for his lifetime of service. He was a member of the school board and served on the Board of Health, as Deputy Coroner, as an examiner for selective service, and participated in many bond drives during both world wars.

He was a life member of the Minnesota State Medical Association, the American Medical Association and had been a member of the Park Region District and County Medical Society since its inception. He was on the staff of St. James Hospital in Perham.

Dr. Miller is survived by his wife, Jessie; a sister, Mrs. F. G. Brabec, Perham; a son, Maurice, New York Mills; two daughters, Mrs. Oscar A. Carlson and Mrs. Eino Kyllonen, both of Minneapolis, and six grandchildren.

Of General Interest

The award of merit of the Ohio Valley Proctologic Society has been given to **Dr. Louis A. Buie,** senior consultant in the section of proctology at the Mayo Clinic, Rochester. The award was made during the recent annual meeting of the American Proctologic Society in New York City.

* * *

Dr. Merrill D. Chesler, Minneapolis, was the principal speaker at a meeting of the Blue Earth Valley Medical Society at Madelia on May 19. He discussed "Burns—Skin Grafts and Flaps" in regard to plastic and reconstructive surgery.

* * *

Dr. J. A. Abullarade, Cokato, staff member of the Cokato Hospital since November, 1954, reported on May 8 at Fort Sam Houston, San Antonio, Texas, for service in the armed forces. He entered the Army as a captain in the medical corps.

* * *

A paper entitled "Osteoporosis and Compression Fractures from Prolonged Cortisone and Corticotropin Therapy" was presented by **Dr. William Sawyer Eisenstadt** and **Dr. Ephraim B. Cohen,** Minneapolis, at a meeting of the American College of Allergists at Chicago, Illinois, on April 28.

* * *

Dr. and Mrs. L. Kenneth Onsgard, Houston, received congratulations from over 200 friends at a reception in Houston on April 24 in honor of their twenty-fifth wedding anniversary. They were married on April 20, 1930.

* * *

Dr. Howard M. Frykman, Minneapolis, was elected to fellowship in the American Proctologic Society at its annual meeting in New York City June 1 to 4. Dr. Frykman presented a paper on "Abdominal Proctopexy and Primary Sigmoid Resection for Rectal Procidentia," for which he received the Piedmont Award for the best scientific paper of the meeting.

* * *

Dr. Burton C. Ford, Marshall, was the principal speaker at graduation exercises at the School of Vocational Nursing, Rochester, on May 1. Dr. Ford is a representative of the Minnesota State Medical Association on the Minnesota Board of Nursing.

* * *

"Management of Strabismus" was the title of a paper presented by **Dr. Richard C. Horns,** Minneapolis, at a meeting of the North Dakota Academy of Ophthalmology and Otolaryngology at Bismarck, North Dakota, on May 2. The meeting was held in conjunction with the North Dakota State Medical Association annual meeting.

* * *

Dr. Clyde A. Undine, Minneapolis, attended a meeting of the American College of Physicians in Philadelphia, Pennsylvania, in April.

Dr. David F. Struxness, Glencoe, reported at the Great Lakes Naval Training Center on May 3 to begin a tour of duty with the Navy. He holds the rank of lieutenant (jg).

* * *

Among the speakers at a meeting of the McLeod County Medical Society at Hutchinson on April 20 were **Dr. Erling S. Platou** and **Dr. Albert J. Schroeder,** Minneapolis, who discussed "Abdominal Pain in Childhood."

* * *

After a year of practice at Heron Lake, in association with **Dr. Richard W. Nicholson, Dr. A. R. Robbins** announced early in May that he was discontinuing his practice there and was moving to the east coast about the middle of the month. At the time he stated that he had no definite commitments as to where he planned to locate.

* * *

Dr. B. W. Kusske, Minneapolis, was the winner of an automatic skillet for which the Paul Revere Life Insurance Company held a drawing at its booth at the annual meeting of the Minnesota State Medical Association in Minneapolis.

* * *

Dr. Walter A. Fansler, Minneapolis, was a guest speaker at a meeting of the Texas Medical Association at Fort Worth, Texas, April 25 to 27. Besides participating in a round-table discussion on "The Treatment of Hemorrhoids and Fissures," he presented two papers on "The Cause and Care of Extensive Rectal Fistula" and "Changing Concepts in the Treatment of Diverticulosis and Diverticulitis."

* * *

Principal speaker at a meeting of the Business and Professional Women's Club at Mankato on April 20 was **Dr. Burton P. Grimes,** superintendent of the St. Peter State Hospital, St. Peter. Dr. Grimes discussed reasons for mental illness and methods of treating patients.

* * *

Dr. John M. Wolff of the Duluth Clinic, Duluth, attended a postgraduate course in internal medicine sponsored by the American College of Physicians at Rochester May 9 to 11.

* * *

"The Dermatologist's Role in the Treatment of Skin Cancer" was the title of a paper presented by **Dr. Henry E. Michelson,** Minneapolis, at a meeting of the Illinois State Medical Society on May 19.

* * *

Dr. Frank J. Hill, former Minneapolis health commissioner, has been named director of the Monroe County (Florida) Health Department. Previously he was director of the Gadsden and Liberty counties health unit.

Dr. Paul G. Boman and **Dr. Frank J. Hirschboeck** of the Duluth Clinic, Duluth, were among those attending the annual meeting of the American College of Physicians in Philadelphia, Pennsylvania, during the last week of April.

* * *

Dr. and Mrs. Duane R. Ausman, St. Paul, left on May 9 for a five-week tour of Europe. While in Switzerland, Dr. Ausman attended a meeting of the International College of Surgeons at Geneva, May 23 to 26.

* * *

Principal speaker at a meeting of the Kiwanis Club at Willmar on April 12 was **Dr. F. T. Sorum** of the Willmar State Hospital. He spoke on "Child of Today, Citizen of Tomorrow."

* * *

Dr. Mario M. Fischer, Duluth, director of health for Duluth and St. Louis County, spoke on "Polio Vaccine" at a meeting of the Parent-Teacher Association in Hermantown on April 20.

* * *

Dr. Irving C. Bernstein, Minneapolis, clinical instructor in the department of psychiatry and neurology at the University of Minnesota, presented a paper on "The Psychiatric Aspects of Dysmenorrhea" at a meeting of the North Dakota State Medical Association in Bismarck, North Dakota, on May 3. On the previous day he spoke on "Psychosomatic Problems in Obstetrics and Gynecology" at a meeting of the North Dakota State Sociey of Obstetricians and Gynecologists.

* * *

Dr. and Mrs. B. J. Cronwell, Austin, attended the annual meeting of the American College of Physicians in Philadelphia, Pennsylvania, during the last week of April. It was their twentieth annual trip to the convention.

* * *

The Minnesota Heart Association awarded a one-year fellowship to **Dr. J. Leo Wright,** Rochester, at a meeting of the executive committee in St. Paul on May 26. Dr. Wright, a fellow of the Mayo Clinic and formerly a fellow of the National Heart Institute, will be given financial support for heart research beginning July 1, 1955. A graduate of the New York University Medical School, he was the recipient of a Ford Award in anatomy in 1950.

* * *

Dr. S. A. Whitson, Albert Lea, was the principal speaker at a meeting of the graduate nurses at Naeve Hospital, Albert Lea, on April 27. Dr. Whitson discussed the subject of diabetes.

* * *

Dr. William G. Strobel of the Duluth Clinic, Duluth, attended the annual meeting of the American Proctologic Society in New York City June 1 to 4.

* * *

After serving for thirty-nine years on his local school board, **Dr. Benedik Melby,** Blooming Prairie, announced in late April that he was retiring from the board. Dr. Melby, who began medical practice in the area in 1903, has served continuously on the school board since he was first elected to it in 1916.

* * *

Dr. Maurice B. Visscher, head of the physiology department at the University of Minnesota, has been named winner of the 1955 American Cancer Society medal for Minnesota. The medal, which was awarded for "important contributions to the control of cancer," was presented to Dr. Visscher at a meeting of the Minnesota division board of directors in Minneapolis on May 6. Previous medal winners in Minnesota have included **Dr. Owen H. Wangensteen, Dr. John J. Bittner, Dr. Arthur Kirschbaum, Dr. E. T. Bell** and **Dr. Arthur H. Wells.**

* * *

Dr. Henry F. Helmholz, Rochester, was the principal speaker at a dinner meeting for delegates from Parent-Teacher Associations in Hennepin and Anoka counties at Richfield (Minneapolis suburb) on May 5. Dr. Helmholz, who is head of the pediatrics section at the Mayo Clinic and vice president of the Minnesota Congress of Parents and Teachers, spoke on the subject, "Health and Community."

* * *

Dr. Michael Kosiak, a native of Chisholm, has been awarded a three-year fellowship grant by the National Foundation for Infantile Paralysis for a special study of physical medicine and rehabilitation. He will begin his work July 1 at the Mayo Memorial Center at the University of Minnesota.

* * *

Dr. Vernon A. Harrington, Duluth, attended the American College of Physicians postgraduate course on "Early Detection and Prevention of Disease," which was conducted in Philadelphia in April. Dr. Harrington is on the internal medicine staff of the Duluth Clinic.

* * *

Guest speaker at a meeting of the Business and Professional Women's Club in Hutchinson on April 14 was **Dr. Carl Bretzke,** Hutchinson, who showed a motion picture on cancer and discussed the importance of early detection of cancer.

* * *

Dr. Charles L. Steinberg, chief of staff at Children's Hospital, St. Paul, spoke on "Progress in Pediatrics—Recent and Anticipated" at the annual meeting of the hospital's association in St. Paul on April 25.

* * *

Main speaker at a meeting of the Parent-Teacher Association at Kenyon on April 18 was **Dr. Ezra Bridge,** medical director of Mineral Springs Sanatorium, Cannon Falls. Dr. Bridge discussed the Mantoux test.

* * *

The annual John W. Bell Tuberculosis Lecture was presented by **Dr. Donald L. Paulson,** Dallas, Texas, at a meeting of the Hennepin County Medical Society in Minneapolis on May 2. Dr. Paulson, a graduate of the University of Minnesota in 1937, is associate professor of surgery at Southwestern Medical School in Dallas. His talk, given in honor of the late **Dr. John W. Bell,** was concerned primarily with the diagnosis and treatment of pulmonary nodules.

* * *

At the annual meeting of the American College of Allergists in Chicago April 28 to 30, **Dr. Albert V. Stoesser,** Minneapolis, served as chairman of the pediatric session and as a panel member in an allergy clinical conference. During the pediatric session **Dr. Lloyd S. Nelson** of Minneapolis presented a paper entitled, "Anaphylaxis in Man—A Serious Problem."

NEW LOCATIONS

Dr. James H. Reinhardt has returned from service in the Air Force and has reopened his offices in Alexandria, where he practiced for four months before entering military service in 1953.

* * *

Dr. Rosemary R. Frear has opened an office for the practice of medicine at Brooten. She has been living near Brooten for several years.

* * *

Dr. Luther H. Zick has moved from Grand Rapids, where he practiced since 1953, to Benton Harbor, Michigan, where he is limiting his practice to surgery.

* * *

Dr. Warren W. Haesly has become associated in practice with Dr. W. O. Finkelnburg in Winona. Dr. Haesly recently completed two years of service in the Air Force. Previously he practiced at Wykoff and Rushford.

* * *

Dr. Dennis E. Lofstrom has begun practice at Pine River, where he is associated with the Pine River Clinic.

* * *

Dr. Raymond C. Magnuson has returned to Cambridge after two years of service in the Army and has reopened his offices at the Cambridge Clinic.

* * *

Dr. Donald Halverson has begun practice at Winnebago, where he is associated with **Dr. John L. Mills.**

* * *

Dr. B. Niles Batdorf has moved from Good Thunder, where he practiced for several years, to Mankato, where he has opened offices in the Doctors' Building.

* * *

Dr. John A. Bolz has returned to Grand Rapids and has opened new offices for the practice of medicine. He recently completed two years of service in the Army.

MINNESOTA BLUE SHIELD-BLUE CROSS

Blue Cross

During the month of March, 1955, 5,164 contracts representing 12,743 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of March 31, 1955 to 1,006,685 participant subscribers.

The year 1955 has had a healthier start for Minnesota Blue Cross subscribers as compared to the same three-month period last year, according to statistics just released by the Blue Cross office.

The analysis just completed reveals that 434 persons per 1,000 contracts were hospitalized during the first three months of 1955 as compared to 454 per 1,000 contracts during the same period in 1954.

The decrease in the number of subscribers using hospital care was attributed to a decline in the number of respiratory conditions such as tonsillectomies, adenoidectomies, influenza, pneumonia and others.

The study also pointed out that maternity cases among Blue Cross subscribers decreased from 74 cases per 1,000 contracts during the first three months of 1954 to 71 cases per 1,000 contracts during the same period in 1955.

The total cost for the above cases was higher in proportion to last year. The total benefits provided for Minnesota Blue Cross subscribers for the first three months of 1955 amounted to $4,861,977.24 as compared to $4,450,115.85 during the first three months of 1954 or an increase of $411,861.39.

Blue Shield

In the Blue Shield statistical data of the first three months of 1955, there are to be found a number of facts of interest to physicians. Among them is the total number of Blue Shield payments made, and the increase in the number of services paid as compared with the same data in the identical three-month period in each of the prior two years. These comparisons are worthy of a more detailed consideration.

During the first quarter of 1953, Blue Shield allowances were provided for 37,354 services with a total payment of $1,088,849. In the same quarter of 1954, 38,879 services, for total payments of $1,192,317 were made. In the identical period, the first three months of 1955, 45,231 services provided by doctors were paid in the amount of $1,371,667.

In comparing these data for the first quarter in the years 1953 to 1955, it can readily be seen that payments to doctors are 25.9 per cent greater for the first three months of 1955 than during the first quarter of 1953. Also, the number of services during the same three-month period in 1955 was more than 6,000 greater than during the same period in 1953. This represents an increase of 21 per cent. These figures also show that there has been an increase in the average payment per service.

With the constant increase in payments noted each year, it is apparent that if the trend in the first quarter is continued, Blue Shield payments to doctors of medicine for services rendered Blue Shield subscribers will strike an all time high for the year 1955.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

Books Received for Review

THE MANAGEMENT OF OBSTETRIC DIFFICULTIES. Paul Titus. Revised by J. Robert Willson, M.D., M.S., Professor of Obstetrics and Gynecology, Temple University School of Medicine; Head of the Department of Obstetrics and Gynecology, Temple University Hospital; Consultant in Obstetrics and Gynecology and Chief of the First Service, The 737 pages. Illus. Price $12.50, cloth. St. Louis: C. V. Mosby Co., 1955.

SHOULD THE PATIENT KNOW THE TRUTH? A response of physicians, nurses, clergymen and lawyers. Edited by Samuel Standard, M.D., and Helmuth Nathan, M.D. 160 pages. Price $3.00, cloth bound, $2.00, paper bound. New York: Springer Publishing Co., 1955.

PERINATAL MORTALITY IN NEW YORK CITY. Responsible Factors. Analyzed and reported by Schuyler G. Kohl, M.S., M.D., Dr. P. H. 112 pages. Price $2.50, cloth. Cambridge, Mass.: Harvard University Press, for the Commonwealth Fund, 1955.

THE MAYO CLINIC—Second Edition. Lucy Wilder 69 pages. Illus. Price $3.75, cloth. Springfield, Illinois: Charles C Thomas, 1955.

MIDWIFERY—Ninth Edition. By Ten Teachers, under the direction of Frederick W. Roques, M.D., M. Chir., F.R.C.S., F.R.C.O.G. Edited by Frederick W. Roques, John Beattie and Joseph Wrigley. 607 pages. Illus. Price $7.00, cloth. Baltimore: Williams & Wilkins Co., 1955.

THE BIOLOGIC EFFECTS OF TOBACCO. With Emphasis on the Clinical and Experimental Aspects. Edited by Ernest L. Wynder, M.D., Head, Section of Epidemiology, and Associate, Sloan-Kettering Institute for Cancer Research. 215 pages. Price $4.50, cloth. Boston: Little, Brown & Co. 1955.

VIRAL HEPATITIS. Clinical, Laboratory and Public Health Aspects. Heinz F. Eichenwald, M.D., Chief, Hepatitis Investigations Unit, Epidemiology Branch, Communicable Disease Center. United States Department of Health, Education and Welfare publication. 57 pages. Price 55c, paper cover. Washington, D. C.: Superintendent of Documents, U. S. Printing Office, 1955.

PROGRESS IN CLINICAL SURGERY. A Symposium edited by Rodney Smith, M.S., F.R.C.S., Surgeon, St. George's Hospital, London; Surgeon, Victoria Hospital for Children; Consultant Surgeon, Wimbledon Hospital; Late Hunterian Professor R.C.S. 414 pages. 112 illustrations. Price $7.50, cloth. Boston: Little, Brown & Company, 1954.

This book proved to be so worth while that the reviewer decided to study it in detail. The volume presupposes that the reader has a knowledge of surgery and is either actively practicing or intends to practice surgery. Its primary purpose is to bring the reader up to date on the developments in the ten years since World War II in the various fields of surgery including some of the subspecialties. Each of the twenty contributors is an outstanding and well-known British surgeon in his particular field. While the book originates from England, the point of view is essentially universal and, with few exceptions, expresses well the point of view of surgeons in the United States. Space has not been devoted to some of the common subjects in the field of general surgery and each individual reader will find some subject omitted which he would like to have had included. The thirty chapters are brief and to the point, for the most part emphasizing surgical indications and surgical treatment. There are, however, good reviews of the various diagnostic procedures that have become available in recent years. The surgical statistics used are up to date. A few of the more recently developed surgical procedures are beautifully illustrated and are given in a fair amount of detail.

While this volume will prove to be particularly useful to the young surgeon preparing for his American Board of Surgery examination, the book should also prove to be helpful to the older surgeon in keeping abreast of the modern point of view in a diverse and ever-expanding field of surgery. Sufficient references are included with each chapter to substantiate the point of view as well as supply up-to-date material for those who wish to pursue the individual subjects in greater detail. The title, Progress in Clinical Surgery, is appropriate in describing the material the reader can expect to find in this small but carefully written text. It should be emphasized that this is not a yearbook which merely would contain a condensation of recent papers in the surgical literature.

G.J.S.

THE PHYSICIAN IN ATOMIC DEFENSE. Atomic Principles, Biologic Reaction, and Organization for Medical Defense. By Thad P. Sears, M.D. 308 pages. Illus. Price $6.00. Chicago: Year Book Publishers, 1953.

This book is of value to anyone who desires an elementary knowledge of atomic energy, as well as to the physician who might serve as a member of a civil defense group. I believe it would be helpful for all physicians to read it.

J.M.R.

ENDOCRINE TREATMENT IN GENERAL PRACTICE. Edited by Max A. Goldzieher, M.D., and Joseph W. Goldzieher, M.D. 474 pages. Prices $8.00. New York: Springer Publishing Co., 1953.

As indicated in its title, this book serves as a useful review to the man in general practice in the recognition and management of some of the more common endocrine disorders.

The book presents the treatment of endocrine disorders in an orderly and systematic fashion which would enable the practitioner to follow directions without much difficulty. However, it is not a textbook for students or specialists in the field of endocrinology.

John G. Fee, M.D.

STATISTICS FOR MEDICAL STUDENTS AND INVESTIGATORS IN THE CLINICAL AND BIOLOGICAL SCIENCES. By Frederick J. Moore, M.D., Associate Professor of Experimental Medicine, University of California School of Medicine, and Frank B. Cramer, B.A., Research Fellow, and Robert G. Knowles, M.S., Research Associate, Department of Experimental Medicine, University of Southern California School of Medicine. 113 pages. Price $3.25. New York: The Blakiston Co., 1951.

Consideration of the monograph, containing 113 pages, a preface, five chapters, three appendices, eleven figures, sixteen tables and innumerable equations, has resulted in the following conclusions preceded by some observations.

Observations.—Most doctors[1] do not use mathematics in daily practice and in general are wary of and shy away from anything containing arrangements of numbers.[2]

Only recently has there been any attempt to introduce a course in statistics into the medical curriculum.[3]

Conclusions.[4]—The lack of an ability to critically appraise medical statistics is an almost universal failing among most doctors (old grads, that is).

The actual opportunity and need for original compilation and appraisal of statistical material is rare for the doctor in practice, whereas those in environments "significant" enough for so doing have available hired help either human or electronic[5] for this purpose.

This book bridges the "fiducial limits" established by the preceding conclusions in a brief and concise way. For us, it made rigorous reading and difficult digesting, falling somewhere between somethings that come back to us as if from a dream. I think the somethings were Physics 133 (Optics) Fall, M-W-F, 8 to 9, (Valasek), 3 cr. and Human neurology III, Fall, M-T-W-Th-F, 8 to 9, (Rasmussen), 5 cr.

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1. Excluding some professors, radiologists and ophthalmologists.
2. Excluding certain ciphers endorsed on forms obtained free annually from the Government Printing Office.
3. 100 per cent of a series of two medical students (one Sophomore, one Junior) polled knew the meaning of "skewness" and "kurtosis," showing that contemporary teaching is not in vain.
4. Probably neither "ultimate" nor "valid."
5. Automation rearing its ugly triodes on the medical horizon.

Leo A. Nash, M.D.

PERIPHERAL CIRCULATION IN MAN: A CIBA FOUNDATION SYMPOSIUM. By G. E. W. Wolstenholme, Editor for Ciba Foundation, assisted by Jessie S. Freeman and Joan Etherington. Price, $6.00. Pages 219, 72 illustrations. Boston: Little, Brown & Company, 1954.

This is another excellent Ciba Foundation Symposium held in London in May, 1953. It contains a group of papers on various peripheral vascular problems. They include a survey of available methods of measuring human blood flow, the response of peripheral circulation with exposure to cold, and a group of papers which has to do with the neurogenic control of circulation. They are all excellent presentations. Probably the most important part of the symposium is the discussion of problems which follows each group of papers.

This book is an excellent review of the subject and would be of particular interest to individuals studying the physiological problem of peripheral vascular circulation in man.

C.V.K.

RHEUMATIC FEVER . . . A symposium held at the University of Minnesota, November 29, 30, and December 1, 1951, under the sponsorship of the Minnesota Heart Association. Edited by Lewis Thomas, M.D. 349 pages. Illus. Price $10.00. Minneapolis: University of Minnesota Press, 1952.

This book consists of publication of the articles presented at a rheumatic fever symposium held at the University of Minnesota in 1951. The natural history, etiology, pathology, diagnosis and treatment are discussed in detail. The sequence of articles has been well arranged and gives an impression of continuity which is unusual for books of this kind. This will be a valuable book for anyone who is called upon to diagnose and treat rheumatic fever.

Robert Lindell, M.D.

PROGRAM FOR PROFESSIONAL PERSONNEL IN NATIONAL EMERGENCIES

(Continued from Page 443)

gineers, nurses, dentists, and others with training and experience in the field of health and medicine to serve with maximum effectiveness in a national emergency.

Maintaining an active-duty health organization large enough to deal with great national emergencies is, of course, out of the question. Our active duty forces must be kept strong—we must also have a large reserve force that can be mobilized when and if a major national crisis develops.

That, in essence, is the basis for the announced decision to expand and strengthen the Commissioned Reserve of the Public Health Service.

The Public Health Service needs physicians, nurses, sanitary engineers, dentists, for periods of active duty during great national emergencies.

Expansion of the emergency reserve provides an opportunity for qualified people engaged in or interested in public health to arrange in advance to serve with the Nation's principal health agency in a national emergency.

Address inquiries and applications to: Surgeon General, U. S. Public Health Service, Washington 25, D. C.

Original Contributions

Hysterectomies

A Study of 762 Cases

CHARLES H. McKENZIE, M.D.
Minneapolis, Minnesota

A STUDY of the hysterectomies done by the surgical staff of Asbury Methodist Hospital between January 1, 1935, and December 31, 1945—eleven years—was undertaken for the following reasons:

1. All the surgeons were willing to co-operate in the follow-up of these cases; such follow-up might add to our knowledge of the fate of the cervix in supra-cervical hysterectomies.
2. The morbidity before and after the use of sulfa drugs might be established.
3. The hospital stay and morbidity before and after the era of early ambulation might be shown.
4. The type of surgery and the complications found at a good general hospital during the decade could be compared.

The charts of all patients on whom any type of hysterectomy was performed during these eleven years were studied. Cesarean hysterectomies are not included. Many charts are incomplete in various details, e.g., on seven charts the age of the patient is not given. Consequently the figures on the tables to be presented do not always give the total of 762.

The number of hysterectomies done by years is as follows:

1935	53
1936	63
1937	63
1938	71
1939	51
1940	70
1941	65
1942	63
1943	71
1944	103
1945	89
	762

The variation in the number of operations done in each month for the years 1935-1945 is as follows:

	Hysterectomies
January	76—Most
February	64
March	70
April	76—Most
May	68
June	73
July	54
August	72
September	52
October	57
November	61
December	39—Least
	762

Types of Operations

Before proceeding further, the types of operations performed might well be classified. The most common approach has been, of course abdominable. However, fifty-one vaginal operations were done. The abdominal operations have been classified according to whether the cervix and/or ovaries were removed, as follows:

A = Cervix and one or both ovaries left in.
B = Cervix only left—both ovaries removed.
C = One ovary left in—cervix out.
D = Cervix, tubes, both ovaries removed—panhysterosalpingoöphorectomy.
V = Vaginal hysterectomy.

By far the greater number of operations were supracervical hysterectomies:—655 (86 per cent). Only 56 (7 per cent) were total hysterectomies in which the cervix was removed. The remainder — fifty-one — were vaginal hysterectomies. The accompanying table shows the types of operations according to the age of the patients.

CLASSIFICATION BY TYPE OF OPERATION AND AGE OF PATIENT

Age	A	B	C	D	V	*Totals*
20-29	20	2	1			23
30-39	185	21	5	8	5	224
40-49	277	51	9	15	13	365
50-59	62	22	2	8	16	110
60-69	4	2	1	6	16	29
over 70	1	1		1	1	4
not given	7					7
	556	99	18	38	51	762

Supracervical hysterectomies (A and B)—655 (86 per cent)
Bilateral oophorectomies (B and D)—137

A study of 762 hysterectomies performed at Asbury Methodist Hospital, 1935 through 1945 (follow-up on 506).

Presented before the Minneapolis Academy of Medicine, December 21, 1953.

Presented before the Minnesota State Obstetrical-Gynecological Society, April 24, 1954.

It is interesting to note also that the number of vaginal operations and of total (i.e., cervix removed) abdominal operations remains almost constant from 1935 to 1945 as follows:

	1935	1936	1937	1938	1939	1940
Vaginal	3	6	3	3	3	4
Total	4	5	5	5	4	5

	1941	1942	1943	1944	1945	
Vaginal	3	7	4	10	5 =	51
Total	7	5	5	10	1 =	56

The preference for the supracervical operation instead of the total removal may be found in the discussion of complications and mortality. Recently, the type of 300 consecutive hysterectomies performed prior to June 30, 1953, was tabulated as follows:

Supracervical	181—60.3%	
Total abdominal	85—28.3%	39.6%
Vaginal	34—11.3%	
	300	

Age of Patients

The ages of these patients are shown on the following charts. Note: Seven charts had no record of the patients' ages and are, therefore, omitted, leaving 755 for the following charts.

By decades, the number of operations were as follows:

Age	*Operations*
20-29	23
30-39	224
40-49	365
50-59	110
60-69	29
Over 70	4
	755

The youngest patient was twenty-two, the oldest seventy-nine; the greatest number of operations, forty-five, were done on patients at the age of forty years.

The age at which bilateral oöphorectomies should be done electively at hysterectomy is always a matter of judgment and experience. The following table shows the ages in this group:

Age		*Per Cent of Total in Age Group*
20-29	2	8.7%
30-39	29	12.9%
40-49	66	18 %
50-59	30	27.2%
60-69	8	27.6%
Over 70	2	50 %

Vaginal hysterectomy was performed in only fifty-one cases (6.7 per cent). The age groups of this operation are as follows:

Age	*Number*	*Per Cent in Age Group*
20-29	0	0.0%
30-39	5	2.2%
40-49	13	3.5%
50-59	16	14.5%
60-69	16	55.2%
Over 70	1	25 %

The indications will be discussed later.

Anesthesia

During the decade 1935-1945 anesthesia underwent remarkable changes, and a study of the kinds of anesthesia for the hysterectomies done during this period is interesting.

Anesthesias are grouped as general, spinal and local, according to the type used at the beginning of the operation. General anesthesias include nitrous oxide, ethylene and cyclopropane plus oxygen, used chiefly to induce anesthesia, followed by ether. Occasionally in the 1940's pentothal was used to induce anesthesia. Curare was used a few times to produce relaxation, once with an unfortunate result. It was customary also to switch from one type of gas anesthesia to another, so that no table of the various types of general anesthesia can be derived from the records.

Spinal anesthesia was still popular, and local anesthesia was occasionally used. However, both of these anesthesias were frequently supplemented by general anesthetics. No local anesthesia was tried after 1940.

The following table gives the number of operations under the various types of anesthesia.

		Supplemental	
General	516		
Spinal	229	79	33⅓%
Local	10	9	90%

The number of patients requiring supplemental general anesthesia explains the popular demand for the easy induction by pentothal induction.

It is doubtful if the anesthesia contributed in any way to complications, but the following table of complications and mortality following operations done under these anesthesias is of interest.

	Operations	*Complications*	*Deaths*
General	516	58 or 11.2%	5 or 1 in 103
Spinal	229	24 or 10.5%	5 or 1 in 46
Local	10	1 or 10 %	0
	755		

It is interesting also to note that none of the vaginal hysterectomies was done under local anesthesia during this decade, twenty-seven being done under general anesthesia, twenty-four under spinal anesthesia.

Although spinal anesthesia is rarely used now, it will be of interest to note the kinds and amounts of the various drugs used.

Metycaine was used in only nine cases in doses varying from 100 to 150 mg. Procaine was used in doses varying from 120 to 400 mg., commonest doses being 150 in fifty-six cases, 200 mg. in thirty cases.

Pontocaine was most popular, being used in 120 cases in doses varying from 16 to 20 mg. Of these 120 cases, sixty-seven received 20 mg. of pontocaine.

Spinal Anesthetics

Metycaine	9 cases	100-150 mg.
Procaine	56 cases	150 mg.
	30 cases	200 mg.
Pontocaine	67 cases	20 mg.

In quite a few records the dosage of drug used could not be found so that complete tabulation is not possible.

Indications

The indications for the operations performed are derived from the internes' histories, for it is only occasionally during this decade that the surgeon included his indications in the dictated operative record. It is well known that private patients in a private hospital often resent internes' questions. This applies peculiarly to female patients, who often do not wish to give the intern much information concerning bleeding and pelvic pain. Therefore, the indications as tabulated may not be correct and may be incomplete. They are presented, however, as typical of a private hospital records. Frequently more than one indication is given, but no attempt has been made to classify these indications as main or secondary. On many charts no indications were noted.

The following indications are recorded:

Menorrhagia	188
Pelvic pain	144
Menometrorrhagia	124
Pelvic tumor noted by patient	81
Backache	39
Prolapse	25
Dysmenorrhea	20
Post-menopausal bleeding	16
Urinary retention	5
Leukorrhea	3

Of the twenty-three patients in the 20-29 age group, fourteen were operated on for profuse and prolonged bleeding. Of these, ten had dilation and curettage prior to operation.

Prolapse was treated by vaginal hysterectomy in twenty-three of twenty-five cases, twice by supracervical hysterectomy. Of the vaginal hysterectomies for prolapse, eighteen were done after age fifty.

Pathological Findings

The pathological findings in these pelvic operations have been gleaned from (1) the surgeon's dictated operative record and (2) the pathologist's report. In most cases, where both are given, there was agreement, except that many surgeons tended to overestimate the size of myomas and ovarian cysts. The largest myomatous uterus removed weighed 11 pounds. There were some uteri containing small fibromyomata. These the pathologist tended to call "fibroid" uteri and these are listed as such. No definite correlation could be found in the histories between symptoms of menorrhagia and size of myomata.

Prolapse and retroversion are noted in the pathological findings, these conditions being, of course, obtained from the surgeon's operative records.

Many women had multiple pathological pelvic findings. The pathological conditions are noted as follows:

Myomata, or fibromyomata		473
Fibroid uterus		55
Fibrosis uteri		29
		557
Cystic ovary or ovaries		147
Chronic P.I.D. Frozen Pelvis acute P.I.D. and chronic salpingitis		59
Hyperplasia of endometrium		41
Endometrial polyp		40
Multiple adhesions		34
Chronic endocervicitis		15
Endometriosis		4
Tuberculosis - Salpingitis		1
Prolapse of uterus		38
Retroversion		16
Dermoids of the ovary	Age 30-39	3 cases
	Age 40-49	2 cases
	Age 50-59	1 case
		6 cases

Pregnancy was associated with pelvic pathological conditions as follows:

Early Pregnancy	3 cases
Incomplete Abortions	2 cases
Ectopic Pregnancy	2 cases
	7 cases

There were 697 women in the age group 20-50 years; or, pregnancy in 1 per cent in the child-bearing age.

Malignant conditions were found in forty-three hysterectomies as follows:

Carcinoma of the fundus	28 cases
Carcinoma of the cervix	9 cases
Carcinoma of the ovary	4 cases
Carcinoma of the tubes	1 case
Sarcoma of the uterus	1 case
	43 cases

Concomitant Surgery

In 285 cases, the operation performed was confined to the pelvic organs—uterus, tubes and ovaries. In 470 cases, however, or roughly two-thirds, one or more other organs or tissues were repaired or removed as follows:

Appendectomy	274
Repair cystocele and rectocele	85
Dilation and curettage prior to hysterectomy	79
Freeing adhesions	56
Cautery to cervix	17
Repair cervix	14
Hemorrhoidectomy	10
Repair ventral hernia	8
Repair inguinal hernia	3
Lepectomy	3
Breast biopsy	2
Cholecystectomy	2
Hymenotomy	1
Presacral neurectomy	1
Resection of sigmoid for carcinoma	1
Remove cervical polyp	1

In six cases angiomata, and other skin blemishes in other areas of the body were removed while the patient was under anesthesia.

Morbidity

Morbidity is difficult to define, but for purposes of comparison the temperature records were noted in all cases and the number with temperature of 100.4 degrees for any day have been considered as having morbidity. This is a very strict interpretation.

Comparing those with no concomitant surgery and those in which appendectomy was an added procedure, the following is noted:

		Morbidity	*1 day*	*2 or more days*
No concomitant surgery	285	47 or 16.8%	53	185
Appendectomy	274	47 or 17.1%	63	164

It is obvious that appendectomy done at the same time as hysterectomy does not add to morbidity. It was noted, however, that no case in which hemorrhoidectomy was done escaped rise of temperature to 100.4 degrees or above. In general, all the other types of concomitant surgery do not add to morbidity.

Postoperative Complications

The complications noted on the fronts of the hospital charts are given, these complications being severe enough to have caused concern to the surgeon. Possibly a careful review of the progress notes might reveal other minor complications.

The following respiratory complications are then noted:

Pneumonia and atelectasis	19 cases
Pleurisy	3
Pulmonary infarction	2
Bronchitis	2
Upper respiratory infection	1

An attempt was made to correlate respiratory infections according to season of year, but when it was noted that five cases occurred in May, one in February, two in January, one in August, it was evident that no correlation existed.

Also, the cases with respiratory complications with and without concomitant surgery revealed no correlation.

With concomitant surgery	17 in 470 cases 3.6%
Without concomitant surgery	10 in 285 cases 3.5%

Other complications were as follows:

Infected wound and hematoma of wound	23
Urinary tract infection	9
Phlebitis	7
Pelvic abscess	3
Intestinal obstruction	2
Evisceration	2
Atony of bladder	2
Vesico vaginal fistula	1
Hemorrhage from cervix, postoperatively	1
Intra-abdominal-hemorrhage	1
Brachial artery thrombosis	1
Acute dilatation of stomach	1

Again, in these complications, there is no correlation with added surgery.

Hospital Stay

It was thought that after 1940 (1) the use of the sulfa drugs and later penicillin, and (2) early ambulation, might shorten hospital stay. However, this was not evident before 1946.

The shortest stay both before and after 1940 was six days. The longest stay in the period 1935-1940 was seventy-nine days. This patient had a pulmonary abscess and recovered. The longest stay in 1941-1945 was sixty days.

The average stay before 1940 was 15.1 days; after 1940, 14.3 days, a difference of very little significance.

Effect of Sulfa Drugs and Antibiotics

Before 1940 the sulfa drugs were used very infrequently, not more than three or four times in this series. After 1940 sulfa drugs were used occasionally postoperatively and penicillin a few times in inadequate doses according to present standards. In the period 1940-1945 only 113 patients in this series, about 29 per cent, received sulfa drugs and/or penicillin.

A table of morbidity before and after this date and showing the use of sulfa and/or penicillin is interesting.

MORBIDITY BEFORE AND AFTER THE USE OF SULFAS AND PENICILLIN

	1935-40	1940-1945		1935-45
		a*	b*	
Supracervical	86.0%	80.0%	81.0%	83.7%
Total and vaginal	95.7%	87.4%	79.4%	91.5%
All cases	87.7%	81.3%	85.8%	85.2%

a*—without sulfas and/or penicillin.
b*—with sulfas and/or penicillin.

It will be noted that the morbidity for all the hysterectomies over the eleven-year period is 85.2 per cent. Before the use of sulfa the morbidity ran 87.7 per cent. Note, however, that the morbidity when the cervix was removed during this earlier period was 95.7 per cent, whereas the morbidity for supracervical hysterectomy during this same period was only 86 per cent. Perhaps this will explain partially the preference for the subtotal operation.

After 1940 and the more general use of the sulfa drugs morbidity dropped to 85.8 per cent for those in which sulfa was used, and 81.3 per cent when sulfa did not appear indicated. It is in the series in which the cervix was removed that the greatest advance appears, for in this group of twenty-nine patients in which sulfa and/or penicillin was used, the morbidity dropped to 79.4 per cent. Remarkably, also, the morbidity in total hysterectomies done during this period, but where sulfa was not given, also dropped to 87.4 per cent. There is apparently an added safety factor by the general use of sulfa and/or penicillin in the hospital.

Mortality

Ten patients died in the first month after operation.

Ages	
37 years	1
40	1
41	1
45	2
46	2
47	1
48	1
51	1
	10

The causes of death were as follows:

	Year of Death
One died during operation—possibly due to curare	1945
One died of shock 65 hours after operation	1944
Three died on 6th day postoperatively	
—one of generalized infection	1941
—one of cardiac failure	1935
—one of pulmonary embolism	1945
One died on the 7th day, pulmonary embolism	1936
One died on the 13th day of paralytic ileus	1935
One died on the 20th day of pulmonary embolism —9 hours after hemorrhoidectomy	1937
Postoperative day—autopsy	
One died on the 22nd day of acute intestinal obstruction (autopsy)	1936
One died on the 30th day of acute intestinal obstruction	1937

Days after operation	Immediate	6	7	13	20	22	30
Number of deaths	2	2	2	1	1	1	1

Causes	No.
Pulmonary embolism	3
Acute intestional obstruction or paralytic ileus	3
Shock	1
Anesthesia	1
Generalized infection	1
Cardiac failure	1

Two autopsies were obtained.

Two died in 1935
Two died in 1936 } 6 in 371 or 1 in 62
Two died in 1937

Longest period without a death—July, 1937 to February, 1941—238 cases.

One died in 1941
One died in 1944 } 4 in 391 or 1 in 98
Two died in 1945

Two deaths followed panhysterectomies, i.e., two deaths in thirty-eight operations (5.2 per cent). Eight deaths followed supracervical hysterectomies: eight in 655 (1.2 per cent).

Follow-up

The ideal follow-up in any such series would be a pelvic examination with vaginal smears and indicated biopsies on all the patients by a single qualified individual. Manifestly this is impossible, so in any follow-up multiple contacts are attempted.

1. Each surgeon was mailed a card with a short résumé of each patient and was requested to supply any follow-up he might have about the patient. The response to this was very prompt and gratifying. Nearly 300 cards were returned within two months.

2. Subsequent admissions to Asbury Hospital of every patient were recorded by the record room. In many cases this record supplemented the surgeon's follow-up.

3. If the surgeon has moved or died, the cards were sent to the surgeon taking over his practice. Very few of these cards were answered.

4. Some surgeons suggested that the referring

doctor be contacted. This brought about ten replies.

5. Any patient, not accounted for as above, and whose name appeared in the phone book was sent a personal questionnaire. Out-of-town patients were also sent this questionnaire. There were thirty-four replies.

6. The Mayo Clinic gave a complete follow-up on one patient who died there of carcinoma of the duodenum.

Using these various means, a follow-up has been obtained on 467 cases. Of these there is a follow-up *for more than one year on 411 cases of supra-cervical hysterectomies.* This does not include carcinoma cases, on which special study is made.

Some report is given on forty-four cases during the first year. Of those followed less than one year the following two cases are of interest:

One patient had a bilateral salpingectomy for acute salpingitis two months after vaginal hysterectomy.

One patient had four laparotomies in the first year for intestinal obstruction.

Of those followed more than one year, the number is as follows:

	No. Cases	
1-2 year follow up	24	
2-3	21	
3-4	29	
4-5	23	
		97
5-6	29	
6-7	18	
7-8	40	
8-9	50	
9-10	40	
10-11	36	
11-15	125	
Over 15	32	
		370
		467

Of these 467 followed, seventeen were reported as dying of the following:

Cardiovascular disease	8
Diabetes	1
Carcinoma of G. I. tract	2
Carcinoma of breast	1
Epilepsy	1
Pernicious anemia	1
Tuberculosis	1
Pneumonia	1
Cancer of ovary (5 years postoperative)	1
	17

Remote Complications and Treatments

Urinary Complications.—There is no record that a ureter was tied or cut at the time of surgery. However, the follow-up reveals urinary complications in seventeen cases. Of these several had numerous cystoscopies. One patient had a nephrectomy, and the history in one other patient suggests that nephrectomy was done.

PELVIC COMPLICATIONS REQUIRING SUBSEQUENT OPERATIONS

Ovarian cysts removed	8 cases	
Bowel obstruction	6 cases	
Ventral hernia repaired	3 cases	
Appendectomy (15 mo.-15 yrs.)	2 cases	
	19 in 467	4%

TREATMENTS TO CERVICAL STUMP

Cautery to cystic and/or eroded cervix	23 cases	5.6%
Polyps removed from cervix	6	
Cervix removed	5	
Bleeding from cervix (cause not given)	3	
Developed carcinoma of cervical stump (Both found within one year of surgery. One patient died in six months; one, treated with radium. is alive and well after seven years.)	2 cases	0.5%
	39 cases	9.5%

Total treatments to cervix: 39 in 411 supravaginal hysterectomies, or 9.5 per cent.

Follow-up on Cases of Malignancy

There were forty-three cases of malignant disease for which hysterectomy was performed:

Carcinoma of fundus	28 cases
Carcinoma of cervix	9
Carcinoma of ovary	4
Carcinoma of tube	1
Sarcoma of uterus	1
	43 cases

Two of these died postoperatively, one from shock, one from septicemia.

Follow-up on the remaining forty-one cases was done by questionnaires to the surgeons, and to the patients, and by reviewing hospital records. Two other sources were used:

1. Dr. McKelvey kindly allowed a cross-matching of the names of all patients with carcinomas of the cervical stump in his records, with this list of patients. None of the Asbury patients were found in his group. (This provides a "negative" follow-up).

2. Dr. Rosenfield of the State Department of Health crossmatched their files. Two cases, not otherwise followed, were found in the death certificates on file at the State Department of Health. In spite of these searches, no follow-up can be found on four patients. Thirty-nine patients with malignant disease however have been followed,

three of them over fifteen years. This group added to the 467 makes a total follow-up of 506 cases in 762 hysterectomies (66 per cent).

The following table shows the follow-up on these thirty-nine cases:

	Fundus No	Yes	Cervix	Ovary	Tube	Saf	
Postoperative mortality.			1	1			2
Died Ca in 1							
Less than 1 year	5	1	3	1		1	11
Less than 2 years	6		1				7
Died other causes							
No recurrence	4 yr.	5 yr.		8 yr.			3
Alive and well							
5-10 yrs.	6	1	3	1	1		12
11-15 yrs.	2	2	1				4
over 15 yrs.	1	2					
5-yr. cures	9	6	4	2	1		22
Per cent 5-year cures	43%	85%	44%	50%	100%	0	51%

These figures, are, of course too few for statistical analysis but possibly may show a trend. Note that radiation to the pelvis for fundal carcinoma before hysterectomy gave a five-year survival rate of 85.7 per cent whereas in those cases not receiving x-ray, only 43 per cent survived five years.

The most remarkable case in this series is one of carcinoma of the cervix. This patient had a dilation and curettage and removal of cervical polyp prior to supracervical hysterectomy. Pathological report showed squamous cell carcinoma of the cervical polyp. Six months later the patient received 2500 mg. radium. She is alive and well in Minneapolis today.

The follow-up of the whole group shows twenty-two survived five years or more; and presuming that the cases unaccounted for have died of carcinoma, the survival rate for five years of the group of malignancies is twenty-two of forty-three (51.1 per cent).

Summary By Surgeons

Forty different surgeons performed these 762 hysterectomies.

16 surgeons performed	a total of	42 operations
18 surgeons performed	a total of	290 operations
6 surgeons performed	a total of	430 operations
		762

470 operations were done by fellows of American College of Surgeons, 195 by general practitioners, and 97 by the obstetrics-gynecology group.

Acknowledgments

I wish to thank the following persons for their co-operation: first, all the surgeons of the staff who replied to the questionnaires and résumés. Second, Dayton Shields, administrator of Asbury Methodist Hospital, who signed the numerous letters used in the follow-up of these cases. Third, the record librarians, particularly Alice Knudtsen, without whose help such a complete follow-up would not have been possible.

Discussion

Paul N. Larson, M.D.—It is evident that Dr. McKenzie has taken much time from his busy private practice to make this interesting survey. The detail and follow-up have brought many revealing facts to light.

The need for consultation in diagnosis and indication for surgery becomes apparent when 144 of 762 hysterectomies are performed for pelvic pain, or when eighty-six are found to be fibrosis uteri or fibroid uterus (not fibromyomata). Out of 762 hysterectomies only one in seven was done by a gynecologist.

Nine cases were performed for carcinoma of the cervix, but no other coincidental carcinomas were found in the remaining cervices removed routinely for benign conditions. One should find carcinomas in at least 2 per cent routine total hysterectomies for benign conditions.

In his follow-up he shows that 0.5 per cent of the sub-total group developed carcinoma in the stump. A total of 10 per cent of the stumps required subsequent surgery for one reason or another. Certainly these complications would make an operator think twice before doing a sub-total.

The morbidity figures in these series are very strict and difficult to compare since most groups use 100.4 for two successive days.

There was a low incidence of pre-operative diagnostic dilation and curettage, but that can be explained on the basis of only one case in seven being performed by a gynecologist.

A survey of this sort has brought many facts to our attention which have not ordinarily been considered. A city-wide study would no doubt demonstrate similar circumstances in all hospitals.

Let us again thank Dr. McKenzie for bringing us this review, for no doubt much good comes from stark realization.

James F. Shandorf, M.D.—I wish to take this opportunity to congratulate Dr. McKenzie on his most interesting paper.

On listening to this fine paper there are several points that impress me. First, I believe there were twenty-three patients in the 20 to 29 age group, and as I recall of this small group only ten had diagnostic curettages prior to surgery. I strongly feel that before any hysterectomy, regardless of age, it should be preceded by a diagnostic curettage.

Secondly, in the pathological summary, I believe there were only four cases of endometriosis. This seems like a very low figure, and one questions whether this entity was recognized as it is today or are we seeing more endometriosis than we did in the years covered by Dr. McKenzie's paper.

(Continued on Page 488)

Indications and Contraindications for Adenotonsillectomy

The Poliomyelitis-Tonsillectomy Problem

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ADENOTONSILLECTOMY continues to be the most frequently performed single surgical operation in Minnesota, as it is elsewhere. Proof of this is readily seen when one examines the financial reports of Blue Shield. Blue Shield records show that payments to physicians and surgeons for adenotonsillectomy exceeds the payments for any other single operation.

In recent years the lay public has been exposed to much publicity in the lay press regarding the possible danger of performing adenotonsillectomy during an epidemic of poliomyelitis. Physicians and surgeons, also, have been bombarded with voluminous medical literature concerning the relation of adenotonsillectomy to poliomyelitis. A review of the indications and contraindications to adenotonsillectomy, therefore, seems timely and desirable.

A few decades ago it was believed by the lay public and surgeons alike that everyone should have his tonsils and adenoids removed. Now-a-days even the domestic magazines warn parents that such is not the case and that there are definite indications for adenotonsillectomy. The operation can be very beneficial to an individual if indications for its performance are definitely present. What are these indications? Foremost in importance, and the most commonly used indication for the operation, I believe, is frequency of upper respiratory infections. When parents bring their youngsters to me with the question, "Should the tonsils and adenoids be removed?" my first question is, "How much trouble has the youngster had?" A helpful way to ascertain the frequency and severity of upper respiratory infections is to find out how much school the youngster has missed because of disabling upper respiratory infections. Frequent disabling attacks of colds, bronchitis, sore throats, tonsillitis, otitis media, cervical adenitis, and quinsy, undoubtedly are the most common indications for the operation. When there is a history of such frequent upper respiratory infections, and when the physical examination reveals deeply embedded, enlarged tonsils, and enlarged adenoids, one can be fairly certain that removal of such tonsils and adenoids will reduce the number of disabling upper respiratory infections.

In examining the tonsils, one should look for certain signs: Are the pillars chronically red? Is there a spotty exudate over the crypts? Also, are the tonsils embedded, such as shown by letter A in Figure 1, and can pus or exudate be expressed from the crypts by pressure on the anterior pillars? If the answer to these questions is yes, and if there is a history of frequent upper respiratory infections, I believe a diagnosis of chronic tonsillitis is justified. One can visualize large embedded tonsils if the anterior pillars bulge forward markedly when the individual gags. Also the examiner can get a good idea of how large and embedded the tonsils are by using a head mirror and reflected light, or a head light, and with a tongue depressor in each hand, exert gentle pressure on the anterior pillars. This will squeeze the tonsil to the midline and also express any deep-seated pus in the crypts. Such deep-seated pus would be overlooked by merely looking at the tonsil.

Some authorities[1] emphasize the importance of the way the tonsils are lodged between the palatal arcs and the role of mechanical factors acting on the tonsils. It was noted that in cases in which an assumption of focal infection was correct, as seen from the improvement following tonsillectomy, the tonsils were found to be lodged deeply between the pillars or embedded, as illustrated by letter A in Figure 1. The motions of swallowing were thought, therefore, to have an important role in the production of focal symptoms referable to embedded tonsils. Whereas,

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This paper was awarded the Southern Minnesota Medical Association Medal for best paper presented at annual meeting, September 13, 1954.

when the tonsils are normally situated, the constrictive action of the pillars and tongue during swallowing express the tonsillar content toward the oral cavity (Fig. 1, letter B), deeply seated tonsils are compressed readily in swallowing, with their contents being arrested along lines of least resistance, that is to say, toward the capsule of the tonsil. Special examination, with an opaque media injected into the crypts, seems to bear out these postulates.

When tonsillar contents are arrested deep in the crypts, they are more apt to set up chronic infection and thereby act as a focus of infection. We have, then, a second indication of adenotonsillectomy, namely, a focus of infection. I have found that tonsils which looked quite innocuous on their small exposed surface, but which were found to be deeply embedded, and from which I could express pus from deep in the crypts, were acting as a focus of infection, and were contributing to such conditions as chronic iritis, vasomotor rhinitis, possibly through the mechanism of bacterial allergy, nephritis, rheumatoid arthritis, rheumatic fever, and conditions simulating brucellosis, with weakness, sweats and chills. Gratifying improvement to complete relief have occurred in these conditions following removal of such deeply embedded tonsils.

Carriers of diphtheria can be rendered free of infection by tonsillectomy. It was found in a study[2] of a large group of British soldiers in Italy during the last War that 81.5 per cent were found to be free of infection by the fourth day after tonsillectomy.

Asthmatic individuals with large obstructing tonsils and adenoids can expect benefit by adenotonsillectomy. This benefit occurs as the result of possibly one or two mechanisms: One, removal of the large obstructive adenoids creates a normal nasal airway, so that the nasal mucosa can adsorb the inhalant allergens responsible for the asthma. Similar benefit occurs to adult asthmatics by removal of obstructive nasal polyps. Secondly, removal of large infected tonsils and adenoids eliminates a possible bacterial allergic factor in the cause of the asthma. Even if the lymphoid tissue in the throat becomes hyperplastic again, as it commonly does in allergic individuals after adenotonsillectomy, the operation is justified if this indication is present.

A very important indication for adenotonsillectomy is hearing impairment caused by large obstructive tonsils and adenoids. It has been a common experience to encounter children with impaired hearing who have large obstructive adenoids and dull, retracted eardrums. Complete adenotonsillectomy, bilateral myringotomy, and removal of the tenacious mucoid matter usually found in the mid ears by spot suction, has restored normal hearing to these individuals. The committee on conservation of hearing of the Minnesota State Medical Association has emphasized the importance of thorough and complete adenoidectomy in these cases. Thorough adenoidectomy is possible only when a combination of direct inspection and finger palpation of the nasopharynx is used during the operation.

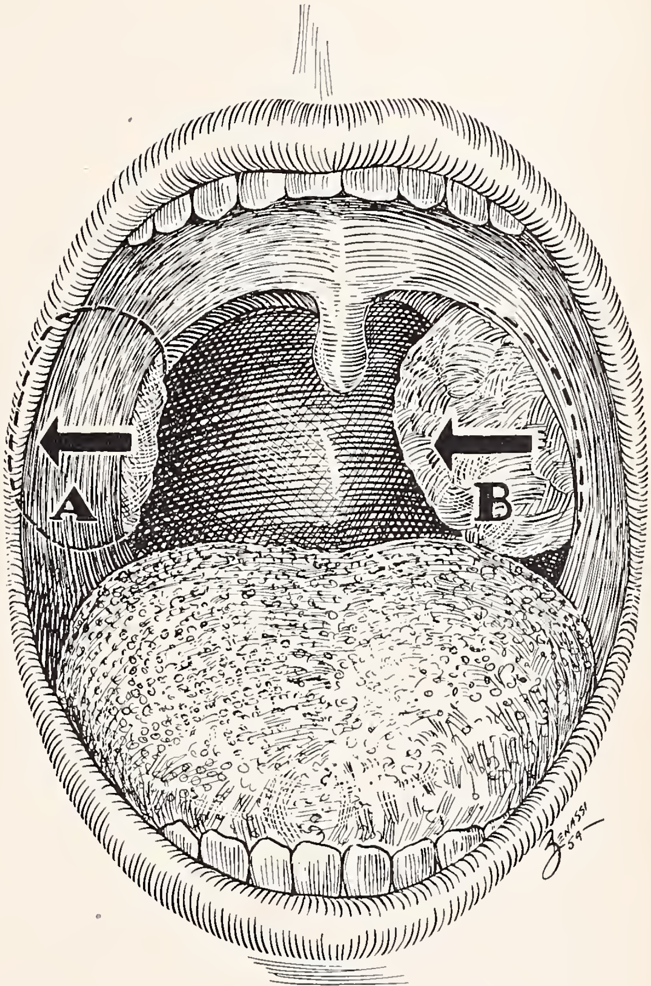

Fig. 1.

A minor indication for adenotonsillectomy is halitosis caused by retained insipissated debris in the crypts of the tonsils and adenoids. Halitosis can be a very important social and economic problem to an individual.

The last indication for adenotonsillectomy that I wish to mention is a common one. I believe that all of us here have seen youngsters who have marked mouth breathing, snoring, restlessness in sleep, irritability, hypochromic anemia, and who are underweight, undernourished, poor eaters, and backward in school, as the result of large tonsils and adenoids. It is very gratifying to the surgeon to hear the parents state that removal of tonsils and adenoids have made an entirely different and better person out of such a youngster.

In regard to contraindications to adenotonsillectomy, I do not believe there are any absolute contraindications, unless, possibly, a severe blood dyscrasia, such as proven hemophilia. The age factor is a relative contraindication. It is inadvisable to remove tonsils and adenoids in a youngster under three years of age, because if it is performed before this age, the lymphoid tissue is most apt to recur and because it is now known[3,4] that the tonsils and adenoids play an important role in the development of natural immunity in the early years of life. I would consider removal of tonsils and adenoids in a child under three years of age only if they are so large that the child is having extreme trouble swallowing and breathing and is having exceedingly frequent severe upper respiratory infections which do not respond to antibiotics or other conservative therapeutic measures. If adenotonsillectomy is performed in such a youngster, I believe the parents should be warned that no matter how thoroughly the tonsils and adenoids are removed, the child may have to have a secondary removal, particularly of nasopharyngeal lymphoid tissue, later on.

Other relative contraindications are presence of an acute respiratory infection; blood dyscrasias, as leukemia and purpura; cardiac decompensation; the period of convalescence after any debilitating disease, and proven enlarged thymus gland in the early years of life.

Lastly, epidemics of contagious or infectious diseases are a contraindication to adenotonsillectomy. It is in this category that I wish to discuss the relation of adenotonsillectomy to poliomyelitis. As stated in the introduction, physicians, surgeons and the lay public alike have been exposed to a great deal of literature and publicity regarding the possible danger of performing adenotonsillectomy during the so-called poliomyelitis season. Doubtless, we, as surgeons, have often postponed the operation more out of fear of being condemned by the public for performing the operation when any poliomyelitis is present in the community than by an actual fear that a danger does exist. This relation of adenotonsillectomy to poliomyelitis is still an unsolved problem in medicine. Surgeons have been, and are, faced with the question, "Should adenotonsillectomy be performed if there is any poliomyelitis in the community?" and, for that matter, "Should the operation be performed during the months when poliomyelitis is most likely to occur?" In an attempt to find some answers to these questions, I shall summarize some of the literature to date and try to reach some conclusions therefrom.

Some surgeons have had actual cases of children developing poliomyelitis within its incubation period following adenotonsillectomy, and yet Harkness[5] had an unique experience wherein he performed adenotonsillectomy very freely during the severe poliomyelitis epidemic in the Davenport, Iowa, area about twenty-five years ago. He did this on the recommendation of Rosenau, who at that time felt that the anaerobic streptococcus was responsible for poliomyelitis, and he performed this surgery particularly on children who had had possible contact with poliomyelitis. Among these numerous tonsillectomized children, Harkness did not have a single case develop poliomyelitis.

McNaught[6] of Stanford University has had a wide experience with the controversial tonsillectomy-poliomyelitis problem. In a personal communication he states that it should be stressed that there is no "poliomyelitis season" and indicated adenotonsillectomy should not be postponed because of the calendar. He does believe that the operation is contraindicated during a local epidemic of poliomyelitis.

It appears that most of the studies concerning the tonsillectomy-poliomyelitis problem have been of a statistical nature. Miller[7] surveyed the 1,229 cases of poliomyelitis which occurred in Los Angeles County in the epidemic year of 1949. His statistical analysis of these cases indicated no clinical or statistical significant deviation between the actual and the expected incidence of poliomyelitis developing in patients recently tonsillectomized, even in the months of July through October.

However, the results of other studies do not agree with Miller's conclusions. Mills[8] has reviewed the literature on the tonsillectomy-poliomyelitis problem and found unmistakable evidence that the attack rate of bulbar poliomyelitis in many epidemics has been found to be significantly higher on recently tonsillectomized patients, and evidence is increasing that bulbar poliomyelitis, rather than the spinal form of the disease, is more likely to occur in patients tonsillectomized at any time than in those with tonsils intact. Mills concludes that adenotonsillectomy should not be performed during an epidemic.

In 1950, Cunning[9] surveyed 36,000 cases of poliomyelitis and 100,000 cases of tonsillectomy compiled over a four-year period. He could find no definite causal relation but he did conclude that adenotonsillectomy, or, for that matter, any elective operation, should not be performed during a poliomyelitis epidemic or any other epidemic, and conversely in the absence of an epidemic, adenotonsillectomy should not be postponed during the summer months merely because poliomyelitis is apt to occur at that time.

Pedersen[10] reviewed 492 cases of poliomyelitis hospitalized in San Francisco during 1941 to 1945. He found that there were almost twice as many deaths from poliomyelitis in patients with tonsils removed as there were in patients with tonsils present. He also found that there was a higher incidence of bulbar and bulbospinal type poliomyelitis in tonsillectomized patients than in nontonsillectomized patients, the ratio being two to one.

In a summary of our own memorable 1946 Minnesota poliomyelitis epidemic, it has been shown that 13 per cent of the bulbar cases had had some nose and throat surgery performed within two to three weeks before the onset of the illness. Such a history was not obtained in the spinal poliomyelitis cases.

A very comprehensive statistical study of the 1946 Minnesota epidemic was recently completed by Anderson[11] and Rondeau. They used 2,669 histories of poliomyelitis in their study. They found that for all age groups 71.4 per cent of the 535 bulbar cases gave a history of tonsillectomy as contrasted with 28.2 per cent of the 936 severe spinal cases. Their studies showed that "if a child who still had his tonsils *in situ* developed recognizable poliomyelitis, the chances were only one out of twelve that the infection would be bulbar, but, if the child had at some time had the tonsils removed, the chances of bulbar involvement were more than one out of three." Furthermore, it is apparent from their studies that the length of time after adenotonsillectomy is not a factor in the high percentage of bulbar infection in tonsillectomized poliomyelitis patients. These findings suggest that it is important that a suitable indication is present before adenotonsillectomy is performed. As an epidemiologist, Anderson suggests an explanation for the controversial results in other statistical studies by saying that the lack of bulbar cases in the particular areas of those other studies may be due to concentration of poliomyelitis in ages before tonsil removal.

Aycock[12] found that poliomyelitis occurring within thirty days after tonsillectomy is predominately bulbar. His studies show that post-tonsillectomy bulbar poliomyelitis occurs almost exclusively during epidemics of the disease, and the onset of symptoms nearly always occurs six to twenty-four days after operation, with the peak at fourteen to sixteen days.

These figures agree closely with the incubation period of poliomyelitis in general, suggesting that the virus may be introduced at the time of operation. Aycock has also pointed out that there are many other possible contributing factors as important as tonsillectomy in increasing the susceptibility to poliomyelitis, both as to frequency and severity. Some of these factors indicate and include pregnancy, over-exertion, inoculations for active immunization, and operations.

Other studies relative to the tonsillectomy-poliomyelitis problem have been experimental in nature. Faber[13] and his associates have found in their laboratories that in order consistently to produce poliomyelitis infection in a divided nerve, it is necessary to apply virus to the central cut end before it is sealed off by blood. They found, experimentally, that application of poliomyelitis virus to the pharynx of cynomolgus monkeys, followed by enucleation of the tonsils, was followed in 100 per cent of cases, after seven to ten days, by primary bulbar paralysis, accompanied less often by high spinal paralysis. They believe that this effect is probably due to introduction of virus into the innervations of the peritonsillar tissues, notably muscle.

In a second series of experiments on monkeys, they found that intrathalamic inoculation of poliomyelitis virus followed three days later by tonsillectomy was regularly followed within two more days by bulbospinal paralysis, in which all levels of the cord were involved. They believe this effect probably indicates that trauma or physical stress of surgery increases the vulnerablility of nerve cells to infection already present in the central nervous system.

In a third series of experiments on monkeys they found that oropharyngeal swabbing with poliomyelitis virus and no tonsillectomy, spinal paralysis usually resulted, with bulbar manifestations in only one instance. In even a fourth series of experiments on six monkeys, they found that oropharyngeal application of poliomyelitis virus followed by oropharyngeal application of 2 per cent tincture of iodine and then by tonsillectomy, all six animals remained well.

Faber has tested a large number of virucidal agents and has found that 2 per cent tincture of iodine is still the best. On the basis of these experiments, McNaught has routinely swabbed the fauces and nasopharynx before adenotonsillectomy with 2 per cent tincture of iodine and has had no untoward reactions.

Adams[14] and his associates were able to confirm Faber's findings. They found that 20 per cent of monkeys fed poliomyelitis virus and tonsillectomized, subsequently developed bulbar poliomyelitis, whereas in the nonoperated control group of twenty monkeys, fed the same virus in an identical dosage, no poliomyelitis developed. It is encouraging to know that Adams[15] and his associates have found experimentally that the poliomyelitis attack rate in tonsillectomized monkeys fed poliomyelitis virus was 65 per cent, whereas in the animals which were fed poliomyelitis virus, then given human gamma globulin, and then tonsillectomized, the attack rate was only 12 per cent, and in nonoperated control animals fed poliomyelitis virus and given gamma globulin the rate was 13 per cent. Nine of ten operated monkeys given gamma globulin later resisted rechallenge with the poliomyelitis virus, suggesting that the monkeys acquired immunity.

In a personal communication,[16] Adams states that he and his associates were able to prevent poliomyelitis in monkeys fed poliomyelitis virus following tonsillectomy by giving human gamma globulin twenty-four hours prior to the feeding of the virus. In mice he and his associates showed that the time interval was very important, getting their greatest protection when they gave the gamma globulin four days prior to the inoculation of poliomyelitis virus.

What conclusions can then be drawn from these statistical and experimental studies in the poliomyelitis-tonsillectomy problem?

1. The weight of evidence favors the conclusion that if an individual has had a recent adenotonsillectomy and is exposed to poliomyelitis, his chances of developing bulbar poliomyelitis are greater than if he had not had surgery.

2. Adenotonsillectomy should not be performed if there is an epidemic of poliomyelitis in the community.

3. If the indication for adenotonsillectomy is definite and pressing, I believe the operation need not be deferred during the summer months if there is no poliomyelitis present in the community.

4. On the basis of experimental evidence, I believe it is advisable to give poliomyelitis human gamma globulin at least four days before adenotonsillectomy is performed during the months when poliomyelitis is most likely to occur. The gamma globulin should be given in the dosage of .2 cc. per pound of body weight.

5. Also on the basis of experimental evidence, I believe it is advisable to swab the oropharynx and nasopharynx with 2 per cent tincture of iodine just prior to adenotonsillectomy.

6. These precautions in the performance of adenotonsillectomy should prevail until unquestionable methods of preventing poliomyelitis exist.

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Hemolytic Anemia

Diagnosis and Treatment

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HEMOLYTIC anemia is rather common.[3] Increased awareness on the part of clinicians and the use of certain laboratory tests have made the diagnosis of excessive red cell destruction a frequent occurrence.

The size of the circulating red cell mass is fundamentally the net result of production and loss of red cells. Any anemia, therefore, has to result from decreased production, increased loss or combinations of these two factors. Loss of red cells occurs from two possible mechanisms, hemorrhage or destruction of red cells.

We are concerned here with the recognition and treatment of anemias due to an increased rate of destruction of red cells. The normal average life of erythrocytes is 100 to 120 days.[4] In hemolytic anemias the average life of the red cells is always reduced, sometimes to only a few days.

Cross-transfusion studies of serologically identifiable red cells have shown that the hemolytic anemias fall into two major categories.

Table I lists hemolytic anemias due to intrinsically defective cells. The familial hemolytic anemias are in this group.

TABLE I. HEMOLYTIC ANEMIAS DUE TO INTRINSICALLY DEFECTIVE RED CELLS

- Hereditary Spheroidocytosis (familial hemolytic anemia, congenital hemolytic jaundice)
- Abnormal Hemoglobins (Sickle (S), C, D, E)
- Thalassemia ("Mediterranean")
- Hereditary Non-Spheroidocytic
- Paroxysmal Nocturnal Hemoglobinuria

The other major division includes the hemolytic anemias due to extrinsic or environmental factors. These constitute a large group caused by many different agents. Table II lists some of these agents. Anemias due to immune body reactions are the most frequently encountered.

"Idiopathic" acquired hemolytic anemia is in this group. Evidence of immune body reactions is often found in such patients.

TABLE II. HEMOLYTIC ANEMIAS DUE TO ABNORMAL ENVIRONMENT

- Septicemia
- Intracellular Parasites
- Burns
- Animal and Vegetable Poisons
- Chemicals
- Immune Body Reactions
    - Isoagglutinins related to the various blood groups including Rh sensitivities
    - Cold hemagglutinins
    - "Cold" and "Warm" hemolysins
- "Idiopathic" Acquired
- Symptomatic or Secondary
    - Lymphocytic Leukemia
    - Myelocytic Leukemia
    - Hodgkins Disease and other Lymphomas
    - Carcinomatosis
    - Hepatic Cirrhosis
    - Primary Atypical Pneumonia
    - Infectious Mononucleosis
    - "Felty's" Syndrome
    - Polyarteritis Nodosa
    - Salmonellosis
    - Uremia
    - Hyperthyroidism
    - Lupus Erythematosis
    - Sarcoidosis

An especially important type of acquired hemolytic anemia is symptomatic or secondary hemolytic anemia. In these patients the hemolytic anemia is associated with an underlying, primary disease. Table II includes a list of some of the diseases with which increased hemolysis has been found. Obviously, if the clinician is aware that increased red cell destruction can be related to these illnesses, he is more likely to make the proper diagnosis of the anemia.

Diagnosis

There are many leads to the diagnosis of hemolytic anemia. A family history of anemia may indicate a diagnosis of one of the congenital hemolytic syndromes. The possibility of exposure to industrial chemicals must be inquired into. A detailed history as to the ingestion of drugs, chemicals, or other poisons may be revealing. The large number of diseases with which

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hemolytic anemia has been associated must be kept in mind.

Certain observations during the course of a patient's illness may suggest the diagnosis of a hemolytic process. For example, if, without evidence of hemorrhage, the hemoglobin concentration falls faster than about 1 gm. per 100 ml. of blood per week, excessive hemolysis is likely. If a patient, without evidence of hemorrhage, requires transfusions of 500 ml. or more of blood per week to maintain a hemoglobin level, increased red cell destruction is probable. Again, when hemorrhage is not present, if 500 ml. of blood does not increase the hemoglobin by an average of about 1 gm. per 100 ml. of blood, abnormal hemolysis should be suspected.

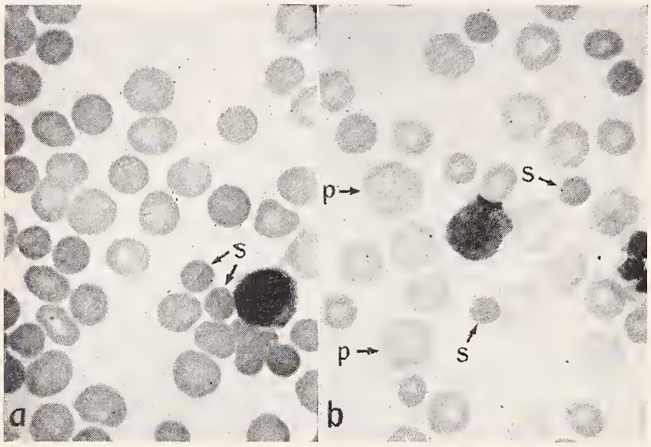

Fig. 1. s=spheroidocyte. p=polychromatophilic cell (these cells have a grayish-blue tinge and usually are larger than normal adult cells).

The presence of splenomegaly and/or jaundice in an anemic individual suggests that increased hemolysis may be present. It is important to realize, however, that hemolysis can be present with or without either of these signs. The presence or absence of jaundice depends simply upon the ability of the liver to excrete the bilirubin as rapidly as it is formed.

Several laboratory studies are helpful in the recognition of increased red cell destruction.

Examination of a stained film of peripheral blood may reveal spheroidocytes. Spheroidocytosis is highly suggestive of increased hemolysis.

Figure 1*a* shows spheroidocytes from a patient with hereditary spheroidocytosis, in which the cells are intrinsically defective.

Figure 1*b* shows spheroidocytosis in the blood of a patient with "idopathic" acquired hemolytic anemia. It is obvious that the pathogenesis of spheroidocytosis cannot be deduced from the morphology alone. This figure also illustrates polychromatophilia, a characteristic of young cells. Increased numbers of polychromatophilic cells indicate increased red cell regeneration. This finding, coupled with spheroidocytosis, is highly suggestive of an active hemolytic process.

Poikilocytosis is another sign associated with a shortened life of the red cell as, for example, in thalassemia. Sickle cells can occasionally be found in the ordinary stained blood film. Increased numbers of target cells may indicate the presence of certain short-lived cells.

Another helpful examination is the reticulocyte count. Normally 0.5 to 1.5 per cent of the circulating red cells stain supravitally as reticulocytes. Increased numbers of reticulocytes represent increased red cell regeneration. If blood loss and response to therapeutic agents can be ruled out, persistent reticulocytosis without a rise in hemoglobin generally suggests increased red cell destruction.

The osmotic fragility test is another aid in the diagnosis of hemolytic anemia. Because spheroidocytes rupture more easily than normal cells in hypotonic salt solutions, increased osmotic fragility demonstrates the presence of spheroidocytes and confirms the impression gained from examination of the peripheral blood smear. A simple 4-tube test can be performed in the office. Figure 2 shows increased osmotic fragility in such a test. The test blood was taken from a patient with hereditary spheroidocytosis two weeks after splenectomy.

The serum bilirubin is often increased in hemolytic anemia and exhibits a predominantly indirect or delayed direct Van den Bergh reaction. However, there may be even marked hemolysis without elevated serum bilirubin.

Since immune antibodies are present in many of the acquired hemolytic anemias, tests for antibodies should be made whenever hemolytic anemia is suspected. The demonstration of antibodies suggests that abnormal hemolysis is present. However, antibodies may be present without increased hemolysis, and increased hemolysis may occur without the demonstration of antibodies. Tests for antibodies include: the Coombs test (direct and indirect), trypsinated red cell test, cold agglutinin titer, Donath-Landsteiner reaction, special examinations for other "cold" hemolysins, and tests for "warm" hemolysins.

Other pertinent examinations include, at times, a sickle cell test performed with a chemical reducing substance, test for hemosiderinuria, and occasionally a bone marrow examination.

The discussion thus far has related to signs which suggest the presence of increased red cell destruction as a cause of anemia. Attention will now be given to diagnostic signs of increased red cell destruction.

The most unequivocal of these is the presence of hemoglobinemia and hemoglobinuria. Hemoglobinemia results from intravascular hemolysis. Hemoglobinuria occurs whenever the plasma hemoglobin level exceeds the renal threshold for hemoglobin. Hemoglobinemia is rarely seen in hemolytic anemia, because in most patients hemolysis is at a slower rate and apparently occurs "extravascularly." On the other hand, hemoglobinemia frequently occurs with severe transfusion reactions and certain septicemias. Patients with paroxysmal cold and paroxysmal nocturnal hemoglobinuria regularly show hemoglobinemia, but usually the diagnosis of increased hemolysis must be made on other evidence.

Another approach is to observe in a patient the fate of identifiable red cells. If the average life of the cells is shortened, an increased rate of destruction is present. These methods may utilize the naturally occurring agglutinogens of the red cells (A, B, M) or certain isotopic tags (Cr^{51}, N^{15}, C^{14}). At present, tagged cell techniques are research methods and are not feasible in general practice.

The most useful and practical means to diagnose an increased rate of red cell destruction is the quantitative determination of the average daily output of fecal urobilinogen in four-day stool collections.[10] Heavy nitrogen tracer studies have indicated there is more than one source of fecal urobilinogen.[8] However, from a clinical point of view, except in the case of pernicious anemia, an increased fecal urobilinogen indicates an increased rate of red cell breakdown. To be of most help, the fecal urobilinogen should be related to the circulating hemoglobin mass either as a "hemolytic index"[9] or an "apparent hemoglobin wastage"[11] figure.

The examination of random stool samples for Ehrlich reacting substances[12] leads to the same general conclusions as the more elaborate quantitative method.

It is important to realize when interpreting fecal urobilinogen values, that falsely normal results can occur. Antibiotics such as aureomycin, terramycin, erythromycin and neomycin given even in small doses can alter the intestinal flora so that the bilirubin is not converted to urobilinogen in the usual way. Constipation and diarrhea may lead to erroneous results. The sample should weigh at least 300 gm. to be considered an adequate four-day stool specimen.

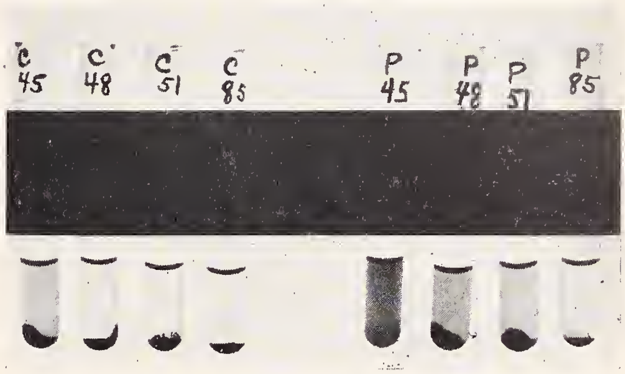

Fig. 2. C=control, normal blood. P=patient's blood. Numerals denote NaCl solution, e.g., 45=0.45 per cent NaCl.

The test is a slight modification of the screening test outlined by Ham.[6] One tenth ml. of blood (collected by syringe and mixed with double oxalate[7]) was added to 1 ml. of each saline solution. The tubes were inverted three times, centrifuged 10 minutes and read. We have used the four solutions as illustrated. However, the 0.51 per cent NaCl is critical. In eighty-five control tests* no hemolysis was visible in the 0.51 per cent NaCl, but 15 per cent showed some hemolysis in the 0.48 per cent and all hemolyzed in the 0.45 per cent NaCl.

*With the technical assistance of Mrs. Patricia Shelander.

There may be enough variability of fecal urobilinogen excretion to require serial examinations for proper evaluation of the rate of red cell breakdown.

Treatment

Since increased erythrocyte destruction may arise from a variety of causes, rational therapy demands that the pathogenesis of excessive hemolysis be diagnosed accurately.

When an infectious or parasitic disease is the cause, appropriate specific therapy should be given. In the case of most toxic agents there is no specific treatment.

In patients with symptomatic hemolytic anemias, treatment directed at the primary diseases may prove fruitful. For example, x-ray treatment in leukemia or lymphoma rarely may decrease the rate of hemolysis.

Otherwise, the available therapeutic measures for hemolytic anemia consist mainly of blood transfusions, ACTH and cortisone, and splenectomy.

Blood transfusions are the mainstay of supportive therapy. However, there is greater danger of transfusion reactions, especially in the patients with hyperimmune states. Each unit of blood introduces about 250 mg. of iron into the patient to add to the hemosiderosis usually present in chronic hemolytic anemia. To limit the number of transfusions it is best not to attempt to keep the hemoglobin level above 10 gm. per 100 ml. of blood.

ACTH and cortisone are used in acquired hemolytic anemia particularly when a hyperimmune mechanism has been demonstrated. The rationale is based upon the hope that ACTH or cortisone will interfere with the antigen-antibody reactions believed to exist in these hemolytic anemias. Results are not constant.[1] However, if there are no contraindications, it appears worth while to try either of these agents in immunohemolytic disease before splenectomy is undertaken, at least as a means of improving the preoperative status.

The results of splenectomy are variable. Splenectomy is effective nearly 100 per cent of the time only in hereditary spheroidocytosis. There has been no notable success in the other anemias due to intrinsically defective red cells, unless there has been a complicating acquired hemolytic component. In acquired hemolytic anemia, whenever the spleen is large and the hemolytic process fulminating, splenectomy may give dramatic results. Often, however, there is no real response. In certain selected patients with leukemia or lymphoma, in whom excessive hemolysis is the immediate problem, splenectomy is often helpful[2,5]

The course of acquired hemolytic anemia is often characterized by remissions and exacerbations, making it difficult, at times, to evaluate the results of therapy.

In conclusion, we will briefly summarize the courses of two patients to illustrate some of the problems of treatment.

Case Reports

Case 1.—The patient was a thirty-four-year-old man who had Hodgkin's disease complicated by hemolytic anemia.[4] He had fever, splenomegaly, leukopenia and thrombocytopenia.

Figure 3 shows the results of survival studies of transfused normal red cells in the patient before and during treatment with cortisone. The Ashby differential agglutination technique was used to determine the average red cell life. Cortisone in an average dose of 150 mg. per day was given for sixty-two days. There was clinical improvement with abatement of the fever. The hemoglobin rose from an average of 10 to 13.6 gm. per 100 ml. of blood, reticulocytosis decreased from 8.5 to 3.8 per cent, and the average fecal urobilinogen decreased from 897 to 559 mg. per day. It can be seen that there was also some improvement in the survival of transfused red cells, but the survival was still far from normal.

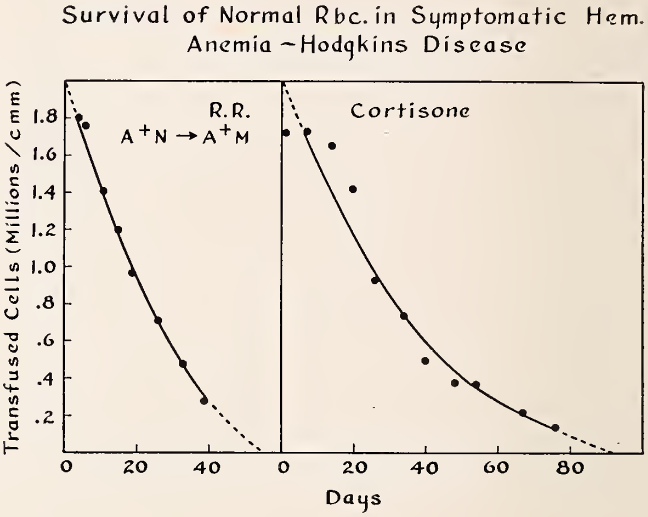

Fig. 3. In each study the test cells were followed by the Ashby differential agglutination technique.[13]

The patient was given ACTH or cortisone for most of the ensuing seventeen months, but he required an average of 432 ml. of blood per week to maintain an average hemoglobin level of about 9.8 gm. per 100 ml. of blood. Splenectomy was then done in the hope of decreasing the transfusion requirement and improving the persistently low leukocyte and platelet counts. The spleen weighed 3475 gm. The transfusion requirement was reduced to an average of 288 ml. per week for the ensuing forty-three weeks. There was also improvement in the white blood cell and platelet counts.

Case 2.—The patient was a thirty-eight-year-old man who had "idiopathic" acquired hemolytic anemia. Survival of the patient's own cells was studied in September, 1953 (Fig. 4), using radiochromium to mark the test cells. The tagged cells disappeared rather rapidly. During this time the average fecal urobilinogen was 1068 mg. per day. At the end of this study there was a marked exacerbation of the hemolytic process. The Coombs test became positive, the hemoglobin fell to 5.3 gm. per 100 ml. of blood, and there was a reticulocytosis of 23.2 per cent. The fecal uroblinogen increased to 2407 mg. per day. He was given ACTH, 80 mg. per day for twenty days. The hemoglobin rose to 8.5 gm., the reticulocytosis decreased to 5 per cent and the fecal urobilinogen fell to 686 mg. per day. Splenectomy was then performed. The spleen weighed 480 gm. ACTH was discontinued. There was improvement in the hemoglobin to as high as 14.2 gm. per 100 ml. of blood. However, the positive Coombs test persisted and there was macroscopic cold agglutination. Restudy of cell survival in February, 1954, showed some improvement, but increased red cell destruction persisted. During this period of observation, average values were as follows: Hemoglobin, 13.2 gm. per 100 ml. of blood; reticulocytes, 3.5 per cent, and fecal urobilinogen, 428 mg. per day.

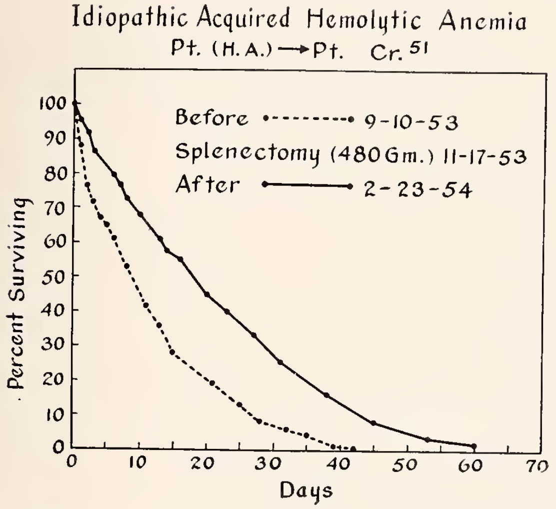

Fig. 4. In each study an aliquot of the patient's blood was incubated at room temperature for 1 hour with radioactive Na_2CrO_4 solution. The red cells were washed with physiologic saline and injected into the patient. A well type scintillation counter was used to measure the radioactivity of serial blood samples. The counts were corrected for radioactivity decay and variations in hematocrit and expressed as per cent of the count of the initial sample.

Summary

1. The diagnosis and management of hemolytic anemia are discussed.

2. Two cases of hemolytic anemia are presented.

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DIGITALIS THERAPY AND DIGITALIS INTOXICATION

Properly used, digitalis is the most valuable agent in the management of congestive heart failure. It is also helpful in the therapy of many cardiac arrhythmias. When used improperly or when actually contraindicated, it may cause serious intoxication, cardiac arrhythmias and even death. Congestive heart failure, regardless of its etiology, is the primary indication for digitalis therapy. The only absolute contraindication is the presence of digitalis intoxication or the presence of full digitalization.

There are many digitalis preparations in use, and each has its own clinical advantages. For rapid oral digitalization digitoxin is preferred. Whole leaf preparations are used for slow digitalization. Lanatoside C, digoxin and gitalin are effective when rapid intravenous digitalization is indicated.

Whole leaf preparations are used chiefly for maintenance therapy where there is no urgency in establishing full digitalization. Digoxin is rapidly excreted, hence it can approach toxicity to obtain full digitalization. Gitalin has been suggested as the preparation of choice by Batterman and co-workers because the therapeutic dose is only one-third of the toxic dose, hence there is a wider margin of safety with this drug. Moreover, in advanced heart disease, gitalin may be useful when other preparations have failed because of toxicity. Its rate of excretion falls between the rapid rate of digoxin and the slow rate of digitoxin.

There is no "average" dose of any preparation that will digitalize or maintain every patient. Each patient must be managed on trial-and-error basis by careful observation.—R. M. DENHAM, *J. Kentucky St. Med. Assn.,* 53:209 (March) 1955.

Cancer of the Thyroid

Fifteen-Year Study in Duluth

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THE literature is replete with many excellent articles dealing with thyroid cancer. The largest series of cases in this country are reported from the major medical clinics, but they do not always get a representative cross section of a given pathologic entity. Then, too, it is always interesting and instructive to review the pathologic material in your own community. Therefore, we have made a study of this subject in Duluth.

There have been, and still are, many problems concerning thyroid cancer. Some of them are: what constitutes malignancy, classification of thyroid neoplasms, lateral aberrant thyroid, treatment by surgery including the place of radical neck dissection, treatment with radiation and radioactive iodine. Some of these problems have been resolved but most of them are still subject to difference of opinion.

TABLE I. INCIDENCE OF THYROID CANCER IN SURGICALLY TREATED GOITERS FROM 1938 TO 1953 IN DULUTH, MINNESOTA

Diagnosis	*Cases*	*Cases in Which Carcinoma Was Present* *Number*	*Per Cent*
1. All surgical goiters	1,485	42	3
2. Diffuse toxic goiter	327	0	0
3. Toxic nodular goiter	353	3	1
4. Nodular goiter	1,073	42	4
5. Nontoxic nodular goiter	720	39	6

Incidence

Cancer of the thyroid is uncommon. According to Hendrick, it makes up 1 per cent of all human cancer. Autopsy figures are not accurate because autopsies are done on a select group of people, the thyroid is not always examined, and if inspected it is often examined only grossly. Furthermore, many patients dying of thyroid cancer do not die in the hospital.[6] Surgical figures of incidence are probably not entirely accurate either as they represent a select group of patients.

From 1938 to 1953 there were 1,485 surgically treated goiters in the three Duluth hospitals. Among the series are forty-two patients who had cancer of the thyroid, an incidence of 3 per cent. There were no cancers in the diffuse toxic goiters. There were three cancers in toxic nodular goiters. The incidence of malignant disease to nodular goiter treated surgically was 4 per cent. These figures on the incidence of thyroid cancer are slightly lower than others published for the Great Lakes area.[2,6]

In the Great Lakes area, where there is such a high incidence of multinodular goiters, it is very difficult clinically to distinguish between single and multiple nodular goiters, especially in the age group over forty. We did not separate our nodular goiters into single and multiple groups.

Materials and Methods

This report is based on fifty-six patients with thyroid cancer studied in the three Duluth hospitals during the years 1938 to 1953 inclusive. They were distributed as shown in Table II.

TABLE II. CANCER OF THE THYROID IN DULUTH YEARS 1938 TO 1953

St. Luke's Hospital	31 patients
St. Mary's Hospital	20 patients
Miller Hospital	5 patients
Surgical material	43 patients
Autopsy material	3 patients
Clinical diagnosis or those patients where tissue not obtainable	10 patients

The histologic material from forty-six of these patients has been reviewed and a simplified histological grouping devised. All of the Duluth pathologists were very helpful in the re-examination of the pathologic material.

Generally speaking, there are two main types of thyroid cancer, the differentiated and the undifferentiated. The differentiated group is of relatively low grade malignancy and occurs predominantly in young people. The undifferentiated

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Presented to the Duluth Surgical Society, March 25, 1954.

cancers are highly malignant and are found chiefly in the over fifty age group.

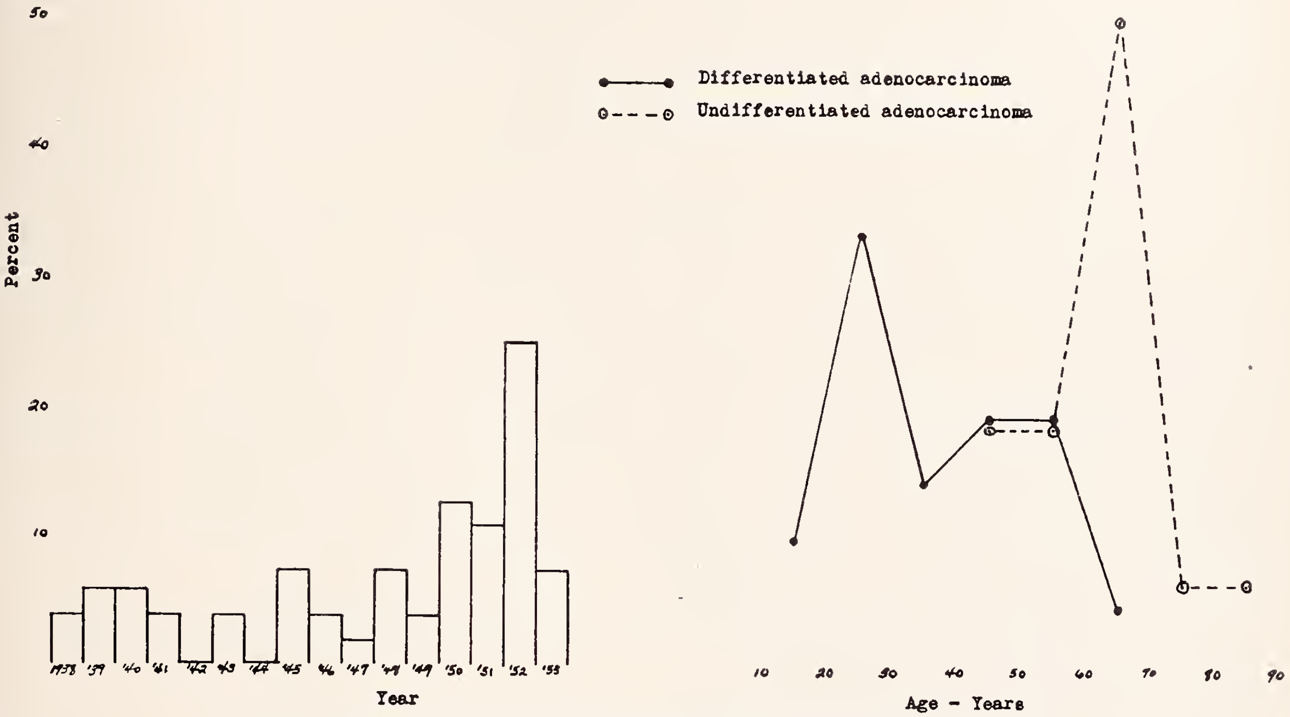

Fig. 1. Occurrence of thyroid cancer in Duluth by year and age groups.

The following table is a modification of Shields Warren's most recent classification. Although the classification is not ideal, it is based on a correlation of histologic structure with clinical course and attempts to indicate which tumors will be sensitive to I^{131}.

TABLE III. FORTY-SIX PATIENTS WITH CANCER OF THE THYROID IN DULUTH

	Patients	Primary Carcinoma Per Cent	Response to I^{181}
A. Differentiated			
1. Papillary adenocarcinoma	10	41	poor
2. Mixed papillary and follicular adenocarcinoma	7		fair
3. Follicular and alveolar adenocarcinoma	4	10	good
B. Undifferentiated			
1. Giant cell carcinoma	14	38	none
2. Small cell carcinoma	2		none
C. Miscellaneous			
1. Hurthle cell carcinoma	4	10	poor
2. Lymphosarcoma	1	2	none
3. Secondary tumors	4		none
4. Squamous cell carcinoma			none
5. Fibrosarcoma			none

The papillary and mixed papillary and follicular adenocarcinoma are grouped together because they are clinically similar in their behavior, the main difference being that the mixed one has more affinity for I^{131}. Our incidence of papillary adenocarcinoma (41 per cent) is somewhat less than the presently quoted figures by other authors.[2,9] Our incidence of undifferentiated adenocarcinomas is also somewhat more than in these recent statistics. Papillary adenocarcinoma usually constitutes 60 per cent and undifferentiated carcinoma 20 per cent of primary thyroid cancer. The information in the column response to I^{131} was obtained chiefly from personal observations and from the literature; not from data in this study.

In Figure 1 it can be seen that there has been a decided increase in the number of thyroid neoplasms recorded during the past four years. The cause of this increase is not readily apparent. Most of this increase is in the differentiated type, that is, the papillary and follicular carcinomas. Crile et al state that in recent years, patients with papillary carcinoma are operated upon oftener and earlier and many patients formerly lived their natural lives with low-grade carcinoma. Another thought is that some of the previously pathologically diagnosed adenomas were adequately excised surgically and were actually carcinomas.

Of the differentiated neoplasms 75 per cent were in patients under age fifty; while eighty per cent of the undifferentiated cancers were over age fifty (Fig. 1).

The sex ratio of thyroid malignancy favored the female in a ratio of 3.5 to 1. However, when considering the differentiated tumors separately, the female preponderance jumped to 9 to 1 and in the undifferentiated group the ratio was practically 1 to 1.

Major Types of Primary Thyroid Cancer

The clinical, pathological and surgical picture of the major types of primary thyroid cancer are so varied that they will be considered separately.

It was possible to make a five-year follow-up study in only about 45 per cent of our cancer patients. Consequently, survival rates will not be stressed in this paper. Furthermore, we believe that the five-year survival rates as a criterion of cure in some forms of thyroid cancer are of limited value.

Papillary Adenocarcinoma

(41 Per Cent) 17 Cases

Papillary adenocarcinomas make up the largest histologic group in most reported series and it also constituted our largest single group. However, in our study the preponderance of papillary tumors was not found. The age range in our series with this tumor was from seventeen to sixty-four years of age. The carcinomas were most frequent during the third and fourth decades, the median age being thirty-two years. It might be noted at this time that this is the characteristic thyroid cancer of youth and childhood. The sex ratio was 7:1 in favor of the female. The predominant symptom or sign was the presence of a lump. The duration of goiter varied from a few weeks to twenty years.

Physical Findings.—The lump was often of small size. A single discrete thyroid nodule was palpable in 75 per cent of cases. Two cases showed palpable cervical lymph nodes on admission and these were both called lateral aberrant thyroid tumors both clinically and pathologically.

Clinical Course.—Papillary tumors are the least malignant of all thyroid cancers and often have an extremely prolonged clinical course. In this series of patients with papillary tumors, only one is dead of this disease, but another is living two years after surgery with pulmonary metastasis. The rest are living and well without evidence of recurrence.

It is to be noted that malignancy was suspected in only three cases (18 per cent) and one of these died. The cause of death in this case was widespread metastasis via the blood stream to the brain, kidney and liver.

Papillary tumors are notorious lymphatic metastasizers, but the only two instances recorded of this phenomenon are in the two cases of so-called lateral aberrant thyroid. They are interesting and will be discussed later.

Results of Treatment.—Adequate surgical removal is the best treatment and the results are good. Radiation therapy is of questionable value. In our series, five patients had Roentgen therapy; one of these died, and the other four received questionable benefit. I^{131} was used also in the patient that died, and since histologically the tumor contained no thyroid follicles it is doubtful if there was any uptake of I^{131}.

Evaluation of radical neck dissection cannot be made at present since this approach to treatment has not been in effect for sufficient time and there have not been enough patients treated this way in our study.

Follicular and Alveolar Adenocarcinoma

(10 Per Cent) 4 Cases

The clinical and pathological characteristics of these tumors are very similar to the papillary group just described except that they are more likely to spread by the blood stream,[12] and they have a relatively good uptake of I^{131}. Spread by lymphatics does not occur until the capsule of the adenoma is transgressed.[3] All patients in this group are living but one has a local recurrence and has a tracheotomy. Nevertheless, this patient must be classed as a ten-year survival.

Undifferentiated Adenocarcinoma

(38 Per Cent) 16 Cases

In contrast to the differentiated carcinomas, the median age for this group was sixty-one years. The youngest patient was forty-two and the oldest eighty-four. The sex ratio was practically 1:1. The striking feature of the clinical picture in these patients was the story of a rapidly enlarging painful mass in the neck, and often dyspnea, hoarseness and dysphagia. These late symptoms of thyroid malignancy were present in over 50 per cent of cases.

Physical Findings.—The mass was often of bulky proportions, in contrast to the differentiated new growths, and fixation to adjacent structures was common.

Clinical Course.—Rapid progression of the disease was the outstanding characteristic. Death was often due to local metastasis causing obstruction of the trachea, esophagus or both. Visceral metastasis did occur, but death usually intervened before this complication became significant. All but one of these sixteen patients are dead. The one living is now nineteen months postoperative following a radical neck dissection and radiation therapy. However, he is in the terminal stage of his disease now with local recurrence and tracheotomy. Another patient survived two and one-fourth years after surgery and died of tracheal and esophageal obstruction. The other fourteen patients (87 per cent) had a mean postoperative survival period of only three months; and the mean duration of life from onset of symptoms to death was about seven months. In no other form of human cancer is such a lethal outcome so inevitable.

Since blood stream and lymphatic metastasis, as well as local invasion of adjoining structure are the rule in this condition, it is not unusual that widespread visceral and bony metastasis were found in autopsy cases.

Results of Treatment.—As indicated in the preceding paragraphs, the results of treatment were most discouraging. Surgical excision with hope of cure was attempted in only five patients or 31 per cent. Radical surgery was done in two patients; one of these died six months postoperative and the other is living with an extensive recurrence nineteen months postoperative.

The response to radiation therapy and I^{131} was uniformly negligible.

Hurthle Cell Carcinomas

(10 Per Cent) 4 Cases

The Hurthle cell carcinoma is a specific histologic entity whose tumor cells resemble the parenchymatous cells of the liver. Microscopically these cancers rarely offered a problem in diagnosis except that there were areas of Hurthle cells in two of our papillary adenocarcinomas. The median age was forty-four, with a range of thirty-eight to fifty-seven years. All of our cases were in women. These patients gave a history of a lump; 50 per cent had dysphagia. There is no agreement in the literature regarding the histogenesis of Hurthle cell tumors.

End Results.—It would seem that these cancers are intermediate in their malignant potential between the differentiated and the undifferentiated tumors. They metastasize first by blood stream and later by lymphatics.[11] One of our patients died six months postoperative; another patient is a ten-year survival but has pulmonary metastasis and has had a metastatic lesion of the brain removed on two occasions. The other two patients are living and well.

The treatment is surgical excision; radiation therapy and I^{131} are of littlle vaue.[11,15]

Lymphosarcoma

(2 Per Cent) 1 Case

The histopathologic diagnosis is sometimes difficult. It must be differentiated from small cell carcinoma and Hashimoto's thyroiditis. Our only case of lymphosarcoma reacted in a virulent manner similar to the undifferentiated tumors. Although this tumor is considered radiosensitive, good results are rarely obtained by roentgen therapy.[10]

Relation of Cancer to Hyperthyroidism

Cancer of the thyroid is rare in hyperthyroidism but one should be cognizant of its possibility. Beahrs et al[1] found an incidence of cancer in cases of exophthalmic goiter of 0.5 per cent, but we had no patients in our series. We did have three cases of cancer in toxic nodular goiters.

Relation of Cancer to Previous Goiter

Approximately 20 per cent of our thyroid cancer patients had a goiter for five or more years. The cancer was considered inoperable on clinical examination or at the time of exploratory operation in twenty-six cases or 47 per cent of the total group. This leaves twenty-nine cases (53 per cent) which were treated surgically with hope of cure. Would removal of these pre-existing goiters have prevented the development of carcinoma or permitted removal of cancers before they were inoperable? Since there is no good way of diagnosing curable thyroid cancer short of histologic examination of tissue and since the mortality and morbidity in experienced hands is very low, we feel that at the present time we are justified in operating upon most nodular goiters.

Preoperative Diagnosis

Cancer of the thyroid gland should be seriously considered under the following circumstances: a single discrete thyroid nodule, cervical lymphadenopathy with goiter, a firm Delphian node, a rapidly appearing or recently enlarging goiter, a very firm thyroid nodule or a relatively fixed thyroid gland, and hoarseness associated with goiter.

Of our surgical cases, malignancy was suspected in 41 per cent. But what was the end result of these cases where malignancy was suspected? Eighty-eight per cent were dead or had a recurrence of their cancer. Thus, if you wait to operate upon those goiters where you suspect cancer, you will have discouraging results. The greatest salvage will be obtained by surgically excising nodular goiter.

Furthermore in our series, 45 per cent of the cancers were in multiple nodular goiters and 55 per cent were found in the single discrete nodule. Therefore, you cannot dismiss the possibility of cancer in multiple nodular goiters, especially in the Great Lakes area. However, papillary cancer was found in a discrete nodule in 75 per cent of cases.

The preoperative differential diagnosis of thyroid malignancy includes hemorrhage into an adenoma, calcification in an adenoma, other cervical tumors and the various types of chronic thyroiditis.

Surgical Treatment

Treatment of the Primary Lesion.—We do not believe that biopsy of the thyroid should be done, except in the inoperable cases or where it is impossible to tell if a lesion is thyroiditis or carcinoma. Nodules of the thyroid should also be removed unruptured. In both instances, implantation of malignant cells may occur if the nodule is broken into.

All thyroid malignancies should not be treated alike. The management of a given patient with cancer is dependent upon the histologic type of malignancy and the extent of local or distant metastasis. If carcinoma of the thyroid is strongly suspected, primary total lobectomy and removal of the isthmus should be done.

If subtotal lobectomy or excision of a discrete nodule has been done, the surgeon should examine the cut section of the tissue grossly. If fresh frozen section examination by the pathologist discloses cancer, the procedure should be completed as a total lobectomy and removal of the isthmus.

When carcinoma of both lobes is found, total thyroidectomy is indicated.

When there is true fixation of the cancer to adjoining structures as so often occurs in the undifferentiated type of malignancy, the salvage from total thyroidectomy alone or associated with radical neck dissection is so low that these procedures are not justified. Other deterring factors to these surgical procedures are the increased operative mortality and the subsequent morbidity of the patient.

Treatment of Lymphatic and Vascular Spread.—Cope has emphasized the importance of looking for the Delphian node both preoperatively and at surgery. A tumor in the isthmus may give bilateral cervical metastasis.

Radical neck dissection is rarely indicated except for some cases of papillary adenocarcinoma.

The *undifferentiated carcinomas* are rarely operable and it is only the occasional case where a radical neck dissection would be indicated.

Follicular and alveolar neoplasms metastasize by blood vessels first and by lymphatics only after the capsule is permeated.[3] Occasionally these tumors show vein tumor thrombosis and if the lesion is otherwise resectable, the involved veins are resected along with the tumor. Limited excision of involved nodes is sometimes indicated; radical neck dissection is not advised.

The greatest controversy concerns radical neck dissection for papillary adenocarcinoma. If no lymph nodes are clinically palpable, we believe that prophylactic neck dissection is not necessary. If one or a few lymph nodes are involved, then these nodes should be excised in groups surrounded with their envelopes of fatty areolar tissue. This more limited excision may be accomplished successfully at secondary and tertiary operations for cervical lymph node metastasis if necessary. If there is extensive cervical lymph node involvement, then a radical neck dissection is indicated.

Cattell and Hendrick favor radical neck dissection for all papillary adenocarcinomas while Crile, Black, and Beahrs are against it.

Crile states that the standard radical neck dissection is inadequate for papillary carcinoma because it does not get the lymph nodes that are most commonly involved. He also states that "if the gross tumor is removed completely, metastasis or recurrence are not observed." He further says that "yet today, many patients are subjected to the most mutilating surgical dissections and what remains of their necks is treated with tremendous doses of roentgen rays. I believe that this needless mutilation in this group of papillary tumors causes more disability than it has prevented." Why is local excision of lymph nodes adequate in most papillary carcinomas of the thyroid?

1. The tumors are slow growing, they remain confined to the neck for many years. Spread beyond the capsule of an involved node is delayed and unusual.

2. Metastasis is embolic and not by permeation; therefore *en bloc* dissection is not necessary.

3. Black found only a 38 per cent lymph node involvement in 112 consecutive cases of papillary adenocarcinoma. Two thirds of these cases with nodal involvement had only one to four nodes with cancer. None of the inoperable cases in Black's study were inoperable solely because metastatic nodes could not be removed.

4. The metastasis that kill are usually blood borne and not lymphatic.

5. Metastatic nodes can be handled by local excision at the first, second or third recurrence, and the patient be cured of his disease. Beahrs reported a five-year survival of these tumors of 89 per cent and a ten-year survival of 82 per cent. It is doubtful if the benefit gained from radical neck dissections will be worth while.

McCorkle et al are extending the operation for thyroid cancer by splitting the sternum and doing a complete superior mediastinal dissection.

The final answer on radical neck dissection and other radical operations must await at least a ten-year follow-up of a sufficient number of cases.

Radiation and I^{131} therapy will not be discussed critically. However, suffice it to say that roentgen therapy is of some effect locally in the more anaplastic forms. Some authorities believe roentgen therapy is worth while; others do not.

I^{131} is probably of use in only 5 to 10 per cent of thyroid neoplasms, that is, in those composed chiefly of colloid filled follicles. Total thyroidectomy and thiouracil have been used in an effort to stimulate uptake of I^{131} in metastatic lesions.

Selected Case Reports

Case 1.—A twenty-seven-year-old white woman first consulted her doctor in November, 1945, because of a slowly enlarging nodule in the right side of her neck of many years' duration. She also complained of an occasional tight feeling in the right cervical region. Surgical exploration at this time was made through a vertical incision along the anterior border of the right sternocleidomastoid muscle. A tumor mass about 3 to 4 cm. in diameter was found at the level of the larynx. It was firm and freely movable and well encapsulated. It was excised together with three adjacent nodes.

The pathologic diagnosis was lateral aberrant thyroid. Her convalescence was uneventful. In July, 1952, or seven years later, she came to our office stating that her physician had told her one year previously that she had a recurrence of her goiter on the right side. She volunteered that it had enlarged slightly the past year and complained of a moderate feeling of pressure on the right but no difficulty in swallowing. There were no symptoms or signs of hyperthyroidism. Examination of her neck revealed a very firm nodule on the right about 3 to 4 cm. in diameter. The basal metabolic rate was —4.

Exploration of the thyroid was accomplished through the usual transverse collar incision. The right lobe appeared to be about twice normal size and was very firm and fibrous. A specimen was sent to the laboratory for frozen section and was reported grade I adenocarcinoma. It was felt that total right lobectomy should be done. As the gland was elevated it was found to extend retrotracheal to the point where the extreme left border of the right lobe lay behind the left lobe which was normal. Total right lobectomy was done with some difficulty.

The final pathologic diagnosis was adenocarcinoma of the thyroid, grade II.

Her convalescence was uneventful. Radiation therapy was started while the patient was still hospitalized. She has remained well to date. Because of the diagnosis of adenocarcinoma, interest was aroused in the sections from the previous cervical operation where the diagnosis was aberrant thyroid. Re-examination of these slides by the pathologist were reported as follows: "In view of the increased knowledge of thyroid tumors the original specimens would now be called adenocarcinoma."

Case 2.—A seventeen-year-old white boy consulted his physician in Duluth in 1950 because of a lump in the right side of the neck of four weeks' duration. Examination revealed a large spherical mass in the right supraclavicular area that was freely movable.

In November, 1950, a lateral aberrant thryoid measuring about 8 by 10 cm. in size was removed in a Duluth hospital.

Three months later, at the Mayo Clinic, a right total lobectomy with removal of the isthmus and pyramidal lobe was done. The primary thyroid cancer was only 1 cm. in diameter and located in the pyramidal lobe.

Seven months later, in September, 1951, a group of three cancerous lymph nodes were removed from the posterior triangle of the right neck.

Five months later a portion of the hyoid bone and a group of four submental nodes were removed. Carcinoma was present in the removed tissue.

There has been no evidence of recurrence at the last two examinations one and one-half years after the last operation.

All pathologic tissue removed showed mixed papillary and follicular adenocarcinoma, grade I and at the last operation an autoradiograph of the carcinomatous tissue removed showed fairly good uptake of I^{131}.

There is now general agreement that the so-called lateral aberrant thyroid is metastatic carcinonma of the thyroid, and the primary site will be found in 100 per cent of cases in the parent organ if careful search is made.

These two patients are well without evidence of recurrence three and nine years after the first operation. The follow-up is probably not long enough but these case histories suggest that adequate excision of the primary lesion plus local excision of metastatic cervical lymph nodes is sufficient for cure.

Case 3.—This patient, a fifty-seven-year-old white woman, is of interest because there were two distinct malignancies at the same time in her thyroid gland. In the right lobe was a grade IV large cell carcinoma and in the left lobe was a grade II alveolar and follicular carcinoma.

Case 4.—A thirty-eight-year-old white woman was first seen by one of us in August, 1939, because of a goiter the past four years and rapid growth during the past two to three months. She also complained of nervousness, excitability and irritability.

She cried easily, perspired freely and was intolerant to heat. Slight exertion caused tachycardia and dyspnea. Her strength was poor and she had lost 20 pounds in weight the past four months.

Physical examination revealed an anxious, nervous woman with a fine tremor of both hands. The blood pressure was 140/70 and the pulse 90. The thyroid gland was markedly enlarged on the right, measuring 7 by 4 inches, and extended into the isthmus. The basal metabolic rate was +24.

At operation a large single adenoma firmly adherent to surrounding structures and involving practically all of the right lobe and isthmus was removed. The left lobe was carefully explored and found to be entirely normal.

The thyroid mass removed weighed 180 grams and histologic examination was reported as carcinoma of the thyroid.

Her convalescence was uneventful. During September and October she was given roentgen therapy. Following this she lived a normal life for ten years until a mobile unit chest roentgenogram in April, 1950, showed a nodular infiltration in the right lower lung. Follow-up roentgenograms over a period of time revealed the lesions to be growing slowly. Metastatic malignancy from the thyroid was considered the probable cause. It was felt that the lesions had progressed too far for pulmonary resection.

In August, 1950, she was referred to the University Hospital for treatment with I^{131} in the hope that the nodules were sufficiently differentiated to show adequate uptake of radio-iodine. Metastatic nodules were now present in both lungs. A tracer done of I^{131} showed 17 per cent uptake in the left lobe of the thyroid. In August, 1950, 50 millicuries of I^{131} were given to destroy the remaining normal thyroid tissue if possible. This was followed by 75 millicuries in December.

In May, 1951, a tracer dose of I^{131} showed no uptake in the lungs. In the meantime, serial chest roentgenograms revealed a slowly progressing increase in the size and number of the metastatic nodules in both lungs.

In March, 1952, she came to the office stating that two weeks ago she became forgetful, could not think clearly and was unable to comprehend printed words. She was hospitalized in April and her condition deteriorated rapidly. She was completely aphasic, very lethargic and quite oblivious to her surroundings. There was a hemianesthesia and hemiplegia present. A ventriculogram of the brain showed a large cystic tumor, rounded in shape, in the posterior parietal and anterior occipital region. Because the tumor had proved to be extremely slow growing, Dr. Strewler did a craniotomy in April, 1952. The cystic area contained thick yellow fluid and there was a firm nodule 2.5 cm. in diameter which was excised with the cyst wall.

The pathologic report of removed tissue was metastatic Hurthle cell adenocarcinoma of the thyroid.

Her convalescence was uneventful and she again lived a normal life until December, 1953. She now complained of headaches for three to four weeks that were becoming progressively more severe. There was also occasional nausea and vomiting, vertigo, confusion, and difficulty naming objects. She was hospitalized and a cerebral ventriculogram again revealed a large mass 5 to 6 cm. in diameter, lying in the temporo-occipital lobe and extending upwards into the parietal lobe.

It appeared to be well demarcated and was removed as completely as possible. Histopathologic examination again showed metastatic Hurthle cell carcinoma. After the tracer dose of I^{131} showed no uptake, she was started on thyroid extract, grains 1, three times a day. She also had intramuscular Testosterone Propionate, 100 mg. three times a week. Because of its virilizing effects, this was changed to Neodrol, 100 mg. twice weekly. Since her last surgery, she has remained in reasonably good health.

Summary and Conclusions

Fifty-six malignant lesions of the thyroid gland seen in the three Duluth hospitals from 1938 to 1953 have been reviewed. Carcinoma was found in 4 per cent of a series of 1,073 cases in which nodular goiter was treated surgically. Our incidence of papillary adenocarcinoma was somewhat less, and our incidence of undifferentiated adenocarcinoma somewhat more than the presently quoted figures of other authors. There has been a decided increase in the number of recorded thyroid neoplasms the past four years and most of this increase is in the papillary and follicular adenocarcinoma group.

Malignancy was suspected in less than 20 per cent of the papillary lesions, in none of the follicular and alveolar group, but in 75 per cent of the undifferentiated neoplasms.

The treatment of papillary adenocarcinomas is usually total lobectomy and removal of the isthmus. If one or a few lymph nodes are involved, limited excision of the group or groups of nodes is indicated. It is believed that radical neck dissection and superior mediastinal node dissection have a definite but limited place in the treatment of this malignancy.

Follicular and alveolar adenocarcinomas are managed usually by total lobectomy and removal of the isthmus. The undifferentiated carcinomas are rarely operable and it is only the occasional case where a radical neck dissection is indicated.

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CURRENT CONSIDERATIONS OF TONSILLECTOMY WITH SPECIAL REFERENCE TO POSTOPERATIVE BLEEDING

Tonsillectomy is followed by complications more frequently than is generally realized, and it is the cause of more postoperative deaths than any other nose or throat operation. Poor technique and the custom of considering adenotonsillectomy as minor surgery account for large numbers of these complications and fatalities. Hemorrhage is the most frequent complication.

The authors doubt that remote tonsillectomy has anything to do with the development of poliomyelitis. Statistics are cited from the authors' practice to support their theory that antibiotics have no part in raising the incidence of post-tonsillectomy bleeding. Children under twenty months have only the adenoids removed and adenoidectomy precedes tonsillectomy in all patients. The usual solid food diet is promptly resumed.

Sulfathiazole gum is chewed for thirty minutes every four hours during the day. The authors routinely treat secondary bleeding with an intramuscular injection of double-strength U.S.P. posterior pituitary extract. They suggest the use of Aspergum if the throat is painful. While the salicylates lower the prothrombin time of the blood, this effect is not likely to occur if aspirin is taken in quantities after tonsillectomy.

In twenty-five consecutive cases given aspirin or Aspergum postoperatively, the prothrombin time was never below 80 per cent of normal. In most cases it was between 90 and 100 per cent of normal.

The authors feel that most cases of post-tonsillectomy bleeding are the result of infection and they never operate if there has been a recent infection, sore throat or cold, or if there is a temperature elevation.—McLaurin, J. W., and Raggio, T. P.: *J. Louisiana St. Med. Soc.*, 107: 91 (Mar.) 1955.

Tumors of the Parotid Salivary Gland

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PAROTID tumors are not common, their incidence being approximately 0.06 per cent of hospital admissions.[11] However, they form a sizable segment of patients with a tumor of the head or neck. The volume of literature concerning parotid tumors in recent years is testimony to the importance of these neoplasms, particularly to the individual so unfortunate as to harbor other than a strictly benign lesion.

When one considers parotid tumors, the usual concern is of mixed tumors since these are in the majority (85-90 per cent). Certainly, too, this is the most important group because adequate treatment may be entirely curative, whereas benign tumors will not recur if properly removed and the treatment of a frank carcinoma leaves much to be desired in long-term results.

The simple classification given in Table I is presented as a clinical guide in dealing with these lesions. Several features of this classification are worthy of comment.

Mixed tumors are classified as a separate entity, neither benign nor malignant. The exact nature of these lesions is as yet incompletely understood. It is probable that they are not entirely benign, neither are they malignant by usual criteria.[5] Instances of metastasis of a mixed tumor as such are undoubtedly infrequent if they do occur. However, they tend to recur locally if incompletely removed, and the recurrence may be definitely malignant and metastasize many years later. The classification of mixed tumor into two broad groups rather than more complicated histologic divisions has practical application in that the interstitial tumors are in the majority yet recur less frequently than the cellular tumors.[8] It is not possible to determine microscopically which will recur, recurrence being more a function of treatment than the type of tumor. The more cellular tumors would appear to have more potential in this respect, however. It is likely that mixed tumors are, as suggested by Willis,[14] a step in progression between definitely benign and definitely malignant lesions.

TABLE I. CLASSIFICATION

I. Benign
    A. Adenolymphoma
    B. Adenoma*
II. Malignant
    A. Adenocarcinoma
        Papillary
        Cylindroma
    B. Undifferentiated
    C. Squamous
    D. Sarcoma
III. Mixed Tumor
    A. Parenchymatous
    B. Interstitial

*There is question as to the occurrence of adenomas. The occasional reported case is probably a cellular mixed tumor.

The benign lesion occurring with sufficient frequency to be clinically significant is the adenolymphoma. This is the only parotid tumor that has a sex predilection, being five or six times as common in the male. An adenolymphoma may be cystic and smooth but is clinically inseparable from mixed tumor—at least there is no reported case having been diagnosed preoperatively.[7]

The malignant nature of the cylindroma is recognized by classifying it as a type of adenocarcinoma, although it may not be as malignant as the usual parotid carcinoma.

Diagnosis

Parotid tumor should be suspected in the presence of any mass in the parotid area, therefore within a radius of 4.5 cm. from the angle of the jaw. Typically a mixed tumor projects sharply from the cheek (Fig. 1), is firm or hard and nodular, and displaces the ear lobe. Frequently it gives the false impression of being superficial because the superficial lobe of the gland tends to shift with the tumor. We have been impressed by the frequency of tumors occurring in the isthmus and deep lobe of the gland (nine of twenty in our cases) where they may be mistaken for lymph nodes especially when small (Fig. 2).

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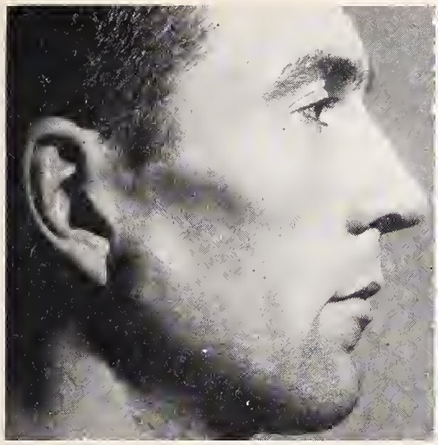
Fig. 1. Mixed tumor in superficial lobe of the parotid.

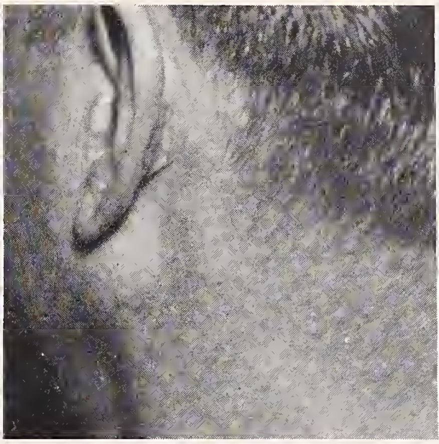
Fig. 2. Mixed tumor in the isthmus.

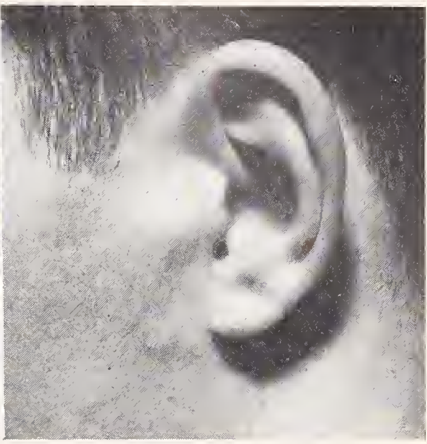
Fig. 3. Recurrent mixed tumor two years after incomplete removal through inadequate, poorly placed incision.

Differential Diagnosis

No attempt will be made to indicate a complete differential diagnosis of these lesions but a few important points may be mentioned:

Primary vs. Secondary.—Lymph nodes and lymphatic tissue exist in the parotid and consequently may receive metastases from a primary tumor elsewhere. This lymphatic tissue may be the site of involvement by Hodgkin's disease or other lymphoblastomas, and a Miculicz type of lesion has been described.

Inflammatory lesions with or without stone. We have encountered patients with unilateral swelling of the gland simulating tumor in whom no duct obstruction or tumor was demonstrated. This likely is due to a low-grade nonspecific parotitis. The presence of calculus may be demonstrated by x-ray, revealing an opaque stone, or by sialogram, and this is probably the most useful contribution of sialography. We have not been impressed by its advantage in demonstrating tumors or aiding in the differential diagnosis of the same.

An encapsulated cyst of the parotid may present as a solitary tumor, may not be associated with duct obstruction at the time and may become secondarily infected to further simulate a mixed tumor with necrosis. We have operated upon three such patients for "mixed tumor" and have done total parotidectomy only to find an encapsulated cyst without evidence of tumor in its wall. Biopsy at the time of operation may have prevented this, but separating inflammatory cyst from necrotic mixed tumor by frozen section presents obvious difficulties.

Lymph nodes, either hyperplastic or pathologic, may simulate tumor. This is particularly true of those behind the ramus of the mandible (v.s.), in which the reverse is more apt to happen, i.e., a parotid tumor is mistaken for lymph node.

Differentiation Between Benign and Malignant

It is usually possible to distinguish clinically between benign or mixed tumor and carcinoma. The chief differential points are listed in Table II, but of these several appear to be particularly significant.

TABLE II

	Benign (Mixed Tumor)	Malignant
Duration	Long	Short
Age	40—	40+
Size (variable)	Slow growth	Rapid or increasing
Contour	Project	May be flat-infiltrative
Consistency	Firm—hard	Hard
Mobility	Slightly	More Fixed
Pain	Not usual	Frequent
Paralysis	Unusual	Frequent
Metastases	No	Frequent

Presence of Pain.—Mixed tumors and benign tumors may occasionally be attended by mild discomfort but are usually asymptomatic. Pain suggests involvement of the trigeminal nerve by malignant growth.

Facial nerve paralysis occurring without previous trauma is an indication of malignancy. Mixed tumors frequently displace the nerve but do not infiltrate it and do not cause paralysis.

Characteristically mixed tumors tend to project sharply from the face and displace the lobe of the ear; carcinomas are more apt to be flat, infiltrative and less conspicuous, save for the obviously large advanced lesions.

The significance of metastases is obvious.

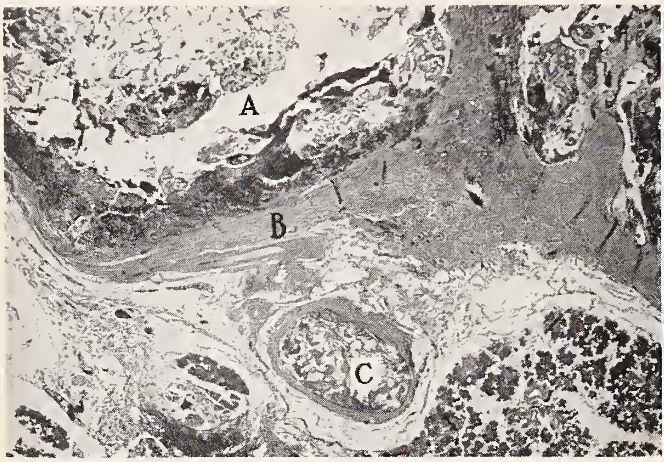

Fig. 4. X15. Typical daughter tumor (C) separated from main tumor (A) by capsule (B). Extracapsular removal of tumor may allow satellite nodule to remain.

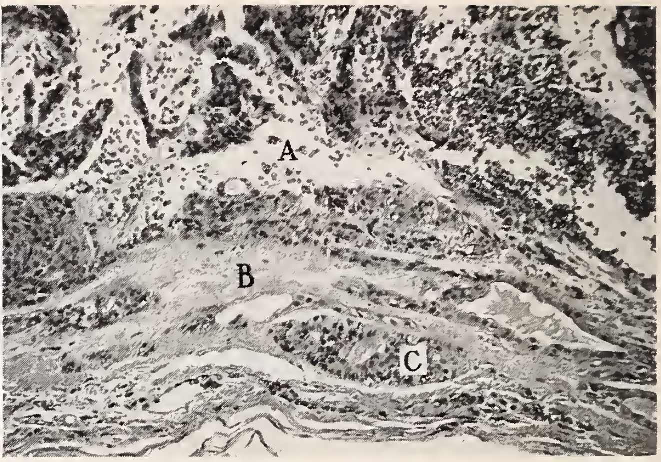

Fig. 5. X100. Penetration of capsule (B) by tumor cells (C).

Treatment

Radiation.—Parotid tumors in general are radio-resistant. Although a few favorable results have been reported in the treatment of mixed tumor, radiation is not recommended. Because of the highly malignant, anaplastic nature of many carcinomas, radiation probably should be used as an adjunct to surgery.

Preliminary biospsy is condemned because this violation of the capsule allows implantation of tumor cells. We have done biopsy at the time of surgery only on clinical suspicion of carcinoma, but it may be advisable to extend its application (v.s.).

Treatment of Mixed Tumor

Recurrence is the menace of mixed tumor. Figures as to recurrence vary, depending on the size of the series, type of treatment and length of follow-up, but a conservative estimate is about 25 per cent.[8]

A number of factors operate to produce this high figure.

1. Incomplete removal because of reluctance on the part of the surgeon to carry out wide excision in an area traversed by the facial nerve. Lack of understanding of the anatomy and unfamiliarity with the problem results in timid excision done too frequently with local anesthesia under the impression that these tumors are always benign (Fig. 3).

2. Error in diagnosis. We have seen a number of patients with recurrent tumors who were first operated upon with local anesthesia for "biopsy of a lymph node" or "removal of a cyst" resulting in incomplete removal of a parotid tumor after a tedious and distressing episode for both patient and physician. Although rupture of the capsule of the tumor may occur in any method of excision, it is probably most likely to occur in the above circumstance. Rupture of the capsule and morcellation of the tumor provides easy opportunity for seeding of tumor cells and invites recurrence.

3. The presence of satellite nodules, a characteristic of these multicentric lesions, is another cause of recurrence when the tumors are removed by the usual extracapsular dissection which may allow daughter tumors to remain. (Fig. 4).

4. Careful histologic examination of mixed tumors will show penetration of the capsule by tumor cells — another invitation to recurrence. Houck demonstrated this in 28 per cent of his series[6] (Fig. 5).

Because of this real threat of recurrence, the increasing risk of facial nerve injury with subsequent operations in a scarred area and the possibility of malignant change or primary low-grade malignancy,[10] the first operation must be thorough, to reduce recurrence, and at the same time should avoid injury to the facial nerve.

A number of approaches to this end have been advocated. Enucleation within the capsule, and enucleation within the capsule with subsequent removal of the latter have been used but are obviously unsatisfactory because rupture of the capsule may result in seeding tumor cells. Extracapsular dissection is more satisfactory but may lead to incomplete removal and recurrence as indicated above. Unfortunately, supplementing any of the above methods by irradiation, either preoperative or postoperative, external or interstitial, or combinations of these cannot be expected to materially alter the recurrence rate.

TABLE III.

	No. Cases	Total Parotidectomy	Superficial Lobectomy	Excision	Nerve Injury	
					Temporary Weakness	Weakness Potrion Mandibular Division
Primary Mixed Tumor	20	17	1	2	7	1
Recurrent Mixed Tumor	2	2				1
Adenolymphoma	1	1				
Cyst	3	3			1	

In contrast to the difficulties encountered in the treatment of mixed tumor of the parotid is the result obtained in these tumors when they occur in the submaxillary gland. Although not as frequent in the submaxillary, the same lesions occur, and here, too, the recurrence rate is approximately the same when simple excision of the tumor is performed. However, after total submaxillary excision recurrence is infrequent,[2,4] suggesting again that this is a function of the treatment rather than the type of tumor. Total submaxillary excision is a relatively simple procedure without many hazards except injury to the lingual nerve, which should not occur with an accurate dissection. Total parotidectomy, however, is attended by considerably more risk of facial nerve injury and has been considered almost impossible without damage to this structure; the usual concept being that the nerve passes through the substance of the gland. Probably the first accurate description of the surgical anatomy of the parotid was given by Bailey,[1] who confirmed the earlier findings of McWhorter.[9] They demonstrated that the facial nerve does not run through the gland in a strict sense but passes between the deep and superficial lobes, enclosed in the parotidomasseteric fascia.

Fortified with this knowledge it was apparent that total parotidectomy with preservation of the facial nerve was possible, and since Bailey's work a number of papers have appeared describing the technique. We have been interested in this approach for a number of years and during this time have been treating mixed tumors by total parotidectomy.[13] The results in terms of injury to the facial nerve are listed in Table III. While the follow-up is too short for statistical evaluation, it should be productive of improved results if one may compare them with similar submaxillary tumors. The operation is done under general anesthesia, is tedious, and requires thorough knowledge of the anatomy. However, the hazard to the patient once the technique is developed is less than the threat of recurrence.

Treatment of Carcinoma

Treatment of carcinoma of the parotid has been unsatisfactory by either surgery, x-ray, or combinations of both. These tumors are frequently undifferentiated and tend to metastasize to regional nodes and to distant areas. Unfortunately, they are usually resistant to x-ray, and although occasionally they may respond,[12] this is too often temporary and not curative. Their location in many instances influences the surgeon to perform a more conservative operation than is consistent with good surgical treatment of malignancy.

One of the important considerations in the management of these patients probably is adequate treatment of mixed tumors in general, their potentiality for undergoing malignant change being recognized.[10] In our own series there are two patients who had removal of what probably was a mixed tumor some twenty-seven and thirty years previously. In both of these there had been prompt recurrence, a long period of quiescence and more recently a rapid growth.

Faced with the problem of the patient with a carcinoma (and this must be confirmed histologically) without distant metastases, experience indicates that a combination of surgical excision and x-ray offers the greatest chance for cure.[3] It is further evidence that the "cured" patients have had radical surgical excision as part of their treatment.[10]

It appears that the loss of function of a portion or all of the facial nerve would be a small price to pay for the possibility of cure by surgical excision. Furthermore, the primary tumor if not completely removed will, in all likelihood, involve the nerve and produce paralysis. We therefore have unhestitatingly removed the facial nerve with the tumor mass, carrying the dissection through the parotidomasseteric fascia, removing the masseter or a segment of the mandible when involved, and if cutaneous extension is present, have covered the defect with a skin graft. A cervical node dissection is done in continuity and postoperative irradiation given. In the postoperative period the cornea on the operated side is protected from ulceration by a shield or a tarsorrhaphy has been done. Long term results of this procedure have been discouraging (Table IV) but several features are apparent:

TABLE IV. CARCINOMA OF PAROTID TREATED BY RADICAL PAROTIDECTOMY, NODE DISSECTION AND X-RAY

Patient	Age	Duration	Previous Operation	Recent Growth	Pain	Paralysis	Type	Nodes	Recurrence	Result
A.L. 110972	53	2 mos.	0	+	+	0	Squamous	+	42 mos.	D 44 mos.
F.K. 111839	58	30 yrs.	30 yrs. 28 yrs.	+	0	+	Squamous	+	4 mos.	D 15 mos.
*A.A. 128344	64	7 mos.	0	+	+	0	Squamous	+	Persistence	D 4 mos.
B.H. 130807	42	10 yrs.	6 mos.	+	+	0	Adeno-carcinoma	+	5 mos.	D 9 mos.
M.R. 139919	46	27 yrs.	27 yrs.	+	+	+	Undiff.	+	4 mos.	D 13 mos.
J.H. 150539	59	20 yrs.	0	+	+	0	Undiff.	+	0	L & W 12 mos.
R.W. 152383	59	1 yr.	0	+	+	+	Adeno-carcinoma	0	0	L&W 7 mos.

*Incomplete removal—extension into carotid artery.

1. Patient F.K., who had a large fungating lesion, received palliation so that his remaining fifteen months were reasonably comfortable and no doubt his existence was prolonged.

2. In patient A.L. the rapid painful growth of his lesion was interrupted and he was without evidence of recurrence for three and one-half years.

3. No five-year cures are evident—two patients recently operated upon as yet show no evidence of recurrence, both of them being probably more favorable patients for cure. It is contemplated to do a fascial sling support to the eye in two years if there is no recurrence.

Conclusions

1. Our experience with some of the more common parotid salivary tumors is summarized.
2. Complete removal of a mixed tumor is best achieved by total parotidectomy.
3. Excision of a mixed tumor with local anesthesia is inadequate, and invites recurrence.
4. Preliminary biopsy allows seeding by tumor cells and is contraindicated.
5. Treatment of carcinoma is discouraging but a combination of radical surgical excision and irradiation appears to offer the best opportunity for cure.

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Intestinal Obstruction due to Inflammatory Strictures

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INTESTINAL obstruction resulting from inflammatory strictures of the bowel has received little attention in the past five years. Although sporadic discussions have appeared in the past, this type of obstruction has become a less prominent problem today. Undoubtedly, the modern management of inflammatory diseases of the bowel, which includes the use of antibiotics and surgical extirpation of diseased segments of the gut, has controlled the primary disease prior to the obstructive stage in many patients. Despite these developments in therapy, obstruction occurring in the course of inflammatory disease of the bowel continues to be seen. Diseases, such as regional enteritis and ulcerative colitis, may progress to obstruction in spite of therapy. In fact, obstruction may actually be the complication that first brings attention to the disease. A consideration of this important though not common type of intestinal obstruction is presented herein.

Clinical Material

This report is based upon a review of forty-five cases of obstructions due to inflammatory strictures seen at the University of Minnesota Hospitals between January 1, 1942, and September 1, 1953. Two additional patients had inflammatory obstruction as an incidental finding terminally in their course, but since the cause of death was other than obstruction they are not included in this study. (One succumbed to acute fulminating ulcerative colitis; the second death was due to rheumatic heart disease, bacterial endocarditis and multiple myocardial abscesses). Only patients with acute abdominal distention with roentgenographic evidence suggesting mechanical bowel obstruction were included. In many cases a diagnosis of primary inflammatory disease of the bowel had been established at some previous admission to the hospital.

TABLE I. AGE DISTRIBUTION OF PATIENTS WITH INFLAMMATORY STRICTURE

Age (Years)	Cases	Per Cent
0-10	0	0.0
11-20	11	24.4
21-40	11	24.4
41-60	12	26.7
61-80	9	20.0
Over 80	2	4.5
Total	45	100.0

Age

There was a correlation between the ages of the patients and the causes of obstruction. In fifteen patients with regional enteritis the average age was twenty-six years. The average age of patients with obstruction from ulcerative colitis was forty-four years. Almost 87 (86.7) per cent of the patients who had obstruction due to diverticulitis were over the age of sixty years. Overall, only 24.5 per cent of the inflammatory obstructions were in patients over sixty years of age (Table I), while in comparison, 40.3 per cent of intestinal obstructions from all causes[8] were in this age group.

TABLE II. THE CAUSES OF INFLAMMATORY STRICTURES. 45 CASES*

Etiology	Cases	Per Cent
Regional enteritis	15	33.3
Diverticulitis	14	31.1
Colitis (ulcerative or regional)	5	11.1
Secondary to Radiation	5	11.1
Boeck's sarcoid	2	4.5
Traumatic	1	2.2
Strictures of unknown etiology	3	6.7
Total	45	100.0

*No deaths occurred among these cases.

Etiology

Regional enteritis and diverticulitis each accounted for almost one-third of the entire group (Table II). Ulcerative colitis, irradiation therapy, Boeck's sarcoid and trauma were less frequent causes.

The most common etiological disease, regional

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enteritis leads to narrowing of the lumen by a process of ulceration and polypoid changes in the mucosa, followed by fibrosis with stiffening of the bowel wall finally resulting in partial or complete obstruction. On radiologic examination this condition frequently produces the characteristic "string phenomenon." In our series slightly over one-half of the patients (53 per cent) had the "string sign" demonstrated by roentgenograms. Although this disease may involve portions of the proximal colon all cases of obstruction seen in this series were of the small bowel. In our experience of 746 cases of small bowel obstruction seen at the University of Minnesota Hospitals from January, 1942, to September, 1953, fifteen cases (1.6 per cent) were due to regional enteritis. Similarly, Bollinger and Fowler[1] found that 2 per cent of small bowel obstructions were due to regional enteritis. Although, its incidence among the entire group of intestinal obstructions is not high, the potentiality of this disease to lead to obstruction is ever present.

The second important cause of inflammatory strictures, diverticulitis, causes obstruction through edema and spasm resulting from an inflammatory reaction within the characteristic mucosal sacculations. Thirteen cases of obstruction due to diverticulitis (one diverticulum occurred in the jejunum) occurred among 506 cases of large bowel obstructions seen during the 1942-1953 period, or an incidence of 2.7 per cent. This correlates fairly well with the findings of Michel et al[5] who reported that 3.8 per cent of acute obstructions of the colon in their series were due to diverticulitis.

A less frequent cause of obstruction was ulcerative colitis, a disease which may involve any part of the colon or the terminal ileum. Shallow ulcerations and denudation of the mucosa in the acute phase progress to diffuse scarring of the bowel wall and narrowing of the lumen, giving the typical "lead pipe" appearance on roentgen examination. Obstruction can occur at any stage of this disease. Colic strictures due to ulcerative colitis accounted for 11.1 per cent of the patients in this series.

A less common cause of obstruction was stricture secondary to abdominal or pelvic irradiation. Recently, Gardner and Arlyon,[3] as well as others[4,9] have called attention to this particular sequelae of irradiation therapy. The acute hyperemia and mucosal edema of irradiation may lead to cicatricial constrictions and subsequent obstruction. The five cases in this study followed roentgen therapy used in the treatment of pelvic malignancies.

Sarcoidosis of the small intestine accounted for two instances of stricture. These cases illustrating isolated sarcoidosis simulating non-specific ileojejunitis have been previously reported.[13]

There was one case of stricture secondary to trauma which had resulted from a self-administered enema. Although not common, blunt trauma to the abdomen with hematoma of the bowel wall has led to stricture formation and obstruction. Also stenosis resulting from limited intestinal infarction by mesenteric vascular occlusion, incarceration within a hernia, and intussusception have been reported.[11]

Tuberculosis has been cited as one of the most common causes of inflammatory strictures of the bowel.[11] However, obstruction of tuberculous origin has not been seen on the surgical service of this hospital for over a decade. This is doubtless due to the fact that patients with chronic tuberculosis are not regularly admitted here. Unfortunately, specific information of the incidence of enteric tuberculosis from sanitoria sources is wanting, but with improved and newer antibiotic therapy it appears reasonable that the incidence would be decreasing.

Diagnosis

Recognition of the presence of obstruction due to inflammatory disease of the intestine is dependent upon clinical awareness of this possible event in the course of diseases such as regional enteritis, diverticulitis and ulcerative colitis.

Of primary importance is the recognition of the presence of obstruction *per se,* and evaluation as to degree, location and possible presence of strangulation rather than the exact etiologic diagnosis. However, identification of the type of underlying mechanism is important, and although the exact diagnosis of the nature of an obstruction is often inferential, careful inquiry with pertinent roentgenograms will lead to a correct diagnosis of the cause of obstruction in most cases.

In this series of cases, most of the patients had been treated medically prior to their obstruction and the diagnosis previously established.

In the presence of partial or complete intestinal obstruction due to inflammatory stricture, careful roentgenographic studies often revealed pertinent information. Aside from the usual flat and upright roentgenograms of the abdomen more detailed information regarding the possible cause of obstruction was obtained in a few instances through the use of special roentgenographic studies. Although most roentgenologists agree that the examination of the terminal ileum may be accomplished by barium enema alone, there are cases in which the competency of the ileocecal valve cannot be overcome. Barium taken by mouth may render a partial obstruction complete. Therefore in such instances, a long intestinal tube (Smith, Miller Abbott, et cetera) passed into the small bowel followed by an injection of a small amount of thin barium just proximal to the site of obstruction[2,6] is the preferred method for visualization of the stricture. This method not only serves as a diagnostic tool, but it also affords an avenue for aspiration of the barium mixture following the examination. In addition, the use of a thin mixture of barium definitely lessens the risk of complete obstruction. During barium enema examination, the column of barium should not pass the site of occlusion in any obstructed patient with colonic distention. In fact, if the distention is severe, laparotomy without delay is indicated.

Treatment

Of the forty-five cases seen, thirty-one patients received operative management, while fourteen patients were successfully treated with suction decompression and intensive antibiotic therapy. Of the thirty-one patients operated upon the stricture was excised primarily in twenty-eight instances. In three cases the bowel was not resected (Table III). Of this latter group, two patients received a decompressing transverse colostomy performed in the face of severe colonic distention. The other patient had a jejunal diverticulum causing obstruction. This was excised with simple closure of the resultant defect.

Although conservative management was successful in a fairly large number of patients (fourteen of forty-five cases), prolonged delay of surgery in the face of unrelenting obstruction is never warranted. Recently, Bollinger and Fowler[1] have stressed the hazard of procrastination associated with suction decompression in the treatment of intestinal obstruction. They have shown an increase in mortality rates with delay in surgery. At the University of Minnesota Hospitals uncomplicated simple small bowel obstructions are currently treated with an approximate twelve-hour trial of intestinal suction,[8] at the end of which time operation is undertaken if there has been no progressive relief of the distention. Distention encountered at surgery is preferably handled by decompressive enterotomy[10,12] or long intestinal tube insertion.[7,12] The problems of postoperative management are considerably lessened by the use of such techniques.

TABLE III. OPERATIVE TREATMENT OF INFLAMMATORY STRICTURES. THIRTY-ONE CASES

Nature of Surgery	Site of Obstruction Small Bowel	 Large Bowel
1. Bowel resection	20	8
2. Not resected	1*	2†
Total	21	10
3. Surgical decompressions used in above surgery		
a. Colostomy	0	5†
b. Ileostomy	1‡	2
c. Witzel enterostomy	1‡	0
d. Long tube insertion at surgery	2‡	0

*Excision of jejunal diverticulum.

†Colostomy concommitant with resection in three patients and decompression by colostomy alone in two patients.

‡Methods of decompression proximal to the site of resection.

Because of the etiology of this type of obstruction, patients with inflammatory strictures should be placed on intensive antibiotic therapy. The fact that almost one-third of these obstructions relented following conservative suction management and antibiotics attests to the value of these measures in certain cases.

In operative cases, a primary resection was done in all cases except where colic distention was of such magnitude as to make this unwise. All surgical candidates should be relieved of intestinal distention prior to bowel resection preoperatively or at the time of surgery. If an operative method of decompression is not employed (enterotomy, colostomy, et cetera), a long intestinal tube should be utilized during the postoperative period for control of distention in the area of the anastomosis.

Mortality Rate

There were no deaths among the forty-five cases reviewed in this series.

The patients in this study fell predominately in the early middle-age group which is in contrast to the older age of patients with obstruction from all causes.[8] The relative young age of patients in this series may be responsible, at least in part, for the fact that there were no deaths; but im-

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Motor Vehicle Safety for Minnesota Children

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MOTOR vehicle accidents on Minnesota's streets and highways during the past five years took the lives of 645 children in the four age brackets from 0 to 19 years. These fatalities to the youth of our state represented 21.8 per cent of the 2,962 deaths resulting from motor vehicle accidents during that period. Deaths in this age group reached a high of 148 in 1952 and accounted for 27.4 per cent of that year's total road toll. Figures and percentages for the five-year period are shown in Table I.

TABLE I.

Year	Total Traffic Deaths	Children Age 0 to 19	Percentage of Total
1950	532	91	17.3
1951	612	120	19.6
1952	539	148	27.4
1953	638	145	22.7
1954	641	141	22.0
Totals	2,962	645	21.8

TABLE II. DEATHS BY AGE GROUPS

Year	0 to 4	5 to 9	10 to 14	15 to 19	Total
1950	17	13	15	46	91
1951	25	28	16	51	120
1952	27	34	27	60	148
1953	33	22	22	68	145
1954	23	22	24	72	141
Totals	125	119	104	297	645

As might be expected, the greatest number of deaths from motor vehicle accidents involving children occur in the 15 to 19 age group because of the greater exposure brought on not only by non-driver activities but because most youngsters in this group begin to drive automobiles. Table II shows the number of deaths by age groups for the past five years. It is interesting to notice that the 10 to 14 age group has the lowest number of deaths for the five-year period.

A total of 176 juvenile pedestrians were killed in Minnesota traffic accidents during the past five years (Table III). It is interesting to see the drastic drop in fatalities in the two higher age groups, indicating a growing awareness of traffic dangers, on the part of youngsters in these groups, and also include youngsters who are old enough to think for themselves and are able to avoid involvement in accidents, even though they may not have parental or adult protection during most instances of their exposure to accidents. *It is tragic that 74 per cent of the deaths involving juveniles fall into the age groups from 0 to 9 years—ages at which close parental supervision should prevent most of these fatalities.*

TABLE III. PEDESTRIAN DEATHS

Year	0 to 4	5 to 9	10 to 14	15 to 19	Total
1950	9	6	6	8	29
1951	11	18	4	—	33
1952	9	20	5	6	40
1953	18	15	4	5	42
1954	13	11	5	3	32
Totals	60	70	24	22	176

TABLE IV. BICYCLE FATALITIES

Year	0 to 4	5 to 9	10 to 14	15 to 19	Total
1950	—	4	5	—	9
1951	—	3	2	—	5
1952	—	3	4	—	7
1953	—	2	3	1	6
1954	—	1	3	1	5
Totals	—	13	17	2	32

All but two of the thirty-two fatalities befalling juvenile bicyclists during the five-year period were in the two age groups of 5 to 9 years and 10 to 14 years (Table IV). This is due, no doubt, to the fact that those in the former group are just learning how to ride bicycles, while those in the latter group are at a more daring and careless age but not probably fully aware of their responsibilities and duties as cyclists. It is entirely understandable that there were no fatalities

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in the 0 to 4 year age group because these youngsters had not yet reached the bicycle-riding stage. (Tricycles and other such vehicles are not classified as bicycles by the National Safety Council, with any deaths involving such vehicles considered as "pedestrian" accidents.) The reduction of bicycle deaths in the 15 to 19 year group is probably due to a decreased utilization of bicycles in favor of automobiles.

TABLE V. MOTORIST FATALITIES

Year	0 to 4	5 to 9	10 to 14	15 to 19	Total
1950	8	3	4	38	53
1951	14	7	10	51	82
1952	18	11	18	54	101
1953	14	5	14	63	96
1954	10	10	16	68	104
Totals	64	36	62	274	436

TABLE VI. SEX AND AGE OF DRIVERS, 1952

	All Accidents		Fatal		Personal Injuries		Property Damage	
	Male	Female	Male	Female	Male	Female	Male	Female
Rural								
14 and under	39	4	3	1	14	3	22	—
15 to 19	1,264	178	46	3	833	67	1,185	98
Totals	1,303	182	49	4	847	70	1,207	98
Total All Age Groups	17,375	1,669	387	40	6,141	631	10,847	998
Urban								
14 and under	72	6	2	—	22	3	48	3
15 to 19	6,316	496	26	2	1,250	102	5,040	392
Totals	6,388	502	28	2	1,272	105	5,088	395
Total All Age Groups	61,164	7,358	197	16	11,416	1,541	49,551	5,801
Totals All Age Groups Rural + Urban	78,539	9,027	584	56	17,557	2,172	60,398	6,799

TABLE VII. BICYCLE VIOLATIONS, 1953

Violations	All Acc.	Fatal Acc.	Bicyclist's Age						
			8 or Under	9 to 10	11 to 12	13 to 14	15 to 19	20 & Over	Not Stated
Inadequate brake	8		2	2		2			2
Improper lights or no lights	13	4	1	1	2	4	1	3	1
Failed to grant right of way to vehicle	55	1	17	18	5	12	2	1	
Improper turning	41	1	12	10	11	6	2		
Wrong side of road	20	1	6	5	4	2	1	1	1
Carrying passengers	10	2	2	3		4	1		
Disregarded traffic control	23	1	3	8	2	6	1	1	2
Failed to ride single file	1			1					
Failed to stop leaving driveway or alley	46		20	13	4	6	1		2
Failed to signal or improper signal	12		5	2	4	1			
Riding on sidewalk crossing alley or driveway	11		5	3	1	2			
Other violations	2			1	1				
Total number of violations	242	10	73	67	34	45	9	6	8
Number of bicycle accidents	320	8							
Percentage of bicyclists in violation	75	75							

By far the greatest number of juvenile fatalities resulted from accidents which are here termed "motorist fatalities," for want of a better name (Table V). They include deaths from such motor vehicle accidents as running off the roadway, collisions, railroad grade crossing accidents and any other accidents where one or more motor vehicles are involved, but excluding pedestrians and bicycles. A total of 436 youngsters in the four age groups were killed during the past five years, an average of more than 84 per year. Deaths in the three lower age groups were not alarmingly high, but those in the 15 to 19 age group alone accounted for more than 60 per cent of all the juvenile fatalities. This greater incidence, of course, is due to the fact that members of this group—the so-called "terrible teenagers"—do a great deal of driving, thereby exposing themselves to more accidents and resultant deaths. This study will not attempt to compare traffic death rates for this age group with older groups.

A number of studies have been made along this line and some comparatively reliable figures are available.

In a study of 87,566 reported traffic accidents in 1952, it was shown that 8,375 or 9½ per cent involved drivers under twenty years of age (Table VI). Their involvement in fatal accidents, however, was slightly higher, with the study showing that they were driving vehicles in eighty-three of the 640 fatal accidents, for a percentage of 11½.

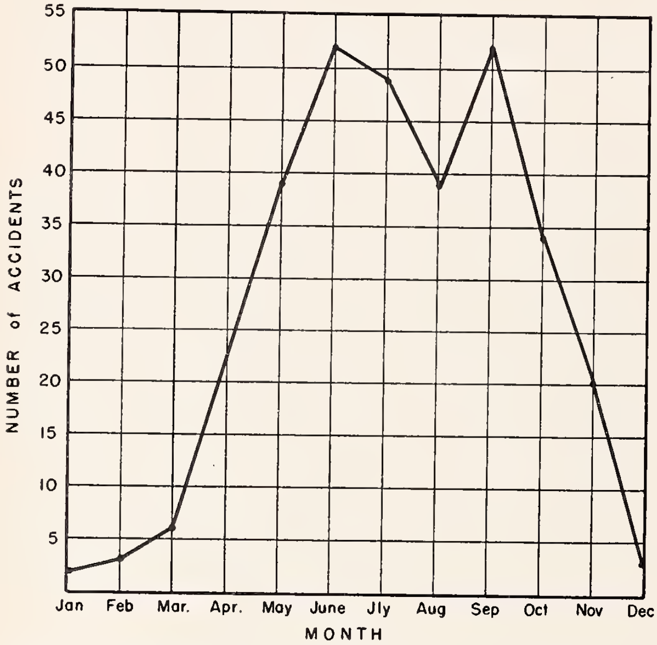

Fig. 1. Bicycle accidents by month of occurrence, Minnesota 1950-1954.

The Minnesota Department of Highways conducted a special study of bicycle accidents reported in 1953 and upon the basis of that analysis the cause for 75 per cent of the accidents was violation of traffic rules by bicyclists themselves (Table VII). The study considered a total of 320 bicycle accidents, involving all age groups, in which there were 242 violations, and of these violations, all but fourteen were charged to bicyclists under twenty years of age. The three chief violations were failure to grant right of way to a vehicle, failure to stop when leaving a driveway or alley, and improper turning.

This study of bicycle accidents also revealed that only fourteen of the 320 accidents occurred during the months of inclement, cold weather—January, February, March and December (Fig. 1). It also revealed that the first month of summer vacation, June, and the first month of school, September, were tied with fifty-two accidents each to rank as the two highest in the number of bicycle accidents.

An analysis of pedestrians' actions which resulted in fatalities during 1953 reveals that a total of fourty-two youngsters in the four age groups were killed (Table VIII). The two leading causes were crossing streets or roads at points other than intersections and running or walking from behind parked cars into the paths of vehicles.

In the above tabulations and discussion, the deaths which befell youngsters as the result of traffic accidents have been presented in some detail. The natural question that now arises is: *"What can we do to prevent a continuance of these tragic deaths to our youth?"*

Despite my background as an enforcement officer as a member and later as chief of the Highway Patrol, I believe our main hope for improvement in this particular field lies not so much in enforcement as in education. And by education I do not necessarily intend to confine this to a formalized, classroom taught method of instruction. I see it as a daily, year-round program of safety instruction, not only by teachers, but by parents, older brothers and sisters and other adults who have control or direction over youngsters.

Imitation, it has been said, is the sincerest form of flattery. The imitation by children of the actions and behavior of adults is a primary application of a basic educational principle. This is especially true among the very young children who are making their initial venturesome trips into the dangers of traffic. They remember more vividly their parents' actions than the last-minute parental admonitions, so are more likely to imitate bad and dangerous walking practices than to observe the safe-walking reminders with which they were ushered from their homes.

It would appear then, that the only way to tackle the problem of safety education for our children is to begin with a safety education program for their parents and other adults in order that their walking and driving practices will conform with accepted standards. Such acceptance of their responsibility, not only to themselves but also to their children, would be a tremendous advance in the field of traffic safety. Not only would it induce their children to "get off on the right foot" as they encounter the difficulties of present day traffic, but it would make them more receptive to further safety instruction as they grow older, begin to ride bicycles and later to drive automobiles.

The 125 youngsters in the age group 0 to 4 years who were killed during the five-year period from 1950 through 1954—an average of twenty-five per year—were, beyond any doubt, killed because of their parents' failure to exercise normal, prudent concern for their safety. The youngsters who toddled out into the street and were struck by passing cars, those others who were crushed beneath wheels of vehicles while they played in streets, other youngsters who were killed when their parents' cars ran off the road or struck fixed objects—all of these deaths are chargeable directly to the parents who failed to keep their cars under control or who failed to exercise proper supervision over their children's activities. One can put forth an academic argument that a child which runs into the street is at fault in the event of a fatal accident—but a child of less than four years of age cannot possibly know the dangers of vehicular traffic. That is a danger which such a young child cannot comprehend. It is only necessary to ask a grieving mother or father to assess the blame for their child's death—they will shoulder the blame themselves because they realize then, when it is too late, that it was up to them to so circumscribe their child's activities to rule out any chance of an accident.

In the next two age groups—from five through fourteen years of age—the parents begin to delegate some of their responsibility for the safety of their children to school teachers. It is essential that every elementary school, regardless of its size or location, include thorough instructions in every phase of safety with which a child in these age groups may be confronted. These should cover virtually every subject of traffic safety, with the possible exception of the operation of a motor vehicle. Safe and proper walking habits should be inculcated in every student. He should be told and shown what is right and what is wrong insofar as his behavior as a pedestrian is concerned. Whenever practicable, school safety patrols should be operated, not only because they have proved themselves to be the best means devised to date for the safe conduct of children to and from school, but because they are a very powerful force in shaping the walking habits of children at other times in the absence of safety patrol officers.

TABLE VIII. PEDESTRIAN FATALITIES, 1953

Pedestrian Actions	Pedestrians Killed				
		Age			
	Total	0 to 4	5 to 9	10 to 14	15 to 19
Crossing at intersection with signal	1				1
Same—no signal	3		1	1	1
Same—diagonally	1	1			
Crossing not at intersection	10	6	3		1
Coming from behind parked cars	11	4	6	1	
Walking in roadway—sidewalks not available	3		1	1	1
Playing in roadway	9	6	3		
Not in roadway	2			1	1
Not stated and other	2	1	1		
Total killed in these age groups	42	18	15	4	5

With a large number of children being transported to and from schools, another important facet of their safety education must deal with the special hazards which mass transportation involves. Here again it has been found that a well-trained bus safety patrol is the best means for assuring orderly embarking, riding and disembarking, and it too instills in the young child an awareness of the dangers which must be faced prior to embarking and after disembarking, not only when buses are concerned but at all times.

Most of the bicycle accidents fall into the two middle age groups, with many of them occurring as students are going to or from school. A complete and thorough course in the study of all traffic laws which apply to the use of bicycles, formation of bicycle safety clubs, and use of reflective materials are all weapons which can and must be used to reduce accidents and deaths to bicycle riders in these age groups.

Room for the greatest gains in traffic accident prevention among our four juvenile groups lies in the 15 to 19 year group, not only because 297 or 46 per cent of the deaths are concentrated in this bracket, but because these teen-agers are the most difficult to reach through safety education programs.

Many of these youngsters, especially in rural areas, are no longer in school and even though they may not have automobiles of their own, they do have ready access to a family car or truck. All too often, they do not have the benefit of formal classroom training in the operation of a car, but acquire varying degrees of knowledge from parents, older brothers or sisters. They have but a fleeting acquaintance with traffic laws and regulations and are quite often totally ignorant of traffic courtesy. Their idea of a "good" driver may be one who gets away first from a traffic light, gets to the next traffic light first, weaves in and out of traffic, makes pedestrians in cross-walks jump for their lives and commits all the other acts, bordering on rowdyism, which they have come to associate with their "ideal" driver. It is extremely difficult to convince such a young driver that this type of driving is wrong, that it breeds disrespect for all kinds of law and order, and that it invites traffic accidents for which he will be blamed by more conservative drivers. In fact, it is entirely possible that he will not take kindly to any of your suggestions that he tone down his driving, but you are most likely to have a challenge hurled at you to prove if he or you are the better driver. How do you get out of such a situation? Simply by accepting the challenge, driving sensibly and entirely within rules and regulations, and thus proving to your young friend that good driving does mean safe common-sense driving. Just as a young child learns good or bad walking practices by imitating his parents, so does a young and impressionable teen-ager acquire safe or dangerous driving habits from his parents.

In my opinion—and this is backed up by men and women who have been in this particular phase of safety work much longer than I—the one great hope for instilling proper attitudes and imparting good driving habits to our young people lies in the driver education program such as is presently being conducted in the high schools of our state. This is forcibly brought out in a yet-incomplete study of 3,000 drivers, one-third of them being untrained drivers, another third having had classroom instruction only, and the remaining one-third having had the benefits of both behind-the-wheel and classroom instruction. Their three-year records revealed that 231 of the first category were involved in traffic accidents, 180 in the group having had classroom instruction and only 119 in the fully-trained group.

I don't believe it is necessary to elaborate further on the results to date of the program. In my own mind I am convinced that a continued expansion of the driver training program, until it eventually is offered by every public and private secondary school in the state, is our one "great hope" in reducing our rate of traffic accidents and deaths. The youth of today, who will be the drivers of tomorrow, must be given the opportunity to learn the right way to drive if they are to be safe drivers. They must be taught what factors are combined to produce a good, safe, intelligent and courteous motorist. When we have done that we will have made motoring not only safe for youngsters, but will have laid the firm groundwork for safer driving for everyone, regardless of age.

HYSTERECTOMIES

(Continued from Page 457)

JOHNS A. HAUGEN, M.D.—I would like to congratulate Dr. McKenzie for his efforts in making such a study. Unfortunately records are so deficient that it is hard to properly classify much of the material. I believe it should be emphasized that the uterus often has to be removed without having inherent pathology. Much of our gynecological surgery entails a hysterectomy, both abdominal and vaginal, for uterine prolapse. Also, in severe degrees of cystocele, rectocele and enterocele, the most satisfactory procedure often includes removal of the uterus. The pathologist, and more recently our new tissue committees, should bear this in mind.

B. A. SMITH, M.D.—The urinary tract complications are very few in this report. In other investigations, ureteral injury is reported in 2 to 2.5 per cent of major pelvic operations and up to 30 per cent in radical hysterectomies. These figures are in part obtained in studies of ureteral injury, in which excretory urograms were made postoperatively as a routine, since the complete ligation of one ureter is silent unless infection is present above.

Lesions involving the broad ligament such as endometriosis or intraligamentous cysts and myomas are particularly prone to ureteral injury.

Discussions of this problem are more and more frequent in urologic literature and the urologist feels that preoperative excretory urography is indicated in all cases where difficulty is expected, even remotely. Inlying ureteral catheters should be placed if any trouble at all is anticipated; the ureters are then usually delineated with ease.

Seminar

THE DRUG THERAPY OF HYPERTENSION

A Review of Recent Literature

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HYPERTENSION of the essential type, as opposed to hypertension due to a specific cause like pheochromocytoma, is a disease which is a composite result of several factors acting upon the organism. Some of these factors are known as a result of direct observation in the laboratory and others are presumed to be present as a result of observations of empirical therapy. Still others are assumed to be present largely on the basis of inductive reasoning. The etiological mechanisms of pathologic physiology in this disease at present defy the utmost in investigative skill so that a definitive and rational application of therapy is almost always an impossibility. It is the purpose of this paper to discuss the merits and limitations of some of the drugs available to the internist and general practitioner for the treatment of this disease. Thus, one may be able to offer to many patients the hope of a relative state of comfort and continued productive existence.

Investigators have generally found it useful to start with an analysis of the disease, breaking it down to fit into two categories. The first is a system of evaluation of the hypertension per se. This is exemplified in the following table from Schroeder[12] (Table I). Table II is the classification of funduscopic changes as listed by Schroeder.[12] Special attention is directed towards the fact that the prognosis of the disease is fairly benign until one reaches diastolic pressure levels of 120 to 160 at which time the patient is at least in a stage of severe benign hypertension if not in the malignant phase of the disease. From this time on, the course of the disease is measured in years to weeks. Apparently, the three principal dangers that will affect the patient will consist of effects on the heart from overwork, deterioration of the kidney with the onset of arteriolar nephrosclerosis, and the acceleration of the onset and progression of generalized atherosclerosis.[1,3,12,13] With this in mind, the therapist must consider not only the fact that he can lower the blood pressure, but also must consider the effects of inducing a state of relative hypotension in an organism physiologically adjusted for years to hypertension. The lowering of blood pressure in a diseased kidney may produce disastrous effects by impairing glomerular filtration rate and affecting tubular function so that a uremic state is further aggravated.[7,9,13] Thrombosis might be induced in diseased arteries.[3,7,9,11] The drug used also may possess side reactions which if not recognized or offset, may cause harmful effects.[1,9] This last possibility applies to all the drugs which will be discussed in the following paragraphs, for although some are better than others in specific cases, none of them approach the ideal, which would be a drug which reduced blood pressure without possible side effects severe enough to threaten the life of the patient.

The second approach to the evaluation of the disease in the patient is the determination of the factors which are most likely to be causing the disease. As previously noted, these factors are rather nebulous, but they are useful in that they can lead to a more intelligent approach to the problem of therapy. In other words, although many drugs cause a lowering of hypertension, they are not interchangeable, for their modes of action vary, and it should be emphasized that the effectiveness of any drug cannot be accurately predicted. It is evident, therefore, that there is no substitute for a trial period of treatment.[1] In many cases of mild hypertension, good results may be obtained by the judicious application of basic psychotherapy, and if drug treatment is considered, an appropriate dosage of a mild sedative may be all that is necessary.[12,16] In the more severe cases of hypertension, such palliative methods are generally ineffective, and the drugs that will now be discussed are generally felt to be

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TABLE I. STAGES OF HYPERTENSIVE DISEASE

Stage	Approximate Diastolic Pressure, mm.Hg.	Ocular Fundi Grade	Rate of Progress	Remarks
0 Prehypertension	85-95	0	Variable	Asymptomatic. No damage unless it progresses to other stages.
I Mild benign	90-105	0-I	Variable	Consistent with long life. May reverse to normal.
II Moderate benign	105-120	I-II	Slow	Consistent with long life unless complicated. Occasionally reverses to normal.
III Severe benign	120-160	II	Usually rapid (years)	Apoplexy and heart failure may develop. Long life rare.
IV Malignant				
(a) Early	130-160	III	Usually rapid but reversible	Danger signal for downhill course.
(b) Severe	130-200	IV	Rapid (months)	Usually fatal unless interrupted.
(c) Decompensated	130-200	IV	Rapid (weeks)	Renal insufficiency and heart failure. Always fatal.

NOTE: The course of the disease can be modified at any stage by the development of atherosclerosis or other vascular disease. The estimations in the above table were made on the assumption that this would not appear in the normal course of events.

Early malignant hypertension as used here is a clinical condition and often represents the transition between the benign and malignant stages. High, fixed diastolic pressures, diminution of renal function and retinitis, usually either severe or with blurring of the nasal margins of the optic discs are present. This is not to be confused with the pathological condition found in renal insufficiency, "malignant sclerosis" with necrosis of arterioles.

—Schroeder, VA TB 10—102, 1954.

TABLE II. CLASSIFICATION OF CHANGES IN OCULAR FUNDI

Grade	Arteries	Veins	Retina	Disc	Interpretation
I	Constricted. May be segmented.	Normal	Normal	Normal	Benign hypertension, young individual, or short duration.
II	Sclerotic. Tortuous, increased light reflex.	Normal or full. Nicking at A-V crossing.	Normal	Normal	Benign hypertension of long duration, or with arteriosclerosis.
III	Same as I or II. May show blurring.	Full.	Hemorrhages and/or exudates of all varieties and sizes.*	Normal or nasal margin blurred.	Early malignant stage beginning. Severe stages present. Danger signal.
IV	Blurred, perivascular edema. Constriction intense.	Full.	Same as III* May be pale. Edema.	Swollen 1-5 diopters.	Malignant stage full blown. Rapid breakdown imminent. Increased intracranial pressure.

NOTE: Vision is usually normal in grades I and II unless severe arteriosclerosis has developed. In grades III and IV, scotomata are present, depending on location of lesions. Vision may vary from no noticeable change to complete blindness. Detachment of choroid or retina can occur.

*Must be differentiated from thrombosis of central vein or artery, usually unilateral.

—Schroeder, VA TB 10-102, 1954.

specific antihypertensive agents and, as such, apparently act by affecting neurogenic, nephrogenic and possibly humoral mechanisms.

Potassium Thiocyanate

This drug has the distinction of being about the oldest available drug used in this country for the control of hypertension, having been used for some fifty years.[4] However, with the advent of the newer drugs and because of the dangers inherent in its use, especially over a prolonged period of time, it has perhaps fallen into disfavor. This is unfortunate, for the drug has real value in the treatment of headaches secondary to hypertension, and apresoline therapy, and has value as a supplementary aid to other anti-hypertensive drugs which are ineffective in alleviating the associated headache. A dramatic and marked effect may be noted in the intravenous administration of this drug.[1]

Besides its effect as a smooth muscle relaxant, the thiocyanate ion also is likely to produce symptoms characteristic of iodism. The prediction of which patient will develop toxic reactions to the drug is not possible, although it is generally agreed that the serum thiocyanate level in the blood should never exceed 15 mgm. per cent. Even at this level, mild toxic reactions may be noted, and above this level, the danger of severe toxicity is very real. A blood level of 4 to 8 mgm. per cent will usually be as effective therapeutically as higher concentrations. It is essential that periodic checks be made on the blood thiocyanate level, for this tends to fluctuate markedly, even in the same individual.[1,3] The usual initial dose is 0.3 gram of the sodium or potassium salt daily for three days. In a report of a case from the Mayo Clinic, a patient's headache was obviated within two hours after receiving 60 cc. of a 5 per cent solution of sodium thiocyanate I.V. Maintenance dosage for this patient was 0.4 to 0.6 grams of potassium thiocyanate daily, and at time of discharge in three weeks, his blood-cyanate level was 8.3 mgm. per cent. A final note of caution is that intolerance to the drug cannot be determined in advance,[1,4] and yet it is felt that in conjunction with the other drugs that make up the armamentarium of the hypertension therapist, this drug has definite value for the conditions noted above.

The Veratrum Compounds

Since it is not clear whether the phenomenon of essential hypertension is due to hyperactivity of the sympathetic system or to hypoactivity of the parasympathetic system, one of the approaches to therapy is the use of drugs which presumably act principally as stimulators of the parasympathetics. These drugs act at the hypothalamic level and appear to induce their hypotensive effects by inducing a decline in cardiac output, bradycardia and an evenly distributed reduction in peripheral resistance.[6] One of the major drawbacks to the use of these drugs is that the therapeutic and toxic levels are so close together that the patients often experience nausea and vomiting. Because of bradycardia-inducing properties, and possible positive inotropic action of the drugs, they have often been used in hypertensive patients with tachycardia. However, because of their vagomimetic action, they should be avoided during the time of rapid digitalization for fear of causing heart block. This latter complication can be relieved by the intravenous administration of 1 mgm. of atropine sulfate.[7] In general, these drugs are most beneficial in the treatment of moderate and mild hypertension. In cases involving reduced renal function with azotemia, the drugs may be more effective than hexamethonium which may be poorly excreted. No special contraindications to their usage are noted except those which apply in general to any hypotensive agent; for example, their use in atherosclerotic patients wherein the dangers of thrombotic complications are a definite risk.

Available preparations vary in their composition of alkaloids derived from Veratrum viride. Vertavis contains whole powdered Veratrum viride and potency is measured in carotid sinus reflex units (CSR). It is available as an oral tablet of 130 CSR units, and the recommended total daily dosage is 3 to 9 tablets. Other preparations are Veriloid, Vergitryl, Unitensin, Veralba and provell maleate. These last two contain protoveratrine A and B, a single ester alkaloid of Veratrum alba. The usual dose of protoveratrine is 800 to 2000 micrograms or about a tenth of the dosage of veriloid. One recommended dosage schedule[1] is to give 200 to 250 micrograms before meals and at bedtime and to increase the dose daily by 100 to 200 micrograms until the blood pressure is satisfactorily reduced or until side effects occur. If at a level of 500 to 750 micrograms four times a day the blood pressure is still high, it is doubtful whether larger doses will be effective. The dosage schedule recommended by Hoobler[8] for orally administered protoveratrine is presented. The initial large dose (.50 to 1.5 mgm.) is given after breakfast at 8:00 a.m. A reinforcing dose of .25 mgm. is given at 10:00 a.m. Another dose of .25 mgm. is given after lunch at 1:00 p.m. If the patient does not respond when the initial dose has been increased to 1.5 mgm., it is unlikely that the drug will afford any benefit. The tablets are taken after meals or at least one and one-half hours before the next meal. The initial doses in the morning and evening must be determined empirically, but they usually range from 750 to 1500 micrograms. Finally, it is noted that if one of the drugs in this group has no effect, it is quite unlikely that any of the others will be any more effective.[1,7,8]

Rauwolfia Derivatives

In the past five years, this group of drugs has been used in this country with varying degrees of success, depending upon the selectivity of cases studied. These drugs act as mild sympathetic inhibitors, parasympathetic stimulators and possibly have a non-specific effect which accounts for a tendency for the patient to show an increase in appetite, to gain weight and to develop languor and a sense of well-being.[16] The various preparations are the crude root (Raudixin), the alkaloidal extract (Rauiloid, Rautensin) and the single alkaloid, reserpine (Serpasil, Reserpoid, Serpiloid). Like the veratrum compounds, these drugs are most effective as solitary agents in the milder types of hypertension. Also, if one of these preparations is ineffective, the probability is that the others will be of no avail. When effective, the blood pressure reduction is gradual and is smoothly maintained, but this probably occurs in only about 50 per cent of mild labile hypertensives.[1] It appears from the literature that these drugs have their greatest use as an adjunct to the control of hypertension with the use of the more potent ganglionic blockers.[16] As an example, they have been useful in the combined treatment of hypertension with apresoline (1-hydrazinophthalazine) and with hexamethonium. In the case of apresoline, these drugs appear to be able to help relieve the patient of the annoyance of the tachycardia that is not infrequently attendant on that drug's use. They also tend to oppose the gastrointestinal stasis that is a serious problem in the use of hexamethonium.

The problem of dosage varies with the individual therapist and, in general, most methods start with divided doses several times a day, although according to one report from the Mayo Clinic, there is no reason why the drug cannot be given once or twice a day.[1] The effect of the drug is cumulative, and it takes from two to four weeks before most patients show any effect. Since these drugs are often used in conjunction with the more potent hypotensive agents,[1,3,14,16] it should be remembered that if the drug is withdrawn, there may be no change in the clinical picture over a period of a week or more.[1,7] This is even true if the drug is used alone. Finally, it has been suggested by Wilkins that these drugs might be found to be of use in the stabilization of the pre-hypertensive individual; that is, one who has a labile blood pressure that tends to skirt the limits of sphygmomanometry above which the hypertensive state presumably begins.[12,16]

Ganglionic Blocking Agents

One of the approaches to the treatment of hypertension has been an effort to block the chain of impulses that lead from the cerebral cortex to the peripheral blood vessels. The chemical mediators in this system are acetylcholine and norepinephrine which transmit nerve impulses through ganglia and from nerve endings to smooth muscles, respectively. As noted above, the Rauwolfia drugs probably have their effect through an interference in this system, and in addition there are now available two agents which are clinically feasible and which act principally on the ganglia, hexamethonium and pentapyrrolidinium bitartrate (Ansolysen).

Hexamethonium is a quaternary ammonium compound and is a chemical analogue of acetylcholine. It probably acts through competitive inhibition, blocking nerve transmission through the ganglion. Besides inhibiting the sympathetic impulses it also blocks parasympathetic nerve transmission, and this is the source of its principal and unfortunate side effects. Among these are paralysis of the muscles of the eye, gastrointestinal stasis and hypotonicity of the bladder.[9,13,14] The last two are especially embarrassing, for the effectiveness of oral administration will depend on normal intestinal activity and uneven absorption will obviously impair the effectiveness of therapy. The creation of urinary obstruction, besides causing acute discomfort to the patient, will have adverse effects on kidneys which are likely to already have impaired function due to hypertensive effects. These side effects often tend to become less intense as the drug continues to be used and is possibly related to the drug tolerance that is seen in its use[14] and which will be discussed shortly. It is imperative that therapy include the use of laxatives and the use of drugs like urecholine and/or prostigmine.[3,14] When the drug is used to optimal advantage, Schroeder and his group[9,13] noticed hardly any evidence of drug tolerance. However, it was their observation that if the drug was discontinued or not given properly that the patients were likely to show a return to hypertensive levels and that the control of the disease then became much more difficult. Emphasis is placed on the fact that this drug is a much more potent hypotensive agent than the ones mentioned previously and that although, because of its rapid excretion in the urine, it has to be given every four hours it is essential that every effort be made to educate the patient so that he is able to recognize the evidence of changes of blood pressure levels and adjust his dosage accordingly. Ideally, this is done by taking the blood pressure immediately preceding the next dose of medication. The pressure is measured in the sitting position. It is of interest that in the initial treatment of hypertension, using a combination of 1-hydrazinophthalazine (Apresoline) and hexamethonium, Schroeder[13] has the blood pressure taken in the supine position, and it is only when the patient is being evaluated for maintenance therapy that the readings are taken in the sitting position.

The dosage schedule of hexamethonium as shown here is the one recommended by Schroeder's group and is given in combination with Apresoline.[13] The patient is started with 125 mgm. every four hours, and this is increased every day by 125 mgm. a dose till 500 mgm. per dose is being given. At this time, Apresoline is started at 25 mgm. every four hours and increased every day by 25 mgm. a dose until 100 mgm. a dose is being given. After this, the dose of hexamethonium is controlled by the blood pressure (systolic) level as follows: over 140, full dose; between 140 and 130, ½ dose; between 130 and 120, ¼ dose; below 120, no medication. The Apresoline itself is continued regardless of the blood pressure and is only discontinued if the patient starts to show toxic reactions to that drug of an uncontrollable degree. When hexamethonium is used alone, the dosage as recommended by Hoobler[7] starts out with 125 mgm. after meals and at bedtime and increases to 750 mgm. per dose as required until at least a systolic pressure of 160 to 180 or preferably of 110 to 120, standing, is attained. A daily laxative is given. If a sphygmomanometer is unavailable, the patient is instructed to stand motionless for one minute before each dose and, if dizziness occurs, the intended dose is halved. Rauwolfia is used concomitantly and occasionally Apresoline. Also, if constipation is present for twenty-four hours, the medication is halted until a bowel movement occurs. In acute emergencies, the drug may be given intravenously at the rate of 1 mgm. per minute. Early in the control of hypertension with this drug, it is recommended that the patient be given the drug parenterally in doses of 5 mgm. every two to four hours and that a rigid control of the blood pressures be taken with variation of succeeding doses depending on the response of the patient. Care should be exercised to avoid excessive falls of blood pressure for fear of further compromising renal blood flow, reducing glomerular filtration rate and impairing the excretion of the drug. Since this drug has such a strong hypotensive action, there has been some degree of dissention about its use in patients with uremia[7] and in those with generalized arteriosclerosis. The limits of uremia beyond which one should proceed with real caution have been set at about 60 mgm. per cent of non-protein nitrogen. This also applies to Ansolysen.

Recently, there has become available in this country another ganglionic blocking agent which seems to offset some of the disadvantages of hexamethonium. This is pentapyrrolidinium bitartrate which will be referred to by its trade name of Ansolysen. It is five times more active than hexamethonium in producing block,[5,14] and the average duration of action is about 40 to 46 per cent longer than with hexamethonium.[5] Given intravenously, whereas hexamethonium is apt to cause a precipitous fall in blood pressure, the response to Ansolysen proceeds more gradually over a ten-minute period. Although this does permit some tendency to exceed the effective dose, this can be counteracted to some extent by having the patient sit up in bed and noting the appearance of hypotensive effects. The mean effective oral dose is about twenty times the effective parenteral dose and is thus somewhat similar to the relationship existent with hexamethonium. However, the predictability of response is much better and more rapid than with hexamethonium in that there is a demonstrable effect in one hour after an effective dose.[5] There is also less daily variation and the extent of tolerance is less than that shown with hexamethonium.[5,14] The degree of cross tolerance between the two drugs is so slight that if a patient were changed from one drug to the other, i.e. from hexamethonium to Ansolysen, the mean effective dose would vary so slightly as to be clinically insignificant. Although the potentiating factors that affect the action of drugs like these are still significant, they are not as marked with Ansolysen as with hexamethonium, in that the variations of blood pressure levels produced generally are not so severe as to require the strict observation which is necessary with hexamethonium. These potentiating factors are noted to be as follows: Ambulation, prolonged standing, alcoholic intake (even a relatively small amount), heavy eating, exercise, and sweating. Along with this last, any form of salt-depleting situation, whether it is by a restricted diet or the use of mercurial diuretics, is likely to cause some embarrassment.[5] Side effects are about the same as with hexamethonium, although not so severe. Freis and associates state that many of their patients required glasses to compensate for lack of accommodation. Impotence was a frequent and troublesome side effect in the male, this being more marked in the older age group. Urecholine (urethane of B-methylcholine) was used to combat this with questionable effect. None of their patients had trouble emptying the bladder, although this is a common problem with hexamethonium therapy. Certain side reactions, like dryness of the mouth and frequent postural hypotension, cleared up after the early stages of treatment although others persisted during the course of therapy.

The therapeutic dose used by Freis[5,18] varies according to the severity of the disease. A combination of Neostigmine, Reserpine and Ansolysen is recommended. Table III is published by Wyeth.[18] Because of Ansolysen's prolonged effect, the drug is given only three times a day at roughly eight-hour intervals instead of every fourth hour as in the case with hexamenthonium.

TABLE III. ORAL DOSAGE SCHEDULE

NOTE: When the desired reduction in blood pressure is achieved, dosage of Ansolysen is stabilized and no further increases are made. Each patient is evaluated independently and the dosage schedule is deviated from freely as required by the response of the patient.

Hypertension Type	Drug	1st Week	2nd Week	3rd Week	4th Week	5th Week	6th Week	7th Week
Mild, labile	Reserpine	0.5 mg. b.i.d. for 2 to 3 weeks. Dosage may then be reduced to 0.5 mg. or less daily, depending on therapeutic response. As little as 0.1 mg. daily may be sufficient.						
Moderate to severe	Neostigmine*—upon arising	0	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.
	Reserpine—1st dose before breakfast 2nd dose 5 p.m.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.
	Ansolysen—1st dose after breakfast 2nd dose mid-afternoon 3rd dose before bedtime	0	20 mg. t.i.d.	40 mg. t.i.d.	60 mg. t.i.d.	80 mg. t.i.d.	100 mg. t.i.d.	150 mg. t.i.d.
Severe or malignant	Neostigmine*—upon arising	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.	15-30 mg.
	Reserpine—1st dose before breakfast 2nd dose 5 p.m.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.	0.5 mg. b.i.d.
	Ansolysen—1st dose after breakfast 2nd dose mid-afternoon 3rd dose before bedtime	20 mg. t.i.d.	40 mg. t.i.d.	60 mg. t.i.d.	80 mg. t.i.d.	100 mg. t.i.d.	200 mg. t.i.d.	300 mg. t.i.d.

*If ineffective, take a laxative at bedtime.
Schedule used by E. D. Freis, M. D., personal communication, as printed by Wyeth (18).

As a final note on the use of this drug, it was found that if the constipation was not controlled by the neostigmine, then it would be best that the drug be withheld till the patient has had a bowel movement. Although syncopal attacks are seen with this drug, the prolonged collapse reactions often accompanied by ileus are not seen as they have been with hexamethonium. Schroeder[11] mentions the use of Ansolysen as a substitute for hexamethonium and states that preliminary observations in his small group were such as to suggest that it may be more effective in providing control. (As noted above he uses a combination of Apresoline and a ganglionic blocker, viz. hexamethonium). Smirk[14] reports that of eighty patients who had been on Ansolysen alone and had not been doing well, sixty benefited by the addition of reserpine 0.5 mgm. twice or three times a day. In these cases, the blood pressure was under better control or there was further freedom from side effects. Late toxic effects with Ansolysen have not been reported. However, in his observations on treatment of hypertension with Apresoline and hexamethonium, Schroeder[9,13] reports the appearance of an acute interstitial fibrosis of the lungs occurring in five of his patients, which was rapidly fatal. Four of these were Negroes, four were males, three had congestive heart failure, and three had renal insufficiency. All had the malignant phase of the disease, and it was felt that the hexamethonium ion may have been the offending agent in this group.

1-Hydrazinophthalazine

This drug is marketed under the name Apresoline and will be referred to as such in the following comments. It is the most effective of the phthalazine compounds and may be classified as a potent hypotensive drug. The mode of action is obscure, but it does seem to locate its action more or less directly on the blood vessels. Experiments have been reported wherein the drug was injected into the femoral artery causing vasodilatation on the injected side while the blood vessels on the contralateral side were unaffected.[12] Whether the mode of action involves some humoral agent or affects an enzyme system concerned with vasoconstriction is not known. While it lowers blood pressure, it also increases renal blood flow and increases cardiac output.[15] It may also cause tachycardia.[15] It may cause an increase in the urinary excretion of sodium. It is interesting to note that pherentasin, a pressor substance obtained from extracts of hypertensive blood, is no longer pressor when treated with this drug.[12] The hydrazine grouping is responsible for this activity. In man, the hypotensive effect may last several hours after injection. The rate of excretion is rapid and effective utilization has been based on frequent two- to four-hour doses.[13,15]

The hydrazines are anti-histaminases *in vitro*, and the side effects seen with these drugs are probably related to the fact that the histamine-destroying enzyme is inhibited.[12] The milder reactions are quite numerous and include lassitude, coryza symptoms, nausea and vomiting. Some patients show a slight fever, back pain, generalized malaise and prostration while on the drug, and recovery upon removal from its influence. Although this sort of reaction usually appears shortly after the start of therapy, it may not appear for two or three weeks. Another unusual effect is the occasional appearance of a "glove and stocking" type of paresthesia which shows up with the administration of high doses but which is relieved upon lowering the dose of the drug.[9] Severe exacerbation of neurodermatitis has been reported and angina pectoris can be made worse. In one case observed by the writer, the patient had been on the drug for about a week and suddenly developed marked tachycardia, increased angina and a protodiastolic gallop. The drug was immediately stopped, and the patient completely recovered in about twelve hours. The reaction can be more severe, and the electrocardiographic changes noted suggest a state of ischemia.[9,13,15] After the patient has been on the drug for about a month, the cardiac output drops towards normal and there is a decrease in palpitations and tachycardia. If the dose is increased, there is likely to be a return of symptoms. Of the group treated by Schroeder et al,[9,13] thirty-five had suffered a coronary occlusion before onset of treatment. On Apresoline and hexamethonium therapy, thirty-two were evaluated for this factor, the other three patients not being included because of either discontinuance of therapy, sudden death, or recurrence. There were no further attacks in the remaining thirty-two. In the rest of the group being studied, there were occasions of cardiac involvement ranging from electrocardiographic changes to actual attacks, three fatal and three non-fatal.[11]

Four patients who showed the electrocardiographic changes of bundle branch block and subendocardial ischemia, showed their reactions during induction of control. Of significance, though, is the finding that in the smaller control group, there were three deaths and two non-fatal attacks. Sudden deaths, possibly of cardiac cause, occurred three times in the treated patients and ten times in the controls. Of ten patients who suffered from angina, eight are asymptomatic, one requires nitroglycerine about once a month as compared to a former need of six to eight times a day, and the one patient that discontinued treatment still has his angina unrelieved.[11]

Late toxic reactions are apparently related to the creation of a collagen-type of disease.[9,12] Among the first symptoms is a polyarthralgia which may be indistinguishable from acute rheumatoid arthritis. It is interesting that the thymol turbidity and cephalin flocculation tests are positive, while the bromsulfalein test is normal. Following this, the patient develops a syndrome suggestive of disseminated lupus erythematosus. The "L.E." cell test may be positive. Reduction of the dose may improve the arthritis but the "lupus" occasionally progresses. Apparently, this phenomenon is relatively rare, and Schroeder mentions that in his experience he has not found it to be a contraindication to the use of the drug in severe and malignant hypertension. As to the mechanism of action, it is hypothesized that it may be the result of true drug sensitivity, or allergy, or from depletion of some necessary trace metal, coenzyme or other substance.[12]

The drug, as used alone, is generally ineffective, although in a few patients it has been shown to induce a very satisfactory state of hypotension. Moyer[10] starts his treatment with 25 mgm. at mealtimes and at bedtime. After a week or so, the breakfast and night doses are increased to 50 mgm., and after several days the noon and evening doses also are raised to 50 mgm. Using such a regime, thirty-five per cent of patients showed a good response after three months, but at the end of two years only 9 per cent of the patients still had adequate blood pressure regulation. Stunkard[15] reports a higher figure of favorable response, about 70 per cent, but his series ran for a shorter period of time. The dose schedule used was 10 or 25 mgm. every four hours from 6:00 a.m. to 10:00 p.m. This was increased by 10 or 25 mgm. increments until blood pressure responses had been met or side effects limited dosage. The average daily dose was 530 mgm. (range 250-1000 mgm.). The schedule, as used by Schroeder and his group, has been already listed in the discussion on hexamethonium.

Sympatholytic Agents

There are available several agents in the pharmacopeia which act by blocking the action of epinephrine and/or norepinephrine without blocking the transmission of impulses at the neurovascular junction and also drugs which induce a total adrenergic blockade. Unfortunately, none of these drugs has enough consistency of action, significantly minimal side effects or prolongation of action to warrant their use in long-term antihypertension therapy. The most important ones are as follows:

Benzodioxane and regitine are used primarily in the diagnosis of pheochromocytoma-induced hypertension. They are short acting and tend to produce tachycardia.

Dihydroergotamines show favorable though transient effects on hypertensive states. Unfortunately, they cannot be taken by mouth. The B-alkylamines, especially dibenamine and dibenzyline, are useful drugs since they are very effective in blocking the effect of epinephrine. However, the small percentage of patients that respond, the high level of distasteful side reactions and the fact that dibenamine is practically non-effective by mouth have led to the preferential use of other drugs.[2]

Summary

1. A brief review of the types and degrees of essential hypertension is presented. When the physician has completed his differential diagnosis, it is necessary to come to some conclusion as to the degree of hypertension present for upon this decision a rational approach to drug therapy can be made.

2. Some of the more important and common drugs are discussed including their indications, contraindications and limitations.

3. At present, there is no ideal hypertensive agent, i.e. a drug which will satisfactorily lower the blood pressure without concomitant obnoxious side effects.

4. The problem of combating hypertension should be met with optimism for with information and skill the present-day physician has at his command the wherewithal to apply effective treatment in a great many of his cases.

5. With the limitations of available drugs, the physician should not hesitate to use combined therapy, for in such an approach very often lies the goal of maximum good. Except for a certain number of mild hypertensives, there is no one drug available which will adequately treat hypertension.

Acknowledgment

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LEUKEMIA OR LEUKEMOID REACTION?

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THE QUESTION posed in the title of this report is one that plagues every physician from time to time, especially internists and pediatricians; the pathologist is usually called on to make the differentiation when such a question arises. On the one hand, a diagnosis of leukemia presently carries a death sentence, while on the other, a leukemoid reaction, which may very closely simulate dreaded leukemia, usually carries a good prognosis.

In attempting to settle this question, the physician must keep a number of things in mind. The first of these is the fact that a great increase in the number of leukocytes, with the appearance of immature cells in one or more series, does not necessarily spell leukemia. Certain conditions may give rise to high levels of granulocytic cells, sometimes with decided immaturity. Among these are the various hemolytic anemias, concerning which mistakes are often made. Leukocytosis following severe hemorrhage may mock leukemia, and there are a host of infections that sometimes do the same thing, particularly pneumonic and bacteremic states. How often has the leukemoid phase of Hodgkin's disease been called leukemia, and how many times have marrow-destroying diseases been missed because of the leukemoid pattern produced by such diseases in the peripheral blood? Pediatricians and other physicians caring for young children are particularly disturbed by diseases accompanied by lymphocytosis and closely resembling lymphocytic leukemia. The high lymphocyte counts of pertussis have been miscalled leukemia many times, and physicians have been repeatedly fooled by so-called infectious lymphocytosis. Infectious mononucleosis can so closely mock leukemia that only a skilled microscopist can separate them, and even he may need the help of other laboratory studies.

The second thing the physician should keep in mind is that the present-day diagnosis of leukemia usually is not made from a study of smears of peripheral blood alone, except in cases of the most obvious and flagrant disease. Other studies are needed, of which the most important is study of the bone marrow. This is a form of biopsy and it is as important to the practice of hematology as is surgical biopsy in the practice of surgery. It establishes a diagnosis in the case of granulocytic leukemic states and rules out other conditions resembling leukemia. It may not be so valuable in lymphocytic leukemia and lymphocytic leukemoid reactions, for occasionally the marrow is not involved in lymphocytic leukemia until relatively late in the disease. In the vast majority of cases, however, the bone marrow has become involved by the time the patient becomes subjectively ill.

Some physicians contend that a diagnosis of leukemia should never be made without confirmatory studies of the bone marrow or biopsy of a lymph node. The study and interpretation of bone marrow are highly specialized procedures. One must have properly interpreted studies of bone marrow to differentiate leukemia from similar states. If the physician cannot do this himself, he can at least learn to make reasonably good preparations of bone marrow and send them to the pathologist; better yet, he can refer the patient to the pathologist for hematologic studies. A detailed history is particularly necessary to the one who must interpret the bone-marrow picture.

The third thing the physician must remember

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is that true leukemia is not always an overwhelming and rapidly fatal disease. It is true that, on the average, those who have the acute forms live less than 6 months and those who have the chronic forms live about 3 years, but there are many exceptions. Leukemia is sometimes a slowly developing state and may require months or even years before manifesting itself. Several patients come to mind to illustrate this point. One was a sixty-year-old man in whom moderate lymphocytosis was found on study of routine smears for differential count. Lymphocytosis was still present a year later but the cells were mature and normal in appearance. He returned eighteen months later with an upper respiratory infection and lymphocytosis again was found. Studies of bone marrow at that time revealed dense infiltration of the marrow by immature and abnormal lymphocytes, and a diagnosis of lymphocytic leukemia was made. Today, five years later, this man is alive and feels well, although he has not received any particular treatment. Only recently has the spleen become palpable. A second patient was a forty-year-old woman who had had lymphocytosis for fifteen years before the first study of her bone marrow was made; this examination revealed only a mild degree of lymphocytic infiltration. Repeated studies of bone marrow at four-month intervals disclosed slowly but steadily advancing infiltration and now, after four years, she has fully developed and easily recognized chronic lymphocytic leukemia. A third patient had had persistent granulocytic leukocytosis for more than seven years. Studies of marrow revealed only granulocytic hyperplasia, but now he is frankly leukemic. Now and then we see patients with leukemia in whom the peripheral blood may show only anemia or pancytopenia.

The physician must bear in mind that repeated observations often are needed to establish a diagnosis of leukemia or to rule it out. New histochemical procedures, not yet generally available, eventually may prove to be of great value. In the meantime, careful observation and adequate hematologic studies will distinguish between many cases of true leukemia and their annoying benign counterparts immediately or after a short period of observation. A few cases must be studied repeatedly and for a long period before a final diagnosis can be made.

"DIRT-EATING" WIDESPREAD IN PARTS OF TEXAS

The practice of dirt-eating—called geophagia—in some areas of North and East Texas is apparently widespread, according to two Dallas radiologists.

In *Radiology* (January) they have alerted their radiological colleagues to watch for the x-ray manifestations of this unorthodox diet regimen.

Making the report are Dr. R. S. Clayton and Dr. Paul H. Goodman, both associated with the Southwestern Medical School of the University of Texas, in Dallas.

They point out that geophagia may be suggested whenever there is an unexplained increase in the radiopacity of the fecal content of the colon. "Since learning the roentgenographic findings in geophagia, we have interviewed many patients who eat dirt or who have eaten it at sometime during their lives," say Drs. Clayton and Goodman.

"We believe the practice is quite common among some females of North and East Texas. Pregnant women occasionally are known to eat dirt but all do not have the habit as frequently as others. Men seem to eat dirt only rarely," they add.

"The patient will often deny the practice unless he is assured by the doctor that no harm or publicity will result from the admission," Drs. Clayton and Goodman continue.

They add that a lack of certain essential dietary factors may cause the craving for the dirt, but they point out further there is little evidence to suggest that the eating of reasonably small amounts of dirt is in anyway injurious to humans.

"The recognition of geophagia may provide a clue to the patient's difficulties for which the roentgenographic study of the abdomen is made," the two Texas doctors conclude.

Current Concepts

THE USE OF QUINIDINE IN CLINICAL MEDICINE

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QUINIDINE is used principally in the treatment of cardiac arrythmias. Since it is a potent drug, it is important that one be well informed as to (1) its pharmacological behavior and (2) when and how to use it.

Pharmacology and Toxicity

Quinidine is a myocardial depressant. It depresses the sinoauricular node, increases the refractory period and decreases conduction time. It causes a prolongation of the P-R, QRS and S-T intervals. It is antagonistic to acetylcholine, which probably initiates the heart beat.[1] Death during the course of quinidine therapy is often due to either asystole or respiratory paralysis. Toxic reactions to quinidine vary from patient to patient and in even the same patient, so that dosage must be adapted to individual needs. Some workers have placed emphasis on blood serum levels as a yardstick for dosage. Perhaps in the proper hands and with very good facilities for measurement, such a method is safe. However, because of the great variations both in individual toxic reactions and in recommended blood serum peak levels, it would seem hazardous indeed to attempt to attain a predetermined level in all patients. Strict attention to blood serum level may leave one unwarned as to impending toxicity. Also, restoration to normal rhythm may take place hours after the peak level has been reached.

When and How to Use Quinidine

Extrasystoles, without evidence of underlying heart disease, can often be eliminated by administration of 3 grains of quinidine, two to three times a day for a few days. The ectopic centers are so sensitive to the action of quinidine that it may be given even in the presence of organic cardiac disease. When such disease is present, 1½ grains given twice daily is often effective and not particularly hazardous. In acute paroxysmal auricular tachycardia, quinidine may not be necessary at all. Pressure on the carotid sinus will terminate the tachycardia in a fair percentage of cases, or the administration of a fast-acting digitalis preparation, such as cedilanid, will often prove successful; if both fail, the use of 3 grains of quinidine every two hours will usually correct the tachycardia.

Digitalis alone often terminates an acute attack of auricular flutter and fibrillation. The flutter may be converted to fibrillation and spontaneously to sinus rhythm. Should digitalis prove unsuccessful, the use of 3 grains of quinidine every two hours will usually correct the tachycardia.

Ventricular fibrillation calls for heroic measures. It is usually a complication of coronary thrombosis of coronary sclerosis. Quinidine should be given in 3 grain doses every hour. If death appears imminent, it should be given intravenously, as recommended by Armbrust and Levine[2]: 0.6 gm. of quinidine hydrochloride with 200 cc. of 5 per cent glucose in distilled water given by slow drip over a period of about one hour, with frequent electrocardiography as a guide. The injection must be stopped immediately when normal rhythm is established or when significant disturbances in conduction occur.

My method for the treatment of chronic auricular fibrillation is based on extensive experimental studies plus the experience of many years of clinical application. It must be emphasized that a fibrillating heart is not a fully compensated one. It is 15 to 80 per cent less efficient than one beating regularly. The administration of a cardiac depressant to an already embarrassed myocardium may aggravate the condition. Therefore, it is necessary to restore the heart muscle to the optimum physiological state before quinidine therapy is started. Digitalis must be administered until the heart rate is reduced to 70 to 80 beats per minute. Once the heart is fully digitalized, a maintenance dose of digitalis must be established and continued. Quinidine therapy is then begun. The drug may be more safely given if administered in small doses with very gradual increments. The size of the dose and the rapidity of administration determine the intensity of the reaction. This aids in establishment of a tolerance for the drug so that the patient is less likely to develop toxic reactions. I advocate the schedule given in Table I.

Doctor Weisman is Clinical Associate Professor of Medicine, University of Southern California.

TABLE I

Day	8:00 a.m.	10:00 a.m.	12:00	2:00 p.m.
1	0.2 gm. (gr. 3)			
2	0.2	0.2		
3	0.2	0.2	0.2	
4	0.2	0.2	0.2	0.2
5	0.33 gm. (gr. 5)	0.33	0.33	
6	0.33	0.33	0.33	0.33
7	0.33	0.67 gm. (gr. 10)	0.67	
8	0.67	0.67	0.67	

This is a flexible schedule. At the first sign of toxicity, the schedule of the previous day (or earlier) is reverted to and the rate of increase is slowed down. If regular rhythm has not been achieved after a dose of 30 grains has been given for a few days, quinidine therapy is discontinued. When it takes more than 30 grains to attain normal rhythm, it is usually most difficult to maintain that rhythm. After normal cardiac rhythm is restored, the dose is reduced 5 grains daily until a maintenance dose of 15, 10, 5 or even 3 grains is reached. Patients can be well maintained on such a dosage together with a maintenance dose of digitalis. Digitalis and quinidine are not synergistic in their action.[3]

Since 1929 I have treated several hundred patients according to this method.[4] Rarely have they manifested toxic symptoms such as marked pallor, Cheyne-Stokes respiration, convulsions or marked drop in blood pressure. When using quinidine for cardiac arrhythmias, one cannot disregard its pharmacologic properties, the need for timely application, or the variation in individual patient needs.

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INTESTINAL OBSTRUCTION

(Continued from Page 483)

proved surgical methods, supportive treatment and antibiotics have no doubt also been important. It is noteworthy that there were no gangrenous obstructions in any of these patients, although two patients had associated perforations.

Summary and Conclusions

1. Forty-five cases of mechanical intestinal obstruction due to inflammatory strictures were seen in this clinic from January 1, 1942, to September 1, 1953.

2. Regional enteritis and diverticulitis each accounted for almost one-third of the cases.

3. Primary resection following control of distention is the treatment of choice for obstructed strictures of the bowel, except in patients with marked dilatation within the greater length of the colon; emergency transverse colostomy with complete deviation of the fecal stream is indicated in the latter group.

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Editorials

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THE DOCTOR AND THE PRESS

There is no substitute for good public relations. This is true not only of a huge corporation, utility company, government or organization, but of every profession and agency which serves or owes some responsibility to the public.

None performs greater service to humanity than the medical profession and few have greater responsibility. Thus it would seem that a major effort of physicians and doctors should be toward developing a sound, workable plan of public relations.

It encompasses many fields at various levels.

Good public relations is a friendly liaison between doctor and patient.

Good public relations is making every use of radio, television, newspapers, magazines and other media for the dissemination of medical information that the public needs and wants to know.

We are living in a new era in the world, an era when medicine is making major inroads in diseases of all kinds, an era of miracle drugs and an era of marvels on the operating table.

The medical profession has a great story to tell, and it should be told. The public wants to know.

Further, it is my belief that the public has the *right* to know.

Surveys show that people worry and think more about their health than they do about George Gobel, baseball or war. They are being fed all kinds of information, accurate and inaccurate, about diets, proteins, cancer, heart trouble and housemaid's knee.

It is screamed at them from glaring advertisements over the radio and from TV screens.

Unfortunately, too much of it is misinformation, pure quackery or distortion, for the simple reason that whoever wrote the material didn't understand it or couldn't get a reputable medical man to "talk."

There is one great danger in peddling such misinformation. It instills fear, not common sense reasoning.

There was the case in Minneapolis not long ago where a woman, so frightened by misinformation, thought she had cancer. Without submitting to a competent medical examination, she went down into the basement and hanged herself.

A postmortem showed that *she did not* have cancer.

The medical man, I know, will agree that this is a horrible state of affairs. But hand-wringing and blistering assaults on quacks are not going to give the people the information they need.

To accomplish this will mean overcoming some historical and rather foolish barriers which have been built up in a misty past and should be relegated to the horse-and-buggy days where they belong.

One is a fear of the press on the part of the medical profession.

"The newspapers get everything wrong!"

"The stories are distorted and sensationalized!"

These are oft-repeated phrases used against public information media.

And this attitude, in turn, has built up a suspicion of all medical men among members of the Fourth Estate.

It is ridiculous.

Upon occasion, when medical men and newsmen have gotten together to air their problems, it is amazing how these barriers have vanished. Such, I am happy to say, has been the case in Ramsey County and in out-state Minnesota during recent years. Doctors and newsmen have been sitting together and talking things over. And a new feeling of mutual respect has been inculcated.

What have been the results?

Television programs, conducted by specialists in the field, on prevention and cure of tuberculosis, heart disease and cancer.

News stories, some attracting nationwide interest, of new developments in heart surgery, for example.

Stories that have been accurate, interesting, but not sensational, about new medical practices.

And all because enough doctors and newsmen were interested in one problem: How best can we tell the story of medicine to the public?

One fear of the medical profession is distortion of fact. This problem arises, mainly, when a reporter either doesn't understand the subject or oversimplifies in presenting it.

It can be solved. A man who knows his subject well enough can present it simply and with interest. And a good reporter, dealing with a highly technical subject, shouldn't be afraid to permit his source to check the accuracy of statements and facts. But this does not give the doctor or medical scientist the right to censor grammar, punctuation or style.

News prepared for public consumption *must be clear* and *easy to read* or it's not going to be read. I, personally, feel that a lot of professions have cloaked themselves in a welter of technical "gobbledy-gook" for which there are much better four-letter words.

One more thing. The day of the alcoholic, devil-may-care newspaperman is gone. The reporter who comes to get a medical story probably is college-trained, stable and eagerly enthusiastic about doing his best. He can be trusted, and he has a code of ethics as strict as those in the medical profession.

There is another level of public relations which should be mentioned. That's the practical, day-to-day contact between the press and the doctor in covering accidents, illnesses and injuries.

Newspapermen do not want doctors to violate their sacred oath by giving away confidences between doctor and patient.

But newsmen do expect that when a doctor is asked, he will not hang up the telephone and not brush away the questions in a brusque manner. The reporter expects that the doctor will confirm reports of illness or injury, and if they are true, will explain, in broad terms, the nature of the injury.

Newspapers are never too much concerned with illness, unless the sufferer happens to be someone in the public eye or unless the illness seems to be a rarity.

Medical men should *know* that when a newspaper gets wind of that kind of story, questions will follow. And it would be a good idea if the doctor discussed the matter with members of the family so that either could have authority to speak to the press.

In the matter of what is news and what isn't, there is only one answer. Newspapermen are trained to know what is news, just as doctors are trained to diagnose and treat illness.

To tell a newspaperman that such and such isn't news is like a newspaperman telling a doctor what he's supposed to treat.

In the realm of rare diseases, I have made this suggestion several times. In order to prevent "toe stubbing" by the press and to insure accurate information on all types of illnesses, it is suggested that each county medical society draft a list of specialists in various fields. This list should be turned over to the newspaper. And when a case of suspected rare illness comes up, the newspaper can easily call the doctor in that specialty and ask him whether it is rare, unusual or not.

This problem has come up frequently in the birth of "small babies." Or relatives will call and say that "Uncle Dudley" is down with a rare disease and shouldn't we write a story about it.

Doctors, I know, have an aversion to anything that smacks of personal advertising. Newspapers respect this desire and, upon request, usually will leave out the name of the doctor concerned, although personally, I don't see where this fear of "names" has too much sound basis.

But still respecting this fear of "personal" aggrandizement, there are many public relations projects a medical society can undertake as a group.

Some of these:

1. Telling people where they can get emergency medical service, day or night.
2. Promoting visiting scientists who speak to the group.
3. Publicizing society projects, such as tumor boards, cancer and heart disease clinics. Also all the free, welfare work done by physicians in any community.

There is, as I said at the beginning, no substitute for good public relations. Tell the people what you are doing, and they will be happier and more understanding of your problems and hopes.

GARETH D. HIEBERT
Saint Paul Dispatch

PRIVILEGED COMMUNICATIONS

Physicians and surgeons are frequently called upon, in their professional capacity, to give testimony in court, and occasionally encounter the question of whether or not testimony sought to be elicited is, or is based upon, a "privileged communication" between doctor and patient. While, as a general proposition, he may safely rely upon the advice of the trial Judge, which he may always seek when in doubt, an understanding of the rule and its ramifications should be helpful to all. The rule applies not only to physicians and surgeons, but to lawyers, clergymen, public officers (in a limited sense) and husband and wife. Consideration is given here only to the rule as it affects doctors of medicine.

The statute covering the matter (595.02 MSA) provides as follows:

> "Every person of sufficient understanding, including a party, may testify in any action or proceeding, civil or criminal, in court or before any person who has authority to receive evidence, except as follows: * * *
>
> (4) A licensed physician or surgeon shall not, without the consent of his patient, be allowed to disclose any information or any opinion based thereon which he acquired in attending the patient in a professional capacity, and which was necessary to enable him to act in that capacity; * * *"

Unless this privilege is waived, a physician cannot testify as to any information acquired by him in attending his patient. The privilege is not waived by the mere fact that the patient testifies concerning his condition while receiving treatment. The statute is for the protection of the patient, and he may waive it; and as a general rule those who represent him after his death may waive it for the protection of interests which they claim under him. A physician may testify as to the simple fact that he has attended a certain person professionally, and as to the number of his visits. Communications are not privileged if made in furtherance of a criminal purpose. Communications not made to enable the physician to act professionally are not privileged. A waiver in an application for insurance has been held not to apply to future communications. A party may consent that his attending physician may testify against him, but a statement made during cross-examination, without an opportunity to advise with his counsel and without a full understanding of his legal rights, that he has no objection to the physician testifying, should not be treated as waiver of his privilege which cannot thereafter be withdrawn. Dentists are probably not physicians within the meaning of the statute.

The testimony of a physician as to the instructions given his patient, and as to whether the patient obeyed such instructions, is within the privilege conferred by the statute. The statute applies, though the services of the physician are gratuitous. In the case of physicians making an examination to ascertain the physical ability of the individual to do a certain type of work, the information not being obtained for the purpose of treating or acting for him, is not privileged, nor is information obtained through an adverse party examination to qualify the doctor to testify, whether he prescribes for the party or not.

If a patient fails to object to a question put to his physician, which necessarily calls for testimony which is privileged under the statute, after a fair opportunity is given him to make an objection, he waives the privilege. The patient does not waive his privilege by bringing an action to recover for the injuries for which the physician treated him, unless the action is against the physician for malpractice (in which event the doctor, of course, is free to disclose in his defense all the phases of his treatment of the patient). Neither does he waive such privilege by presenting evidence in support of his claim, where such evidence is confined to matters outside his transactions with the physician. A waiver by the patient, procured by fraud or misrepresentation, should be disregarded.

Necessarily, the subject, upon which a great deal has been written, is here treated with utmost brevity and does not attempt to cover every eventuality nor to consider the individual Judge's interpretation of the Rule. To be completely safe, when in doubt—Ask the Judge!

Robert V. Rensch
Judge of District Court
Saint Paul, Minnesota

DISEASE: ORAL AND EXTRA-ORAL

In providing the best care for a patient, the physician and the dentist frequently find that their interests in and duties toward the patient overlap.

Intra-oral and per-oral disease may be of local origin, to be detected and treated by either the physician or dentist. Some forms of local disease, such as dental caries and periodontal disease, are traditionally diagnosed and treated by the dentist; whereas, other conditions, such as malignant tumors, may oftentimes be diagnosed by the dentist, but almost invariably are treated by the physician. Some disorders of this region are merely local manifestations of a systemic disease, and the alert dentist may render a great service to his patient if he detects the presence of a serious condition and refers the patient to the physician for early control of the ailment. Conversely, many symptoms in and about the mouth may be detected by or reported to the physician first, and prompt referral to the dentist may be in the best interests of the patient.

In our schools of medicine and dentistry, there is a remarkable overlapping of teaching materials, facilities, and faculties in the basic health sciences. And in hospitals, clinics, and research laboratories the teachings are welding the two professions together. There is room for an even higher level of co-ordination of efforts in the private practice of dentistry and medicine. The most cursory perusal of a few of the textbooks concerned with "oral medicine"† reveals that both the dentist and physician must give consideration to innumerable diseases manifest in or about the mouth, classified under such headings as the blood dyscrasias, infectious diseases, dermatoses, neoplasms, endocrine imbalances, nutritional deficiencies, arthritides, hypersensitivity reactions, developmental abnormalities and many other conditions, e.g., Paget's disease of bone and the xanthomatoses, which do not readily lend themselves to such classification.

Joint efforts of dentists and physicians have resulted in remarkable advances in the development of surgical techniques and ocular, facial, intra-oral and even cranial prosthetic appliances for victims of congenital anomalies, and accidental or surgical trauma. In future years, it is to be hoped that an even greater co-ordination of the practice of medicine and dentistry will result to the advantage of our patients as the members of each profession learn more of the activities, interests, and capabilities of each other.

David F. Mitchell, D.D.S.
University of Minnesota

TRAINING LABORATORY AIDES

In 1952 and 1953, an advisory group of the Council of the Minnesota State Medical Association made a special study of needs in laboratory and x-ray service throughout the state. As a direct result of this study, the University of Minnesota was asked to establish a one-year training program for laboratory aides as an attempt to partially relieve a severe shortage of trained laboratory personnel, especially in doctors' offices and hospitals and clinics in smaller communities. Through co-operative efforts of the Minnesota Society of Clinical Pathologists and the Minnesota Society of Medical Technologists, a curriculum was devised and a training program instituted in the General Extension Division and the College of Medical Sciences of the University of Minnesota in the fall of 1953.

Graduates of accredited high schools who are in the upper half of their graduating class are accepted for the program. In the fall and winter quarters, October through March, students register for formal classes in elementary basic sciences and laboratory technique at the University. These subjects include physiological chemistry, anatomy, bacteriology, basic laboratory technique, routine hematology, routine urinalysis, gastric analysis, electrocardiographic technique and techniques in blood banking. Students who successfully complete this portion of their training continue their training in the laboratories of participating hospitals under the supervision of pathologists and registered medical technologists for an additional six months. Comprehensive examinations are given at the end of the full year of training. The student who successfully completes the entire course receives a certificate from the General Extension Division of the University.

This represents an attempt to provide satisfactory training for laboratory workers at a subprofessional level and as such is an experiment. Its success will depend in a large measure on the continued interest, support, and guidance of official medical and hospital organizations.

Ellis S. Benson, M.D.
University of Minnesota

NATIONAL ASSOCIATION OF BOARDS OF PHARMACY

The National Association of Boards of Pharmacy is a new associate of the American Association for the Advancement of Science. In the April first issue of *Science*, the weekly AAAS journal, P. H. Costello, secretary of the NABP, writes a brief orientation about the NABP. From it, these extracts:

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†Burket, L. W.: Oral Medicine. 2nd ed., Philadelphia: J. B. Lippincott Co., 1952. Thoma, K. H.: Oral Pathology. 4th ed., St. Louis: C. V. Mosby Co., 1954. Comroe, B. I., L. H. Collins and M. P. Crane: Internal Medicine in Dental Practice. 3rd ed. Philadelphia: Lea and Febiger, 1949.

Organization of the National Association of Boards of Pharmacy was effected in 1904 at a meeting of representatives of the pharmacy licensing boards of twenty-six states. This first meeting was held in conjunction with the annual meetings of the American Pharmaceutical Association. The present active membership is composed of the Boards of Pharmacy or licensing bodies of forty-five states, the District of Columbia, Alaska, and Puerto Rico. The Boards of Pharmacy in California, Florida, and New York are associate members.

The objective of the Association declared in the constitution, is to provide for interstate reciprocity in pharmaceutic licensure, based upon a uniform minimum standard of pharmaceutic education and uniform legislation; and to improve the standards of education and licensure by cooperating with state, national and international agencies and associations having similar objectives.

The Association constitutes a forum of the examining boards for the discussion of examination techniques, procedures, and all other subjects that are designed to raise the standards of pharmaceutic education and predicates registration upon the highest practical legal qualification. . . . The Association maintains a file record of all state pharmacy laws, interpretations and legal opinions of record pertaining to requirements and qualifications for licensure, and also the regulations adopted by the State Boards of Pharmacy. It has endeavored to compile annually licensure and census data to indicate by states the number of licensed persons engaged in pharmacy, how engaged, number licensed, and number of pharmacies in operation, and, as a service to the membership, to disseminate this information along with other current information related to the education and licensure of pharmacists, examination procedures, trends in education and legislation pertaining to pharmacy licensure requirements. This material is found in the *Proceedings,* which has been published annually since the Association was founded, and in the NABP *Bulletin,* a bimonthly publication. . .

Recommendations made by the Association with respect to subjects, scope, and length of the examinations that are given to applicants for licensure in all states have been accepted by the membership and have fostered a degree of uniformity in licensing procedure. The establishment of uniformly high legal requirements or qualifications, that applicants for licensure in all states must possess, is due largely to the program of action that has been carried out by the NABP to secure such qualifications and make reciprocal licensure more feasible. . . .

The Association does not speak for, or commit any of, its members who must act upon the applications, but the service facilitates and expedites the prompt completion of applications in complete detail, which is a matter of importance when the urgent needs of an employer, an employe, or a community for pharmaceutical service are concerned.

H.G.M.

THE RORSCHACH TEST

Since the major component of at least one-half of the conditions for which patients consult physicians involves personality factors, and since diagnosis and prognosis of psychosomatic disorders as well as their treatment are influenced to a large degree by these factors, physicians should be familiar with the use of methods of personality evaluation.

One of the most valuable methods of personality analysis is the Rorschach (ink blot) test. This test is administered by having the patient look at ten standardized cards on each of which is an ink blot. Each ink blot, in itself, has minimal conventionalized meanings, so the patient's responses to these blots actually represent functions of his own personality. With this test, a three-dimensional view of the patient's personality is obtained. This has been called a "psychogram" and has been regarded by some as resembling an x-ray of the total personality.

The Rorschach probes beneath observable behavior, and it distinguishes between superficial shock reactions and the more deep-seated character neuroses. In psychosomatic problems, it gives important diagnostic and therapeutic clues and helps to distinguish between those cases in which medical treatment should be primary and those in which psychotherapy takes the lead.

The field of application of the Rorschach technique is almost limitless. In addition to personality evaluations in connection with psychosomatic problems, the test is of value in distinguishing between post-traumatic neuroses and actual organic brain syndromes; in evaluating the personality assets and liabilities of individuals being considered for promotion to executive and administrative positions in industry; in deciding the level at which psychotherapy can be pitched in organic disorders with a heavy functional overlay; and in estimating the prognosis in a vast number of personality disorders.

Like any other test, the Rorschach is not infallible. Like a leukocyte count, urinalysis, blood chemistry, and x-rays, it must be evaluated in the light of clinical findings. When so used, it could be of great assistance to medical diagnosis.

G.R.K.

SOCIAL SECURITY DISABILITY "FREEZE" PROVISIONS

The explanatory note regarding the disability "freeze" provisions of recent amendments to the Social Security Act, published herewith, provoke two observations.

Firstly, the physician is again called upon to be judge and jury in matters that pertain to his patients' incomes. Government has done this repeatedly to physicians because no one else is in a better position to judge, in this instance, the degree of disability, than the patient's physician. We must all be on the lookout for the same pitfalls that we sometimes stumbled into when patients besieged us for certificates that would enable them to get more red points, more blue points, or more gallons of fuel oil. Physicians will feel man's urge to get something for nothing from one more source with these new provisions of the Act.

Secondly, we hope that this doesn't spell a new set of forms to be filled out to certify that someone is disabled, and how. Maybe the Social Security Administration and the American Medical Association can take the same viewpoint, at least so far as using a universal certificate-of-disability-form, for which "organized medicine" has been beating the drum for a long time.

DISABILITY "FREEZE" PROVISIONS

Physicians, hospitals, and clinics will have an important part in assisting their patients to get the benefit of a new provision of the social security law which protects a worker's social security record while he is disabled.

A person who is unable to work because of mental or physical disability, or who is blind, can have his social security earnings record "frozen" under the new law.

The effect of this action is similar to that of the waiver of premium in life insurance policies. It provides that the benefit payable to the person when he qualifies, or to his family in case of his death, will not be reduced because of the period in which he had no earnings.

Persons already receiving monthly old-age insurance benefits, if they were disabled for a considerable period of time before they reached sixty-five, may have their benefits increased under the new law beginning with payments for July, 1955.

The new law protects the Social Security records of people who have been in work covered by social security for five out of the ten years before they were disabled, and for one and one-half years out of the three years before they were disabled; who are unable to engage in substantial gainful activity because of a medically determinable impairment which is expected to be of long-continued or indefinite duration; and who apply to have their records frozen while they are disabled and after they have been disabled six months or more.

A determination that a person is disabled must be based upon medical evidence. The patient himself is expected to secure the initial medical evidence; therefore, he will frequently request a summary of the history, clinical findings, and treatment of his case from his physician or medical facility. In other cases, the request, along with a release signed by the patient, may come from the state agency responsible for the determination of disability, or from the Social Security Administration.

The Social Security Administration hopes that many applicants can be returned to productive work through vocational rehabilitation services. Work in connection with rehabilitation services or in a "sheltered workshop" will not prevent a finding of current disability.

H.G.M.

MOTOR VEHICLE ACCIDENTS—A KILLER OF MINNESOTA CHILDREN

Accidents are the No. 1 killer of Minnesota children. During the past several months, through a series of articles in MINNESOTA MEDICINE, your attention has been focused upon a number of "killers." These have included poisons, suffocation, strangulation, aspiration of foreign bodies, et cetera. In this month's copy, you are urged to peruse Mr. Larimer's data on motor vehicle accidents among Minnesota's children.

In the past five years, 645 children died from motor vehicle accidents in Minnesota. This is a terrific toll of human life. Too many occurred among preschool and grammar school children: as pedestrians, on bicycles, on school buses and as occupants of moving vehicles. Not enough is done by physicians by way of safety education.

Are you using the "new safety vaccine?"

TAGUE C. CHISHOLM, M.D.

President's Letter

THE AMA MEETING

The 104th annual meeting of the American Medical Association is past history. The Minnesota State Medical Association may well be proud of its delegates in their participation in the deliberations of the House of Delegates and reference committees.

Both Dr. Martin's and Dr. Hess' addresses were scholarly and competent in their presentation and recommendations as to the problems facing the medical profession today. I feel sure that the leadership of Dr. Hess will be vigorous and forthright, and under such leadership, the affairs of American medicine are in good hands.

Dr. Cline's report regarding osteopathy was detailed and showed much effort and study. Its value does not necessarily lie in its recommendations, but in the deficiencies that were brought to light in the osteopathic educational system. But it was obvious to most that the element of cultism still exists. The action of the House of Delegates in not accepting the report and in continuing the study of osteopathic problems is in the best interests of the public and the medical profession.

The reference committee on insurance and medical service had a stormy and informative session. The informal discussion again pointed out to me the necessity for medicine to take a firm hand in guiding the future course of medicine in solving the economic problems of the profession.

Again the importance of adhering to basic principles in the furnishing of medical care was emphasized, if the American public is not to sacrifice good medical care for political expediency and minority clamor.

The problem of medical ethics as it pertains to dispensing of drugs was also heatedly discussed, and the action taken was in the best interests of all.

Public reaction to the theme of the presidential installation ceremonies, "Medicine's Proclamation of Faith," was very favorable. At these ceremonies a beloved Minnesota physician received the Distinguished Service medal of the American Medical Association for the year 1955, and every Minnesota physician may be proud of this distinction. We congratulate Dr. Donald C. Balfour of Rochester on this well-deserved honor.

The scientific program and the scientific and commercial exhibits all combined to make the annual session one of unusually high quality. It was a pleasurable experience to be able to participate in the deliberations. I feel that definite advances were made in American medicine.

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

AMA SESSION: DEMOCRACY IN ACTION

The 104th annual meeting of the American Medical Association in June was truly a fine example of the democratic process being enacted.

It is well known and accepted that when a large policy-making body meets for debate on controversial issues, it can easily become bound up in parliamentary snarls of many kinds which, if allowed to remain confused, can be extremely difficult to resolve.

However, the AMA House of Delegates was a striking example of avoiding such snarls, while allowing full and complete opportunity for discussion and deliberation on all issues.

One such case was the report of the famous Cline committee to study the relations between medicine and osteopathy. (For a full report of the results of this discussion, see the summary of the AMA session elsewhere in this issue.) The report itself, twenty-five pages long, was not read on the floor of the House, but it was referred to a reference committee for study and recommendation. The report made several recommendations which, if adopted, would change some basic principles of the medical profession. Full debate and discussion on these issues was held for several hours in the reference committee hearing, and freedom of speech was in no way limited, for all those who wished so were accorded full opportunity to be heard.

But, the democratic process did not end there. The reference committee itself brought both a majority and a minority report to the floor of the House, and open debate was again held on all the major issues involved. The importance of the minority view in America was seen in its full impact when the lone minority member of the reference committee clearly stated his reasons for objecting to the majority view. And, following an extensive and intensive discussion, the House voted to substitute the minority report for that of the majority report.

It is obvious that what could easily have been a confusing and difficult situation, became a clear-cut issue, and exemplified every American's right to be heard and to make known what he thinks is right or wrong, regardless if he is or is not in accord with the majority view.

Officials State Sound Policies

Dr. Walter B. Martin, then AMA president, addressed the House of Delegates at its opening session, and brought to the delegates a message containing many sound statements regarding medicine's responsibilities and problems. He noted that medical education is one of medicine's chief concerns these days. He said:

> "We have a great heritage and one that must be preserved and passed on to those who come after us. Medical education has advanced in this country because it has been free and we must keep it free. For this reason the American Medical Association has opposed federal aid to medical education in any guise that would make possible federal interference or control."

He noted that numerous proposals have been made and plausible arguments have been advanced by eager advocates of federal participation in the financing of medical schools. But, he warned:

> "These arguments, however, have not dispelled the justifiable fear that some degree of federal influence and control would follow federal financial aid in meeting the operational budgets of medical schools. These objections, however, do not pertain to one-time federal grants to medical schools for new construction or for reconstruction of old buildings."

Physicians Should Help

Dr. Martin cited the need for practicing physicians to do all they can to help finance better medical education, and thus help improve the medical care of the people. He stated:

> "Since the financial needs of medical education are real and urgent, other means are being sought, and must be found, for satisfying these needs. One of the greatest contributions that can be made to medicine and to the public good by American physicians is the swift and adequate financial support of medical education. In certain states the response of our profession has been very fine, but these areas have been all too few. If we are to prevent the entrance of political influence into medical education, we must act promptly and effectively and on a broad front."

Hess Lauds Leaders

Dr. Elmer Hess also spoke to the House of Delegates as president-elect of the Association. He noted the fine quality of leadership in state and county medical societies all over the nation, as he had witnessed them during his travels. "I found these men to be vigorous, alert individuals, full of good ideas and boundless energy," he said. "So long as we maintain such quality of leadership in our county and state societies, the American people will continue to get the best medical care this world has ever known."

Mental Health Needs Cited

Dr. Hess told the delegates that one of the greatest medical problems in the United States today is that of mental and emotional illness. "Leading psychiatrists believe that at least 50 per cent of all patients who come to physicians' offices have a mental or emotional disturbance along with their physical disability," he noted. "And because at least half of our patients seem to fall in this category I think that mental health becomes automatically a concern of all practicing physicians—not just the psychiatrists."

Physicians Should Lead

Dr. Hess advised American doctors to take the lead in helping to alleviate this problem, saying:

> "In my book, the nation's physicians should be the leaders in any campaign to overcome the ravages of mental illness. Great numbers of mental patients could be returned to useful, productive lives if they receive the proper medical as well as psychiatric treatment. Instead, today we have a system of understaffed, overcrowded institutions in which the mentally ill have little hope for cure—they are virtually tossed on a human junkpile."

Dr. Hess outlined specific ways in which physicians could work to improve the situation: take an increasingly active part in the development of more psychiatric units in general hospitals; give one day a week to work in state or county mental hospitals near their homes; aiding in development of residency training programs for nonpsychiatric residents in state mental hospitals; encouraging state and county societies to establish psychiatric consultation services for their general physician members; and aiding in reawakening the interest of communities in the public and private mental hospitals.

Another serious problem facing us, Dr. Hess said, is the medical care of the low income, no income group. He went on: "Although more than 100 million of our citizens have voluntarily availed themselves of insurance protection against the cost of illness, there are many of our citizens who cannot afford to purchase even the most modest type of coverage. These include the unemployables, the older people who live on very small pensions and those who cannot improve their economic status no matter how they try." He continued:

> "Traditionally, the physicians have provided free medical service to those who could not afford to pay. However, someone must also look after their hospital care, and I believe that is the responsibility of taxpayers in the local communities. As taxpayers also, we physicians have a double responsibility. Through our state and county medical societies we must provide the leadership to develop adequate state and county public hospital care programs for the low income, no income group of our citizens. We should intensify our efforts in this direction."

Safety Program Urged

Dr. Hess also emphasized the problem of highway accidents as a major concern of the medical profession. He called the automobile a lethal weapon—one that should only be placed in the hands of those who are mentally and physically qualified to drive. "There are many license-carrying motorists today who are temperamentally unfit to drive; they should be weeded out," Dr. Hess declared. "It is ironic that in an age of great medical achievements, aimed at prolonging life, we can be so apparently indifferent to the havoc which results on our streets and highways because of sloppy and arrogant driving."

Threats to Freedom

Dr. Hess noted the many fields in which medicine has become more concerned over threats to freedom in medicine. He noted that the profession has fought hard against such obvious threats as national compulsory health insurance and the numerous fringe measures, and noted, too, that the profession has begun to recognize the socialistic dangers presented by the constant expansion of existing federal medical care plans. The House applauded his next statement:

> "However, it is not enough for physicians to be concerned over threats to freedom in medicine. Nor is it enough for businessmen, industrialists, workers, or teachers to be aroused only when their own spheres of activity are menaced by proposals for increased government

control. All of us, in all social and economic levels of American life, should join together in an outspoken protest against a way of living that is entirely foreign to our fundamental principles of government."

ECONOMIST FORECASTS U. S. IN 1960

J. Frederic Dewhurst, head of the research staff of Twentieth Century Fund, has recently projected a look into what the U. S. economy will be five years hence. According to a recent issue of *Time* magazine, his key prediction, based on the assumption that peace and prosperity will continue, is that 1960's gross national product will be $413.5 billion, up 29 per cent from 1950, 16 per cent from 1954.

Noting especially an increase in almost every phase of American life, Mr. Dewhurst predicted a 16 per cent population growth, 69 million jobs which is 7.5 million more than in 1950, cultivation of 35 million acres of new crop land, an increase in gross private savings and a before-tax per household income of $6,180 average.

Needs Outlined

However, in all these increases and in the apparent prosperity trends, the nation's needs were outlined as difficult to pin down. Dewhurst's researchers tried to calculate how much more money would have to be spent to reach a "standard of living at a health and decency level." As difficult as this is to figure, the economists figured that 1950's gap between needs and supply—$13.1 billion, or 6 per cent of expenditures—would be slashed to $11.4 billion in 1960, only 4 per cent of expenditures.

And, a surprising thing of importance to the medical profession, is this prediction: "The figures showed changes in types of shortage. Housing accounted for 30 per cent of 1950's unavailable needs, only 12 per cent of 1960's. But more than half of the 1960 gap will be a shortage of medical care v. 36 per cent in 1950."

Accounting for this change will be extremely difficult, at best, and meeting those expected needs will be a great challenge, not only to the medical profession, but to all Americans as well.

The causes of health and therefore the factors important to its evaluation and management are often to be found in the individual's personal and community environment.—Ward Darley, M.D., *J.A.M.A.*, April 30, 1955.

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

Minneapolis Man Sentenced on Abortion Charge

Re: State of Minnesota vs. William V. Smith

On May 16, 1955, William V. Smith, 40, 227 Ridgewood Avenue, Minneapolis, who stated he is employed as a promotional manager for a Minneapolis hearing aid company, was sentenced by the Hon. Theodore B. Knudson, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the State Prison at Stillwater for the crime of abortion, the sentence being stayed, however, and the defendant placed on probation for a period of three years. On April 4, 1955, Smith entered a plea of guilty to an information charging him with having aborted a twenty-two year-old unmarried Minneapolis woman on February 6, 1955. The patient became ill the day after the abortion was performed but did not seek medical care until a month later at which time she entered Minneapolis General Hospital on the advice of her physician. According to signed statements given by both the defendant and the woman who submitted to the abortion, the defendant was instructed by her in regard to how the abortion should be performed and the catheter that was used by Smith in doing the abortion was furnished by her. The defendant, who admitted he had no scientific training except for having taken biology in high school and zoology in college, claimed that he had no previous experience with abortions.

When the defendant was asked by the Court what prompted him to commit this abortion, inasmuch as it appeared that he did not charge any money for his services, he replied that he "tried to be a friend in need." Judge Knudson then asked him if he didn't feel that he had to be discriminating in this as well as in anything else and Smith replied that he now realizes that he made a "horrible mistake." Judge Knudson then warned the defendant that if he violated any of the terms of his probation he would have to serve his sentence in prison.

First Prosecution Under Minnesota Physical Therapy Law Results in Conviction of Rochester Woman

Re: State of Minnesota vs. Edith Lundeen

On June 14, 1955, Edith Lundeen, R.N., entered a plea of guilty in the District Court of Olmsted County at Rochester, Minnesota, to a charge of representing herself as a physical therapist without having a valid existing certificate of registration, and she was sentenced to pay a fine of $250.00. However, $150.00 of the fine was suspended by the Court and the defendant actually paid a fine of $100.00. Miss Lundeen had previously pleaded not guilty to the charge when she was arraigned on June 6, 1955, but as stated above, she changed her plea to guilty, thereby making a trial unnecessary. According to Olmsted County officials, Miss Lundeen is now attempting to sell her equipment and has painted over the letters "Ph.T.," which she formerly displayed in front of her home near Rochester. The defendant has also at various times been listed as a physical therapist in the classified section of the Rochester telephone directory.

The records of the Minnesota State Board of Medical Examiners show that in 1934 Miss Lundeen maintained an office in the Lawler Building in Rochester and that according to an advertisement, she was a graduate in physiotherapy, specializing in the treatment of arthritis, myositis and neuritis. At that time, even

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AMERICAN MEDICAL ASSOCIATION

Proceedings of the House of Delegates

104th Annual Meeting

Osteopathy, medical ethics, medical practices, intern training, hospital accreditation and polio vaccine were among the major topics of discussion by the House of Delegates at the American Medical Association's 104th Annual Meeting held June 6-10, 1955, in Atlantic City.

Elected unanimously as president-elect for the coming year was Dr. Dwight H. Murray, general practitioner of Napa, California, who has been a member of the AMA Board of Trustees for ten years and its chairman for the past four years. Dr. Murray will become president of the American Medical Association at the June, 1956, meeting in Chicago, succeeding Dr. Elmer Hess of Erie, Pennsylvania. Dr. Hess took office at the Tuesday evening inaugural program in Atlantic City's Convention Hall.

The House of Delegates voted the 1955 Distinguished Service Award of the American Medical Association to Dr. Donald C. Balfour, surgeon, author and researcher of Rochester, Minnesota, for his outstanding contributions to medicine and humanity. Dr. Balfour has been with the Mayo Clinic since 1907 and he also has been associate director and then director of the Mayo Foundation for Medical Education and Research. His son, Dr. William Balfour, accepted the award for his father at the Tuesday inaugural program.

The Osteopathic Issue

The Reference Committee on Medical Education and Hospitals submitted two reports after considering the recommendations of the Committee for the Study of Relations Between Osteopathy and Medicine. The minority report, which was adopted by the House of Delegates, said:

"One member of the Reference Committee was completely satisfied that an appreciable portion of current education in colleges of osteopathy definitely does constitute the teaching of 'cultist' healing, and is an index that the 'osteopathic concept' still persists in current osteopathic practice. Since he cannot with good conscience approve the recommendation that doctors of medicine teach in osteopathic colleges where 'cultism' is part of the curriculum, he respectfully makes the following recommendations to the House of Delegates:

"1. That the report of the Committee for the Study of Relations Between Osteopathy and Medicine be received and filed; and that the Committee be thanked for its diligent work, and be discontinued.

"2. That if and when the House of Delegates of the American Osteopathic Association, their official policy-making body, may voluntarily abandon the commonly so-called 'osteopathic concept,' with proper deletion of said 'osteopathic concept' from catalogs of their colleges; and may approach the Trustees of the American Medical Association with a request for further discussion of the relations of Osteopathy and Medicine, then the said Trustees shall appoint another special committee for such discussion."

The majority report of the reference committee, which was rejected by the House, made the following recommendations:

"Your Reference Committee after a study of the report of the Committee for the Study of Relations Between Osteopathy and Medicine and the study of other evidence submitted is not completely satisfied that the current education in colleges of osteopathy is free of the teaching of 'cultist' healing.

"In view of the desire to elevate the standards of teaching in colleges of osteopathy, your Reference Committee recommends approval of the recommendation of the Committee that doctors of medicine may accept invitations to assist in osteopathic undergraduate and postgraduate medical educational programs in those states in which such participation is not contrary to the announced policy of the respective county and state medical associations. Such teaching services would be ethical.

"Your Reference Committee approves the recommendation of the Committee that the House of Delegates request state medical associations to assume the responsibility of determining the relationship of doctors of medicine to doctors of osteopathy within their respective states or request their component county societies to do so.

"Your Reference Committee recommends that a committee be appointed at the discretion of the Board of Trustees to confer with representatives of the American Osteopathic Association concerning common or interprofessional problems on the national level."

Change in Medical Ethics

The Reference Committee on Miscellaneous Business dealt with ten resolutions concerning the dispensing of drugs and appliances by physicians. The following committee report was adopted by the House:

"A great many individuals appeared before your committee in the interest of several resolutions submitted to it requesting amendment to or deletion of Chapter I, Section 8 of the Principles of Medical Ethics, and the bulk of your committee's time was spent on this very important and complex matter.

"With reference to this problem, the following resolutions were considered: Nos. 7, 12, 16, 18, 22, 35, 39, 58, 62, and 73.

Your committee recommends that no one of these resolutions be adopted as submitted but does recommend deletion of Section 8, Chapter I of the Principles of Medical Ethics which now reads:

'OWNERSHIP OF DRUG STORES AND DISPENSING OF DRUGS AND APPLIANCES BY PHYSICIANS

'Sec. 8.—It is unethical for a physician to participate in the ownership of a drug store in his medical practice area unless adequate drug store facilities are otherwise unavailable. This inadequacy must be confirmed by his component medical society. The same principle applies to physicians who dispense drugs or appliances. In both instances, the practice is unethical if secrecy and coercion are employed or if financial interest is placed above the quality of medical care. On the other hand, sometimes it may be advisable and even necessary for physicians to provide certain appliances or remedies without profit which patients can not procure from other sources.'

"Your committee recommends that the following be substituted in lieu thereof:

DISPENSING OF DRUGS AND APPLIANCES
BY PHYSICIANS

'Sec. 8.—It is not unethical for a physician to prescribe or supply drugs, remedies, or appliances as long as there is no exploitation of the patient.' "

In reporting to the House the chairman of the Reference Committee explained that in the opinion of the Committee the Code of Ethics should be stated in broad principles rather than attempt to interpret principles in detail. In recommending the change in Section 8 the Committee emphasized that this section should be interpreted in line with Chapter I, Section 6, which reads: "The ethical physician, engaged in the practice of medicine, limits the sources of his income received from professional activities to service rendered the patient. . . ."

Medical Practices Committee Report

The Reference Committee on Insurance and Medical Service, which considered two Board of Trustees reports on the Report of the Committee on Medical Practices, recommended endorsement of the Board's principal conclusions and recommendations. The House of Delegates, however, adopted a substitute motion postponing action until next December. The motion also called for distribution of the entire report of the Committee on Medical Practices to all delegates, so that they can study it carefully before the 1955 Clinical Meeting in Boston.

Internship Approval Programs

The House adopted the following statement presented by the Reference Committee on Medical Education and Hospitals.

"Your Committee has reviewed the report of the Council on Medical Education and Hospitals which includes a summary of the reports previously made to the House of Delegates by the Ad Hoc Committee on Internships and are in agreement with the Council that these conclusions and recommendations are eminently sound and that they should be incorporated into the principles and policies employed by the Council in the conduct of its internship approval programs including subsequent revisions of the Essentials of an Approved Internship.

"Your Committee wishes specifically to reaffirm the following recommendations of the Ad Hoc Committee on Internships:

"1. That a continuing study be made as to what should be the content of an internship; what constitutes sound clinical experience during the internship year.

"2. That the 'one-fourth rule' be adopted: Any internship program that in two successive years does not obtain one-fourth of its stated complement be disapproved for intern training. It was pointed out to your Committee in the hearings that statistical data compiled for a period of two years indicated that enforcement of this rule would have displaced only a few interns."

Hospital Accreditation

The same reference committee considered six resolutions on hospital accreditation and presented the following statement which was adopted by the House:

"Your reference committee has reviewed all these resolutions which in principle are similar and apparently reflect a widespread dissatisfaction with the present functioning of the Joint Commission on the Accreditation of Hospitals, possibly from bilateral misunderstandings. Therefore, your reference committee recommends that the Speaker of the House of Delegates be requested to appoint a special committee to review the functions of the Joint Commission on the Accreditation of Hospitals to consist of seven members, none of whom shall be members of the Council on Medical Education and Hospitals or the Joint Commission on the Accreditation of Hospitals. This special committee should be instructed to make an independent study or survey and report its findings and recommendations to the House of Delegates at the next annual meeting. All physicians and hospitals are urged to pass on to this special committee any observations or suggestions concerning the functioning of the Joint Commission on the Accreditation of Hospitals."

Polio Vaccine

The House passed three resolutions suggested by the Reference Committee on Hygiene, Public Health and Industrial Health in connection with discussion of the Salk polio vaccine and the introduction of new methods in the treatment or prevention of disease.

The first resolution reaffirmed "confidence in the established methods of announcing new and possibly beneficial methods in the treatment and prevention of disease" and also reaffirmed "the need for the presentation of reports on medical research before established scientific groups, allowing free discussion and criticism, and the publication of such reports, including methods employed and data acquired on which the results and conclusions are based, in recognized scientific publications."

The second resolution included the following policy statements:

"Resolved, That the American Medical Association go on record as disapproving the purchase and distribution of the Salk polio vaccine by any agency of the federal government except for those unable to procure it for themselves and that such necessary federal funds therefore be allocated to the various proper state agencies for such purpose; and be it further

"Resolved, That the American Medical Association urge the Congress of the United States to allow the Salk polio vaccine to be produced, distributed and administered in accordance with past procedures on any new drug or vaccine."

The third resolution commended Dr. Salk as follows:

"Whereas, The physicians of this country recognize the great scientific achievement in isolating and perfecting a vaccine for the prevention of poliomyelitis by Dr. Jonas Salk; and

"Whereas, This vaccine is now being used to prevent poliomyelitis among many of our children; therefore be it

"RESOLVED, That the House of Delegates express its profound gratitude to Dr. Salk and its admiration for his monumental contribution to medical science."

Miscellaneous Actions

Among a large number of actions on a wide variety of subjects, the House of Delegates also:

Commended the "Medic" television program;

Reaffirmed its previous recommendation that the United States withdraw from the International Labor Organization;

Approved the Headquarters Survey Report, which included the statement that "the only public relations program of any permanent value is the private and public relations of the individual doctor";

Expressed regret that the Hoover Commission saw fit to alter or eliminate some of the recommendations of its Medical Task Force;

Reaffirmed its opposition to extension of the Doctor Draft Law;

Recommended the creation of an AMA Committee on Geriatrics;

Warned against the danger embodied in state legislative proposals designed to restrict the entire field of visual care to the profession of optometry.

Opening Session

Principal addresses at the Monday opening session of the House of Delegates were given by Dr. Walter B. Martin of Norfolk, Virginia, retiring AMA president, and Dr. Elmer Hess of Erie, Pennsylvania, then president-elect. Dr. Martin declared that the basic philosophy of medicine has not changed and "our obligation is to bring the best that medicine can offer to the individual patient." Dr. Hess said that the nation's physicians must become leaders in a campaign to "overcome the ravages of mental illness" as well as in an "intensive campaign to eliminate the needless bloodshed" of traffic accidents.

Inaugural Program

"Medicine's Proclamation of Faith" was the theme of the Tuesday evening inaugural program, which was broadcast nationwide by the ABC Radio Network. Dr. Hess, in his inaugural address, said that "unless we are willing to give of ourselves and our faith, our science will avail us little." Dr. Norman Vincent Peale, eminent clergyman, who was guest speaker on the inaugural program, pointed out that "the drawing together of medicine and religion is a step in helping man toward proper use of his God-given potentials and qualifications."

Election of Officers

The following officers were elected at the closing session, in addition to Dr. Murray, the new president-elect: Dr. Millard D. Hill, Raleigh, North Carolina, vice president; Dr. George F. Lull, Chicago, secretary; Dr. J. J. Moore, Chicago, treasurer; Dr. E. Vincent Askey, Los Angeles, speaker of the House of Delegates, and Dr. Louis M. Orr, Orlando, Florida, vice speaker.

Dr. Gunnar Gundersen, La Crosse, Wisconsin, was named chairman of the Board of Trustees to succeed Dr. Murray. Dr. James R. Reuling, Bayside, New York, was elected to fill Dr. Murray's term on the Board. Re-elected as trustees were Dr. L. W. Larson, Bismarck, North Dakota, and Dr. T. P. Murdock, Meriden, Connecticut.

Dr. Louis A. Buie, Rochester, Minnesota, was named by Dr. Hess to succeed himself on the Judicial Council. Elected to the Council on Medical Education and Hospitals were Dr. Harlan English, Danville, Illinois, and Dr. James M. Faulkner, Boston, the latter succeeding himself. Re-elected to the Council on Medical Service was Dr. H. B. Mulholland, Charlottesville, Virginia. Elected to the same Council were Dr. A. C. Scott, Temple, Texas, and Dr. R. B. Chrisman, Jr., replacing Dr. Orr.

Dr. B. E. Pickett, Sr., Carrizo Springs, Texas, was re-elected to the Council on Constitution and By-laws, and Dr. Warren Furey was named to the same Council to replace Dr. James Stevenson, Tulsa, Oklahoma.

J. ARNOLD BARGEN, M.D.. Rochester
O. J. CAMPBELL M.D., Minneapolis
GEORGE EARL, M.D., Saint Paul
F. J. ELIAS, M.D., Duluth
Delegates to the American Medical Association

TUBERCULOSIS BATTLE STILL CONTINUES

We are doing a better job in reducing the tuberculosis death rate than in reducing the number of cases. At present the annual number of reported cases of tuberculosis is declining by only 3 per cent in contrast with a 20 per cent drop in the death rate each year since 1951. Last year is the first time in our history when tuberculosis did not rank among the first ten causes of death. In spite of this good showing we have not won the battle against tuberculosis. We will not have won it until we achieve comparable success in reducing the large reservoir of tuberculosis infection still prevailing in most parts of the country. We can do this—but only through a continued and concerted effort to find and treat larger numbers of people annually in the early stages of their disease.—LEONARD A. SCHEELE, M.D., *Bull. Nat. Tuberc. A.*, May, 1955.

Minnesota Academy of Medicine

Meeting of October 13, 1954

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, October 13, 1954. Dinner was served at 7:15 and the meeting was called to order at 8:10 p.m. by the president, Dr. C. N. Hensel.

There were fifty-five members and five guests present.

After a short business session, the scientific program followed.

Dr. James R. Dawson, Jr., of the University of Minnesota Department of Pathology, read his inaugural thesis entitled "The Pathogenesis of Hyaline Membrane Disease in the Newborn." Lantern slides were shown.

THE PATHOGENESIS OF HYALINE MEMBRANE DISEASE OF THE NEWBORN

A Preliminary Report

JAMES R. DAWSON, JR., M.D.

Minneapolis, Minnesota

Almost thirty years ago, Johnson and Meyers[1,2] called attention to a special form of respiratory insufficiency in newborns. Johnson, a pathologist, was impressed by the eosinophilic membranes which were present in the lungs of these babies and he designated the lesion pneumonia with hyaline membranes. Since this original description, several important contributions have been made but there is little general agreement about the disease. For example, there is no universally accepted term for this disease but the "hyaline membranes" described by Johnson are still included in most of the terms in use today. I shall refer to this entity as hyaline membrane disease.

Hyaline membrane disease is now recognized as the commonest cause of death in premature infants and in full-term infants delivered by section or born of diabetic mothers. In Table I are shown the causes of death in autopsies on patients dying during the first week of life. These data are derived from a review of the autopsies performed by Fellows at the University during the four-year period 1949 through 1952. These 195 cases comprise 30 per cent of all autopsies on patients less than sixteen years of age. The importance of prematurity as a contributing factor in deaths during the first postnatal week is shown by the fact that 128 (65 per cent) of the 195 deaths were in prematures. Finally, there were fifty-eight cases of hyaline membrane disease in this group and fifty-three of them (90 per cent) were in prematures.

TABLE I. CAUSES OF DEATH IN INFANTS DURING FIRST POSTNATAL WEEK

Day of Death	Congenital Anomalies	Immaturity	Infections	Other Causes	Hyaline Membranes	All Causes
1	16/5	20/20	3/2	24/16	27/24	90/67
2	2/1	2/2	4/1	7/5	13/11	28/20
3	4/1	2/2	4/0	5/1	12/12	27/16
4	6/1	1/1	1/0	6/3	3/3	17/8
5	2/1	0	2/1	7/4	1/1	12/7
6	5/2	0	1/1	2/0	0	8/3
7	7/1	0	1/1	3/3	2/2	13/7
First Week	42/12	25/25	16/6	54/32	58/53	195/128

Numerator = Total Number.
Denominator = Number Premature or Immature.

The distribution of these fifty-eight cases of hyaline membrane disease, according to the weight of the baby, is illustrated in Table II. There is a peak incidence in the 1500 to 1999 gram group, with only four cases in infants weighing less than 1000 grams and only six in the groups above 2500 grams.

Another feature of interest in hyaline membrane disease is the relationship of length of survival to the development of the lesion. This is depicted in Table III. No infant exhibiting this lesion survived less than four hours and only two lived longer than 120 hours. Almost half of the deaths were in infants less than twenty-four hours old, and the highest incidence was in the six-hour to twelve-hour period. All of these data substantiate the findings in other studies on hyaline membrane disease.

From a clinical point of view, there are no features which enable one to make this diagnosis with certainty. In the classical cases it has been said that the child ordinarily has no respiratory difficulty immediately after delivery. Following this so-called latent period there is progressive impairment of respiration. This is accompanied by retraction and cyanosis. Many of these infants die but some probably recover. Roentgenologic studies of these infants show variable changes which are not considered pathognomonic of the disease.

TABLE II. RELATION OF WEIGHT OF INFANT TO DEVELOPMENT OF HYALINE MEMBRANES

Weight of Infant in Grams	Number of Infants with Hyaline Membranes
Less than 1000	4
1000-1499	17
1500-1999	21
2000-2499	10
2500-2999	1
3000-3499	4
Greater than 3500	1

When one considers the importance of hyaline membrane disease as a cause of death in newborn infants, it is surprising to see how little the pathologist has contributed to knowledge of this entity. This lack of interest on the part of the pathologist is emphasized by the fact that hyaline membrane disease is not even mentioned in most of the popular textbooks of Pathology. However, the pathologists who have concerned themselves with the problem, notably Johnson,[1] Farber,[3] Potter,[4] and Arey,[5] have published accurate anatomical descriptions of the lesions.

Autopsy in the classical cases reveals collapse of alveoli, dilatation of many alveolar ducts and respiratory bronchioles and a dense eosinophilic membrane lining the ducts and respiratory bronchioles. There is ordinarily no cellular reaction and little edema or fibrin but in cases surviving longer than a week these changes may be conspicuous.

The characteristic anatomical and functional changes of this disease are seen almost exclusively in man. Numerous attempts have been made to produce the lesion in experimental animals by instilling amniotic fluid[6,7] and other materials[8] intratracheally, by bilateral vagotomy[9] and by high ambient O_2 pressures.[10] Some membranes have been produced, but the entire picture of membranes and atelectasis has not been duplicated. This lack of a suitable experimental model has undoubtedly been the most important factor in prolonging our ignorance of the pathogenesis of hyaline membrane disease. Failing so completely in the experimental approach, we have been forced to rely entirely on the study of human material to give us a proper understanding of its development.

The hypotheses which have been advanced to explain the pathogenesis of this disease have generally assumed that the atelectasis is resorptive in Type 4; that is, that the lungs were normally expanded with air initially but that, due to the formation of the hyaline membranes, many air spaces were thereby obstructed, the contained air was absorbed and atelectasis resulted. The secondary nature of the atelectasis having been assumed most of the arguments which have arisen are concerned with the nature and origin of the hyaline membranes. Suffice it to say that there is insufficient evidence to establish the origin of these membranes, but irrespective of whether they are derived from the plasma of the fetus or from the amniotic fluid, there is reason to doubt their importance as a cause of the atelectasis.

TABLE III. RELATION OF DURATION OF LIFE TO INCIDENCE OF HYLALINE MEMBRANES

Duration of Life in Hours	Number of Infants with Hyaline Membranes	Cumulation Totals	
		First Day	First Week
Less than 4	0	0	
4-6	6	6	
7-12	11	17	
13-24	10	27	27
25-48	13		40
49-72	12		52
73-96	3		55
97-120	1		56
Over 120	2		58

It is commonly believed that the lungs of a fetus are atelectatic, and it is for this reason that the term "congenital atelectasis" has been used to describe the lungs of stillborn infants and of other infants who die soon after birth. The fetal lungs, of course, are airless and they are incompletely expanded, but certainly they are not atelectatic in the sense that this term is used to describe the postnatal airless lungs which are seen following complete bronchial obstruction. Microscopic examination of the fetal lungs shows a uniform and moderate distention of all portions of the respiratory tract, including the alveoli. The entire tract is filled with fluid which has free communication with the amniotic sac, but it should be mentioned that this communication with the amniotic sac is not essential for the proper development and distention of the alveoli by fluid for in infants with atresia of the larynx, the lungs are filled with fluid and are normally developed. Regardless of its origin, the fluid which fills the lungs has such a low protein content that the normal alveoli, alveolar ducts, et cetera, appear empty on microscopic examination. However, under abnormal circumstances, as in fetal anoxia, amniotic debris may be seen as far out as the alveoli.[4,5]

During delivery of the infant, some of this fluid is squeezed out but much remains and, with the first inspiration, fluid and air are drawn deeply into the lungs. With the establishment of respiration, there is a fall in pulmonary vascular resistance, and blood flow through the lungs is increased. This fall in pressure and increase in flow of blood lead to rapid resorption of fluid, and as the fluid is removed, air takes its place so that the lungs become fluffy and pale.

If, for any reason, there is exudation or transplantation of fluid into the normally expanded lungs, this fluid will displace air, but the alveoli will remain distended. When the situation which led to the edema is corrected, this fluid is ultimately resorbed and its place again taken by air so that the size of the alveoli, within limits, remains unchanged as long as there is free communication through the bronchi to the outside.

For collapse or atelectasis to occur in any area of the distended lungs, that area must be isolated by blocking its communication to the outside. This is readily accomplished by the occlusion of the bronchus. Distal to the occlusion, air and fluid may be completely absorbed so that the walls of alveoli, alveolar ducts and respiratory bronchioles become closely packed, separated at most by an imperceptible amount of fluid.

It is evident, therefore, that the atelectasis seen in infants with hyaline membranes is of a special type for in this condition only the alveoli are atelectatic in the early stages of the lesion. The alveolar ducts are either normally distended or overdistended.

The bright pink membranes lining these distended alveolar ducts have attracted so much attention that they have overshadowed the atelectasis.[11] Most students of the disease have assumed that the membranes came first and that the atelectasis was secondary and resorptive in type.[4] In order for this situation to develop, one must postulate that the hyaline membranes occlude the orifices of the alveoli. It is difficult to understand how this can occur, since the alveoli represent rather shallow outpouchings of the alveolar ducts; their orifices are almost as wide as the alveoli are deep. Furthermore, if the membranes are precipitated from fluid within the lung, one would expect them to be deposited on all surfaces of the alveoli and alveolar ducts, but in hyaline membrane disease, they are deposited almost exclusively along the walls of alveolar ducts.

These observations make it difficult to believe that the hyaline membranes cause obstruction of alveoli and that the atelectasis is of the resorptive type but there is a more compelling reason to doubt that the membranes cause the atelectasis. Hyaline membranes are seen in conjunction with atelectasis in the classical lesion, but if these membranes are really the cause of the atelectasis, one should never see dilated alveolar ducts and collapsed alveoli in the absence of hyaline membranes, and one should occasionally see membranes in alveolar ducts without alveolar atelectasis. Our study of all autopsies on infants dying within two weeks after birth fails to substantiate such a relationship between hyaline membranes and atelectasis. In our material, we find a considerable number of cases which exhibit typical alveolar atelectasis and dilated alveolar ducts in the absence of hyaline membranes, and we have not observed situations in which the membranes were deposited prior to the development of alveolar collapse. Since there are reasons for doubting the primary importance of hyaline membranes in the pathogenesis of this disease, one should explore the possibility that the failure of alveoli to expand properly is primarily responsible for the development of this disease.

Little has been written about the manner in which the lungs of premature infants or animals expand normally, and much of this is probably inaccurate because of the methods of preparing the lungs for study. In order to obtain significant information, one must fix the lungs almost instantaneously *in situ* at a predetermined stage in the respiratory cycle. This has not been done. Lacking such definite information on normal expansion one is perhaps justified in speculating about the process.

The participation of bronchi and bronchioles in the process of normal expansion is probably more or less unimportant, except as simple conduits, for they do not vary greatly in cross-section area. The greatest change in size occurs in the alveoli, alveolar ducts and respiratory bronchioles. In these structures, variations in capacity are dependent upon increase in size of the thoracic cavity during inspiration followed by return to original size during expiration. In the latter phase, the inherent elasticity of the lungs plays some part.

When the size of the thorax is increased during normal inspiration, the lung is stretched in all dimensions. This stretching is a manifestation of the inherent elasticity of the lung, which resides in the elastic tissues and smooth muscle principally. This elasticity offers resistance to the changes in size and shape of the various pulmonary structures. Resistance is so great in the bronchi that they change very little, but in the respiratory bronchioles, alveolar ducts and alveoli, the change is relatively large, and the extent to which each of these structures increases in size will depend upon the pressure to which it is subjected and upon its elasticity. In normal distention of the lung, all of the pulmonary structures must participate, but the extent to which each enlarges must be regulated within rather narrow limits if proper alveolar ventilation and gas exchange are to take place.

The actual increase in size of these structures is related to the increase in lung volume which is probably never more than fourfold. In terms of the change in one dimension of a sphere, this would represent an increase of slightly less than 60 per cent. However, it is unlikely that inspirations of this magnitude occur in the neonatal period. Even a twofold increase would be very great and with this it would be necessary for each of the units to increase its diameter by only 30 per cent. Changes in any one dimension of alveoli or alveolar ducts are therefore quite small.

Expansion of alveolar ducts and respiratory bronchioles with a co-existing collapse of the alveoli might be explained on the basis of differences in distensibility of these units. Each of these structures has elastic tissue in its walls, and it is possible that quantitatively there might be more elastic tissue in the alveolar walls but this is not easily demonstrated. It might also be possible to explain differences in distensibility based on qualitative changes in elasticity of elastic fibrils, but this is even more difficult to prove. Smooth muscle is the other component which resists stretching. This tissue is present in the walls of respiratory bronchioles and alveolar ducts, but it is absent in the walls of alveoli. It is possible that changes in tone of this constituent might readily occur. Clinical examples of increased tone or spasm are well known, and I should like to suggest that decrease in tone of the smooth muscle in alveolar ducts might contribute to abnormal expansion of the lungs which characterizes hyaline membrane disease.

If the tone of the smooth muscle were diminished, then the relative distensibility of the alveoli would be less than that of the alveolar ducts and respiratory bronchioles, and they should expand less readily leading to over-distention of the alveolar ducts and respiratory bronchioles. Any fluid present would presumably accumulate in the alveoli as the ducts filled with air. Resorption of fluid would allow more space for air, and because of their distensibility, the ducts would increase in size still more, so that ultimately the alveolar ducts would crowd out the alveoli. Undoubtedly the explanation is not nearly this simple. The rôle played by surface tension in the pathogenesis of this lesion, although unknown, is quite likely an important one.

Those factors, which might be responsible for changes in tone of smooth muscle in the lung, are not known, nor do we know that loss of normal smooth muscle tone in the newborn would lead to the development of lesions like those seen in hyaline membrane disease. However, this concept offers a new approach which can be pursued from the experimental point of view.

Once the situation develops, with collapsed alveoli and distended alveolar ducts, membranes from several sources might be deposited. The deposition of these membranes would augment respiratory difficulty by covering the small surface available for exchange of gases.[14] In addition, these membranes might be detached from their sites of formation to lodge in the terminal portions of the bronchi, producing collapse of the subtended alveoli, alveolar ducts and respiratory bronchioles. In this way still more of the already inadequate pulmonary parenchyma is thrown out of function, and respiratory distress is increased.

Discussion

The pathogenesis of hyaline membrane disease is indeed complex, for it must include an explanation of the source of the hyaline membrane, the manner in which they are deposited within the lungs, and the relationship between these membranes and the atelectasis. In this paper, only the relationship between the atelectasis and the hyaline membranes has been discussed in any detail.

There are two possible sources for the material which comprises these membranes, amniotic fluid and the infant's own plasma. Although no one has determined the amount of protein present within the membranes, it seems likely that it is large, so large that normal amniotic fluid would not contain such a quantity. For this reason, it seems more reasonable to assume that at least a part of this material must be derived from the infant's own blood. The controversy over this point will continue until unequivocal evidence is adduced for the support of one side or the other. Regardless of the source of this material, there remain the problems pertaining to its deposition and to its relationship with the atelectasis. The latter point has stimulated much argument, but there is no convincing proof for any hypothesis.

The circumstances which lead to the deposition of these membranes have been more or less ignored. It is generally said that the protein-containing material within the lungs is ordinarily absorbed completely via the capillary bed and the lymphatics. Support for this statement stems from the numerous observations on the resolution of pneumonia and on the disappearance of pulmonary edema in heart failure. The exact mechanisms involved in the removal of protein are unknown, but in most situations the protein and the freely diffusable electrolytes and water are removed concurrently. Experimental evidence indicates that there may be some concentration of protein and other non-diffusible substances,[12,13] but on a purely physical basis, this concentration should be limited by the osmotic properties of proteins and other non-diffusable substances. Excessive concentration may occur when exudates on exposed surfaces dry faster than they can be rehydrated, but it is difficult to believe that this can occur in pulmonary parenchyma with its long series of bronchi and with its abundant capillary bed. Another possibility exists, namely the denaturation of protein with an accompanying diminution in its osmotic effect as seen in the transformation of fibrinogen to fibrin, but there is no direct evidence for or against this hypothesis.

Hyaline membrane disease of the newborn remains an enigma. Despite thirty years of work by many capable investigators, it is the major cause of death in premature infants. The problems which this disease presents are clearly defined, but none of them has been solved.

Conclusion

1. Hyaline membrane disease is the chief cause of death in the first postnatal week.
2. It occurs predominantly in infants weighing between 1000 to 2500 grams.
3. The lesions of this disease are not present at birth but require postnatal existence of one or more hours.
4. Anatomically, it is characterized by hyaline membranes lining distended alveolar ducts and by collapse of alveoli.
5. There is insufficient evidence to establish the origin of the membranes, the manner in which they are deposited or their relationship to the atelectasis.
6. It is suggested that abnormal expansion of the lungs is the primary abnormality and that the membranes develop secondarily.

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Discussion

Dr. Edward P. Burch, Saint Paul: I should like to ask whether there was any relationship between this condition and retrolental fibroplasia. There seems to be quite conclusive evidence that retrolental fibroplasia is due to improper use of oxygen in the premature child, and, while I realize this pathologic picture is not the same in the two conditions, I would be curious to know whether the two diseases have anything in common.

Dr. Dawson: There are two reasons why it has been suggested that high oxygen tension plays a rôle in the pathogenesis of hyaline membrane disease of the newborn. First, most infants who exhibit this lesion have been exposed to high concentrations of oxygen and, second, it has been shown by Dr. Allan Hemingway, of the University of Minnesota, that membranes can be produced in experimental animals which have been exposed to high concentrations of oxygen. I do not think there is good evidence to incriminate oxygen as a cause of hyaline membrane disease because this lesion is seen in infants who have not been exposed to high concentrations of oxygen and also because, in many instances, the infant is not put in oxygen until he exhibits respiratory difficulty. So far as I know, there is no significant relationship between retrolental fibroplasia and hyaline membrane disease.

Dr. L. G. Rigler, University of Minnesota: We have been interested in this because of the fact that in the roentgenological literature it has been reported that premature infants, who appear to be exceedingly ill, may exhibit what appears to be normal x-ray films of the chest. This leads to a good deal of puzzlement among those who observe it. The reason apparently lies in the dilated alveolar ducts so that the lungs appear reasonably normal, whereas there is no exchange of gases going on. We ourselves have not seen any that look entirely normal. Perhaps we have not seen them early enough. The fact is, however, that the roentgenograms of the chest do not show as much abnormality as one would expect considering the severity of the symptoms. In most of the cases we have seen there are stippled shadows throughout both lungs indicating the presence of collapsed alveoli. I would like to ask Dr. Dawson whether he thinks there may still be an earlier stage than we have observed in which the amount of air in the alveoli is still normal.

Dr. Dawson: I think the amount of air present in the lungs during life is probably greater than we see here. We have no way of fixing these lungs *in situ*. When we open the chest they collapse. I think this represents collapse over and above that which was present when the child was alive.

Dr. H. L. Ulrich, Minneapolis: What was the condition of the diaphragm of these babies?

Dr. Dawson: So far as we know, there is nothing wrong with the diaphragm. I don't think we have any reason to believe that there is anything wrong with the way the air comes in. It is my idea that there is defective expansion of the alveoli.

Dr. H. B. Sweetser, Minneapolis: Is this a pathologist's disease or do we see it before?

Dr. Dawson: I think there is good reason to believe that some of these infants recover.

Dr. H. F. Flanagan, Saint Paul: We see what clinically we call atelectasis,. Some recover slowly, then a certain number die in spite of good therapy. I think Dr. Dawson is to be complimented on his exploration of this subject. The ones that die shortly after birth are probably those with hyaline membrane disease, and likely of antenatal origin. Many of these infants have a bloody mucous sputum noticed an hour or two after birth. Again, I wish to thank Dr. Dawson for this presentation.

Dr. W. P. Sadler, Minneapolis: How much of this is embryological to begin with?

Dr. Dawson: In infants weighing less than 1000 grams, the alveoli and the capillary bed within the lungs are incompletely developed, but as the lung matures, the structure of the alveoli becomes more apparent, the number of capillaries in the walls increases, and the lining epithelium disappears. The lesions of hyaline membrane disease are not seen in the extremely immature infants. This is not to say that the abnormal expansion does not occur in that period, but membranes are rarely seen. One wonders whether the paucity of blood vessels in the wall and the gland-like character of the alveoli may not be important insofar as the formation of membranes is concerned. I think there is no abnormality in the lungs which predisposes to the development of this disease.

Dr. E. B. Berglund, Minneapolis (by invitation): A couple of years ago before this group, with Dr. Wyatt, I presented the problem of atelectasis in the newborn. At that time, we were trying to save some of these babies with thoracotomy. However, as we went along, we decided we could not determine beforehand which ones are going to live and which are not. Until we had better prognostic ability, we could not determine the value of this procedure of thoracotomy. However, it was of interest that of some twenty-four babies who were most severely ill and on whom we did thoracotomy, twelve were delivered by cesarean section and all had some abnormality of delivery. We were convinced that the method of delivery had something to do with this condition. It may be that immaturity of development as with premature infants, as well as some abnormality of delivery, has something to do with it. Dr. Taylor at St. Mary's Hospital studied the cases there. The great majority of the babies in which atelectasis occurred were born within one to two hours after the mother had received demerol; leading again to the thought that something in the method of delivery, anesthesia, or other procedure, had something to do with the development of atelectasis.

Dr. Dawson: I don't know that I am in a position to answer Dr. Berglund's question in any detail. The maternal records on many of our patients are not too good and we have not looked them over too carefully. As far as demerol is concerned, I have no information on that.

Dr. Harold Buchstein, of Minneapolis, then read his inaugural thesis on "The Radical Treatment of Brain Abscess." (To be published later.)

The meeting adjourned.

Robert E. Priest, M.D., *Secretary*

History of Medicine in Minnesota

THE PIONEER DOCTORS OF CHIPPEWA COUNTY PRIOR TO THE YEAR 1900

LEON G. SMITH, M.D.
Montevideo, Minnesota

(Continued from April, 1955, issue)

Dr. De Willis Little spent most of his active medical career in Appleton, but he practiced in Montevideo a short time. The *Montevideo Leader* of November 6, 1891, had this to say: "Dr. D. W. Little has located in Montevideo. Although a newcomer here, he is by no means unknown to the people of this vicinity. He is a graduate of the Keokuk College of Physicians and Surgeons and has had sixteen years of practice. He will attend calls at any time in either city or country. His office is located over the Golden Eagle Bldg. Clothing House."

On October 30, 1891, he was reported to have removed a cancer from the lip of August Schroeder.

He apparently moved to Appleton in the spring of 1892, from where, according to his card in the Montevideo papers, he held office hours in Milan on Tuesdays and Saturdays, at least for a time. He practiced in Appleton to the time of his death, many years later.

Another doctor, whose name appears in Montevideo for only a short time, was mentioned by the *Commercial* of July 8, 1893: "**Dr. R. F. McMoine**, who is a graduate of McGill University in Montreal, Canada, is locating in Montevideo." The *Leader* stated: "Dr. R. F. McMorine *(sic)* is a new physician, who has placed his card in the business directory and has begun practice in Montevideo. His office will be above J. C. Arnold's Restaurant."

His stay was brief, because his card had disappeared from the business directory two months later, in the September issue, and there is no further information about him.

The past few names have been of only minor interest because these men were in Chippewa County for but a short time. The next one considered ranks well toward the top in importance. This is due not only to length of service in the county, but to his active interest in civic affairs as well.

Dr. Reuben Dewitt Zimbeck was born April 23, 1857, at Sharon, Wisconsin, the son of Henry Zimbeck and Clarissa Lowell Zimbeck. The father was born in Germany and came to America as a boy of eight. The mother was born in New York State and was a relative of James Russell Lowell. The Zimbeck and Lowell families moved from New York between 1835 and 1840, and located near Sharon, Wisconsin, as farmers. There was only one other child in the family, but they decided that Reuben, who was not strong as a child, was not rugged enough for farm work, so he was given a better than ordinary education. He graduated from Sharon High School and from college at Carthage, Illinois. He taught school at intervals to help finance his education and worked a good deal in drug stores in Minda and Crystal Springs, Illinois, where he learned sufficient Pharmacy to become a registered pharmacist. It is probable that during the time he worked in

Minda he studied medicine under the direction of his uncle, Dr. Lorenzo D. Lowell, and through his encouragement, went on to medical school. He took pre-medical work at the University of Iowa and his medical work at Rush Medical College, graduating in 1885.

His first location to practice was in Morton, Minnesota, in 1886. He remained there for four years, and then moved to Montevideo, where he stayed until 1915.

September 18, 1890, at Eagle Lake, Minnesota, he was married to Jessie Van Dolah, whose father, Rev. Thomas T. Van Dolah, was one of the pioneer preachers of Minnesota. Dr. and Mrs. Zimbeck had two children, Vera Mae and Marian Lucille.

In every community in which he resided, Dr. Zimbeck took an active part in all civic affairs. This was especially true in Montevideo, because it was here that he lived the longest (25 years) and those were the years of his prime. His most outstanding contribution in public affairs was in getting telephone service into Montevideo. As Dr. Rogers is given credit for bringing the Milwaukee Railroad division point to Montevideo, Dr. Zimbeck is credited with bringing the telephone here. He helped organize the Citizen's Telephone Company and, as its president, on May 5, 1899, announced that his company had bought out the local business of the Minnesota Central Telephone Company. This consolidation gave the home company the control of seventy-five to eighty instruments. This work took many long hours of his time, for which he received no pay except the satisfaction of helping his community.

In addition, he was a member of the finance committee of Windom Institute. He served upon the Village Council for several terms, some of them as president, and acted as secretary of the District Medical Society for many years.

He was not a member of any religious denomination, but was a regular attendant of the Congregational Church. He was also a devoted member of the I.O.O.F. for many years.

He moved from Montevideo to Maynard in 1915 and practiced there until his death in 1927, leaving his wife, who survived him for several years, and his daughters, Vera Zimbeck Hartley, now living in Deer Lodge, Montana, and Marian, now of St. Peter, Minnesota.

I knew Dr. Zimbeck well, and had great admiration and respect for him. He was a voracious reader, and was probably better posted on matters, both outside and inside his profession, than most of the doctors of his generation.

His practice was never as large as it should have been, commensurate with his ability as a doctor, especially in the field of diagnosis. I think it is fitting to copy an article by Richard Carlson in the *Maynard News,* following his obituary. It is a tribute, not only to Dr. Zimbeck, but to all similarly consecrated country doctors of the old school.

"THE COUNTRY DOCTOR"

"In the death of Dr. R. D. Zimbeck, our community lost a man of great abilities and fine character. The venerable old doctor was a true friend of mankind as well as a competent minister to its bodily infirmities. He always took time to season his prescriptions with a generous dose of sunshine and jaunty hopefulness. In recognition of his merits and work, therefore, let us for a moment reflect on the hardships and responsibilities attending the lot of those splendid heroes, the country doctors, of whom Dr. Zimbeck was one. They stand ready at all times of day or night to come to the bedside of the ailing. While we other mortals are enjoying the warmth of our houses, they are out fighting their way through blizzards and snowdrifts to allay the sufferings of humanity. In times of epidemics, when other people desert their best friends, the ever faithful practitioners stay on the job, bracing their minds and nerves for the extra work, knowing that the added strain and fatigue are undermining their own health. And in too many cases, their only reward is the pleasure of seeing their ministrations yield satisfactory results.

"Such deeds of sacrifice filled Dr. Zimbeck's long career of service to his fellowmen. In homage to his nobility of spirit, let us show our gratitude by trying to be equally unselfish in heart and deed, so that his efforts may find response in our enjoyment of a fuller share of health and happiness."

It is perhaps proper, at this time, to mention briefly the boys of Chippewa County who studied medicine and graduated during this period.

An account has already been given of **Dr. H. W. Rogers** and **Dr. Finn Koren.**

Dr. Oscar Frink was, fittingly, the first to so graduate, as his father, G. W. Frink, was generally considered to be the founder of Montevideo inasmuch as he was one of our earliest settlers, and the city was built on the land which comprised his original farm. Oscar graduated from Medical school February 2, 1891, and located in Wisconsin, where he lived and practiced at Pepin and Alma.

Dr. Oscar Bergstrom was mentioned several times in news items as helping the various doctors, undoubtedly during his vacations from medical school. The last item concerning him appeared December 3, 1892, and stated that he had graduated, was practicing at Cooperstown, North Dakota, and was in Montevideo, attending his brother-in-law professionally.

Dr. Charles Corse graduated June 11, 1897. The *Commercial* of October 1, 1897, told how "he worked upon the Murphy threshing rig until it completed its run. He left last Saturday by bicycle for his new field at Hewitt in Todd County, Minnesota, to locate in medical practice." Another item in the *Commercial* of July 29, 1898, after the beginning of the Spanish-American War, stated that he was an Assistant-Surgeon in the United States Army at Chickamauga; that he had received the twenty-five dollars donated by the loyal women of Hewitt and Montevideo; and that he had turned the money over to the hospital fund for use as needed.

With the exception of the very short time that Dr. N. A. Nelson was in Milan and Watson as early as 1885 or 1886, the smaller towns of the county had no resident doctors before 1894. In that year, one came to Milan and probably, also, one settled in Clara City. Maynard's first doctor located there in 1898 and Watson had none until the early 1900's.

Dr. Kristoffer Lundevall graduated in 1893 from the Christiania Medical College, which was probably a part of the University of Norway. He must have come to the United States soon thereafter, as the Medical Directory listed him in Milan in 1894. The *Milan News* of February 1, 1895, recorded his arrival there, with offices over the bank of Milan, but he was not licensed in Minnesota until October 8 of the same year. His name appeared occasionally in news items until November 26, 1897, when he was reported as leaving Milan to return to Norway. As his name never reappeared in the Medical Directory, it is to be assumed he remained there.

Dr. Henry W. Reiter appears to have been the first doctor to locate in Clara City. The Scott and Carver County records show that he graduated from the University of Minnesota in 1893, and first set up practice in Kerkhoven in Swift County. However, a news story in the *Montevideo Commercial* of September

14, 1895, indicated he must have settled in Clara City for a short time, namely "Dr. Reiter, formerly of Clara City, is now located at Kerkhoven." Later, in the year 1897, he located permanently in Shakopee, where the remainder of his life is covered in the "History of Medicine in Scott and Carver Counties."*

Following Dr. Reiter in Clara City was **Dr. Robert S. Davis,** about whom we have almost no information. There was no report in the Medical Directory except that he graduated from McGill University in 1894 and was listed in Clara City in 1896. An item in a Montevideo paper, August 12, 1896, mentioned his riding over from Clara City on his wheel, and brief mention of him also occurred in 1897, 1898 and finally June 30, 1899. In the Medical Directory of 1900, his name appeared from Lakeville, Jackson County, with the comment "no report." In 1909, his address was given as unknown.

After the names of several men of only passing interest, we come to one of much greater importance, as he practiced in Montevideo a considerable length of time and had the ability and personality to make a deep impression upon the history of the county.

Dr. Hans Waldemar Hendrickson was born near Copenhagen, Denmark, February 20, 1868. He had no full brothers or sisters. He came to America at the age of eight, making his home with an aunt near Watson, Minnesota. School attendance was very irregular, amounting to only a few weeks in rural schools each year. However, he did become well educated by self-teaching, as he used most of his spare money to buy books, and, even while working in the field and resting the horses, he would take a book along to read at such intervals.

By the age of eighteen, he was able to answer the requirements of a rural schoolteacher, and he taught several terms. Some schools had over thirty pupils, several of whom were older than he. In this way, he earned enough money to start studying medicine at the University of Minnesota. So, with no eighth grade or high school diploma, this immigrant boy fulfilled his life-long ambition to become a doctor.

While teaching a rural school in Swift County, he boarded at the home of Peter Ness, whose niece, Thora Ness, born in Vaage, Tessenden, Norway, had come to this country and acted as her uncle's housekeeper.

Hans and Thora were married before he started medical school, so it was hard sledding financially, but he worked in the University laboratories to help pay his way and provide for his family. There were three children, John, Melvin and Ella.

Dr. Hendrickson graduated from the University of Minnesota Medical School in 1893, served a two year internship in a hospital in Canton, South Dakota, and then set up an office for practice in Montevideo in October 1895. He never practiced in any other location.

He was on friendly terms with the three other competing doctors, Rogers, Bacon and Zimbeck, and news stories of the period frequently mention his having assisted, or been assisted by, one of them.

He was a member of the Synod Lutheran Church in Montevideo and belonged to many fraternal orders, including the Odd Fellows, the Woodmen and the Masonic Lodge. He served as Worshipful Master of the local lodge of the latter and also held at least one of the grand offices. He was especially interested in Masonic history and literature, and owned a large library of such works. He was also quite a gardener and horticulturalist and was very fond of music.

***Minnesota Medicine,* p. 798, July, 1948.

Like most doctors of the time, much of his work was country driving, and he kept two teams and hired a driver, so that he did much of his sleeping on the road. As his sons became older, they also helped in the driving. He had a buggy made to order for better resting. In the winter he had a quilt sewed up to form a sack, into which he could slip his feet with hot bricks or soap-stones to help keep warm.

He performed many an emergency operation in his own home upon the extended dining-room table.

He was quite a linguist, being able to speak fairly well in French, Spanish and German as well as his daily-used Scandinavian and English.

During the last few years of his life, his health was poor and he had to give up the more strenuous duties of his profession. However, he did considerable real estate business during this period and took extended trips to Mexico and Canada.

He was sick for several months before his death, which occurred December 1, 1912, due to Bright's Disease. It was a tragedy for his family and his community for so good and brilliant a man to die so young, only forty-four years.

One son, Melvin, died a few years later, but another son, John, lives in Sheridan, Wyoming. His daughter, Ella Hendrickson Nash, in Platte, South Dakota, and his widow, now 89 years old, is making her home with her daughter.

Dr. Tord Neilson was in Chippewa County but a very short time and there is little information about him. Dr. Drought's "History of Ottertail County from 1870 to 1940" states that he graduated from the University of King Frederick, Christiania, Norway, in 1881. He came to the United States soon thereafter, as he was licensed in Minnesota in 1883, with one of the earliest licenses issued, No. 19. At that time his address was Fergus Falls, Minnesota, and he continued to practice there several years. We know he was in Chippewa County in 1895 and 1896 as the following items appeared in the *Montevideo Commercial.* In the issue of January 11, 1895: "Dr. Tord Neilson, from Christiania, Norway, will be at Simon Bue's Hotel January 15." On March 15, 1895: "Dr. Tord Neilson moved into his new offices in Raffelson's Block, Monday." On May 10, 1895: "Dr. Tord Neilson, assisted by Drs. Murphy and Rogers, amputated the arm of an 80 year old man Tuesday."

Dr. Neilson was listed in Polk's Medical Directory as being in Montevideo in 1896, but no mention was made of him in the Montevideo papers after 1895, so it may be assumed that he moved that year to North Dakota. One item, however, might indicate that he first moved to Minneapolis from Montevideo, then returned to Fergus Falls about 1898 and 1899. At any rate, he was listed in Polk's Medical Directory from Kenmare, North Dakota from 1902 to 1906. Apparently he practiced there, and possibly at Edmore and Grafton, North Dakota, for many years.

(To be continued in October issue)

Reports and Announcements

MEDICAL MEETINGS

National

American Association of Blood Banks, eigth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Congress of Physical Medicine and Rehabilitation, Hotel Statler, Detroit, Michigan, August 28-September 2, 1955.

Association of Military Surgeons of the United States, Hotel Statler, Washington, D. C., November 7-9, 1955.

National Society for Crippled Children and Adults, Palmer House, Chicago, Illinois, November 28-30, 1955.

Pacific Northwest Industrial Health Conference, Multnomah Hotel, Portland, Oregon, September 12 and 13, 1955.

International

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

AMERICAN DERMATOLOGICAL ASSOCIATION ANNUAL PRIZE ESSAY CONTEST

The American Dermatological Association is offering a series of prizes for the best essays on original unpublished work on fundamental aspects of dermatology or syphilology. Prizes of $500, $400, $300 and $200 will be awarded for papers judged first, second, third and fourth, respectively. Competition is not limited to physicians but is open to scientists generally.

Manuscripts must be submitted in triplicate, should not exceed 10,000 words, and should be sent in under a nom de plume, with correct identification of the author in a separate sealed envelope. Deadline for the contest is November 15, 1955.

Entries should be sent to Dr. J. Lamar Callaway, Secretary, American Dermatological Association, Duke Hospital, Durham, North Carolina. Further information about the contest can be obtained by writing to that address.

AMERICAN UROLOGICAL ASSOCIATION OFFERS ESSAY AWARDS

The American Urological Association offers prizes of $500, $300 and $200 for essays on the result of clinical or laboratory research in urology. Competition is limited to urologists who have been graduated for not more than ten years and to men in training to become urologists. The first prize essay will be presented at the annual meeting of the association in Boston in May, 1956.

For complete details about the contest write William P. Didusch, Executive Secretary, 1120 North Charles Street, Baltimore, Maryland. Essays must be submitted before December 1, 1955.

MINNESOTA ACADEMY OF OCCUPATIONAL MEDICINE AND SURGERY

At the annual meeting of the Minnesota Academy of Occupational Medicine and Surgery on May 25 the following officers were elected: Dr. Leslie W. Foker, president; Dr. James R. Fox, vice president; Dr. Orley W. Foster, secretary, all of Minneapolis; Dr. John A. Williams, St. Paul, recorder; Dr. Tracy E. Barber, Austin, treasurer, and Dr. Gordon MacRae, Duluth, executive committee.

Named as fellows in the Industrial Medicine Association were Dr. James R. Fox and Dr. Wilford E. Park, both of Minneapolis. Dr. John F. Shronts, Minneapolis, was elected to the board of governors of the Industrial Medicine Association.

DISCONTINUATION OF THE RHEUMATIC FEVER PROGRAM, CRIPPLED CHILDREN SERVICES, DEPARTMENT OF WELFARE

The Rheumatic Fever Program of the Crippled Children Services of the Department of Welfare, which provided medical consultation and hospitalization for children from fifteen Minnesota counties, was discontinued as of June 30, 1955. This action was taken because federal funds for this special pilot project were withdrawn after careful study revealed a diminishing case load. It will be necessary to procure rheumatic fever diagnosis and treatment services for the children who previously would have been referred to the program, from the existing medical centers.

During the year beginning July 1, 1955, however, the Department of Welfare will continue to provide nursing consultation in heart disease to the public health nursing services in collaboration with the Minnesota Department of Health, but on a state-wide basis rather than just to the fifteen counties previously covered. Medical social work consultation will be available from the department on a limited basis upon request.

CONTINUATION COURSES

The University of Minnesota will present a continuation course in *Pediatrics for Pediatricians* September 21 and 22. Under the auspices of the Center for Continuation Study, the course will be held in the Mayo Memorial Auditorium on the University campus. One-half day will be devoted to each of the following general subjects: infectious diseases, hypersensitivity diseases, central nervous system disorders, and endocrine and metabolic abnormalities.

The guest faculty will include Dr. Lewis Thomas, professor of pathology, New York University College of Medicine, New York City, who will also deliver the Irvine McQuarrie Lecture on Thursday evening, September 22. The course will be presented under the di-

(Continued on Page 529)

In Memoriam

EDWARD HAROLD FROST

Dr. E. H. Frost, physician of Willmar, died in Willmar, June 2, 1955. He was seventy-nine years old.

Dr. Frost was born in Willmar in 1875, son of Dr. and Mrs. E. S. Frost. He attended the University of Minnesota and Louisville Medical College. Upon completion of his training, he began the practice of medicine with his father.

He had been a senior warden at St. Luke's Episcopal Church, where he and Louisa Wilcox were married, and where they celebrated their fiftieth wedding anniversary in 1949. He was a member of the Board of Education in Willmar, a city health officer, and division surgeon of the Great Northern railroad.

Dr. Frost was chief of staff at Rice Memorial Hospital and president of the Great Northern Surgeons Medical Association. He was a member of the Crow River Medical Society, the Kandiyohi-Swift-Meeker County Medical Society, the Minnesota State Medical Association, and the American Medical Association. He served for a time as vice president of the state association.

Dr. Frost is survived by his wife and three sons: Dr. Russell Frost, Glen Lake Sanatorium; Charles, an architect in Willmar, and William, district manager of the Travelers Insurance Company at Stillwater.

WENDELL B. GRINNELL

Dr. Wendell B. Grinnell, physican and surgeon of Preston, died May 30, 1955, at his home. He had suffered a stroke at his office on May 9. He was eighty-two years old.

Born in Waubeka, Wisconsin, Dr. Grinnell spent his boyhood in Saukville, Wisconsin. He attended normal school at Oshkosh and taught school before taking medical courses at Milwaukee College of Medicine and the University of Illinois. After a year of practice at Wykoff, Dr. Grinnell moved to Preston where he remained in practice for the next fifty-four years, until the time of his death.

Dr. Grinnell was a member of the Minnesota State Medical Association, and the Olmsted-Houston-Fillmore-Dodge County Medical Society. He was a member of the Preston O.E.S., Masonic Lodge, and Knights Templar. He served as a member of the village council and was county coroner for twenty years.

He is survived by his wife; his daughter, Mrs. Roger Vadheim of Tyler, Minnesota; two granddaughters, and a sister, Mrs. R. W. Brott of Marinette, Wisconsin.

WILLIAM ANTHONY MILLER

Dr. William A. Miller, born in Appleton, Wisconsin, on July 30, 1872, died in New York Mills, Minnesota, June 1, after nearly sixty years in the medical profession.

Dr. Miller graduated from the Kansas City Medical College in 1896. He then went to New York Mills and became an active member in many community activities. In February, 1953, a banquet was held by the Lions in New York Mills to honor Dr. Miller for his lifetime of service. He was a member of the school board and served on the Board of Health, as Deputy Coroner, as an examiner for selective service, and participated in many bond drives during both world wars.

He was a Life member of the Minnesota State Medical Association, the American Medical Association and had been a member of the Park Region District and County Medical Society since its inception. He was on the staff of St. James Hospital in Perham.

Dr. Miller is survived by his wife, Jessie; a sister, Mrs. F. G. Brabec, Perham; a son, Maurice, New York Mills; two daughters, Mrs. Oscar A. Carlson and Mrs. Eino Kyllonen, both of Minneapolis, and six grandchildren.

PHYLLIS E. ROSANDER

Dr. Phyllis Rosander, returning to Minnesota to enter practice after a residency at Sanford Hospital in San Francisco, was killed in a highway accident in Utah, May 29. She was thirty-four years old.

A native of Minneapolis, Dr. Rosander attended South High School, the University of Minnesota and the Woman's Medical College of Pennsylvania. She interned at Minneapolis General Hospital and was a fellow in obstetrics and gynecology at the University of Minnesota.

Dr. Rosander was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

She is survived by her husband, Dr. Richard Leigh of Grand Forks, N. D., and Minneapolis; her parents, Mr. and Mrs. John E. Rosander, Edina; a brother, Dr. John E. Rosander, Jr., San Francisco; and a sister, Mrs. Harry Gustafson, Edina.

NEW CONTRAST MEDIUM IN UROGRAPHIC EXAMINATIONS

A new contrast medium in radiological examination of the urinary tract, called Hypaque, has produced encouraging results, according to two Cleveland, Ohio, radiologists.

Doctors Joseph C. Root and William C. Strittmatter, associated with the department of radiology of the Cleveland Clinic, reported their findings in the May, 1955, issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.* Some 350 patients were examined with the new substance.

Their conclusions were as follows:

The value of Hypaque as a urographic contrast medium is twofold: it makes possible excellent visualization of the urinary tract, and it diminishes the number of postinjection reactions.

In a series of 350 patients, less than 10 per cent had any type of reaction following injection of the medium, and in general the urograms obtained were "pyelogram-like". in quality.

Of General Interest

Two new members have been appointed to the Minnesota State Board of Health by Governor Orville Freeman. They are **Dr. Frank H. Krusen**, Rochester, who succeeds, **Dr. F. W. Behmler**, state senator from Morris, and **Dr. Edgar J. Huennekens**, Minneapolis, who succeeds Professor Charles V. Netz, Minneapolis. Reappointed to the board was Professor Herbert Bosch, Minneapolis, vice president of the board.

* * *

Dr. L. E. Schneider of the Duluth Clinic, Duluth, attended the annual meeting of the American Psychiatric Association in Atlantic City from May 9 to 14.

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Dr. James F. Brusegard, Red Wing, was recently elected president of the Goodhue County chapter of the Society for the Mentally Retarded.

* * *

Dr. Russell M. Wilder, Rochester, was given the gold key award of the University of Chicago Medical School alumni association at a dinner meeting of the group in Chicago on June 9. Dr. Wilder retired in 1951 as professor and chief of the department of medicine of the Mayo Foundation.

* * *

A certificate of life membership in the American Academy of Pediatrics was awarded **Dr. Lillian L. Nye**, St. Paul, on June 18. Dr. Nye is also a life member of the American College of Physicians.

* * *

Dr. Peter J. Pankratz, Mountain Lake, has been elected to the board of directors of the Minnesota Alumni Association. He will serve for a term of three years.

* * *

The Minnesota Society for the Study of Diseases of the Heart and Circulation held its spring meeting at the Duluth Clinic, Duluth, on May 28. Local chairman for the meeting was **Dr. James J. Coll.**

* * *

Dr. Alan R. Jay, director of laboratories at Swedish Hospital, Minneapolis, was the principal speaker at commencement ceremonies at the Minnesota School of Laboratory Technique in Minneapolis on June 23.

* * *

Dr. C. Knight Aldrich, associate professor of psychiatry and neurology at the University of Minnesota, is leaving to become head of the department of psychiatry at the University of Chicago. Dr. Aldrich joined the staff of the University of Minnesota in 1947. While in Minneapolis he has been consulting psychiatrist for the Family and Children's Service and an attending psychiatrist at Minneapolis Veterans Hospital.

Dr. B. F. Pearson, Shakopee, attended the annual convention of International Rotary in Chicago on June 6 and 7. Dr. Pearson is president of the Shakopee Rotary Club.

* * *

Dr. Oliver E. H. Larson, Zumbrota, has been named a member of the medical staff of the Olmsted Community Hospital, Rochester. With his appointment the staff membership now totals twenty-one.

* * *

At the twenty-first annual meeting of the American College of Chest Physicians in Atlantic City, New Jersey, June 1 through 5, **Dr. Herman J. Moersch**, Rochester, was named president-elect of the organization. Elected as chairman of the board of regents of the college was **Dr. John F. Briggs**, St. Paul. **Dr. Jay Arthur Myers**, Minneapolis, was re-elected as chairman of the editorial board of the college journal, *Diseases of the Chest.*

* * *

Dr. Josiah Fuller of the Duluth Clinic, Duluth, attended the annual meeting of the American Association for Thoracic Surgery in Atlantic City from May 24 to 27.

* * *

A citation for outstanding community work was presented to **Dr. Charles C. Cooper**, St. Paul, by Macalester College recently. The citation was one of three which were awarded to alumni by the St. Paul college.

* * *

Among Minnesota physicians who attended a combined meeting of pediatric societies in Quebec, Canada, during the middle of June was **Dr. Albert V. Stoesser**, Minneapolis. The organizations conducting the joint meeting were the American Pediatric Society, the British Paediatric Association, the Society for Pediatric Research and the Canadian Paediatric Society.

* * *

June 26 was Dr. Sogge Day at Windom, with residents of the area and professional colleagues gathering to pay tribute to **Dr. L. L. Sogge** for his fifty years of service in the field of medicine. Keynote speaker on the program honoring Dr. Sogge was **Dr. Frederic J. Kottke**, former resident of Windom and now head of the department of physiotherapy at the University of Minnesota. The days activities included a dinner for medical men of the area, followed by an afternoon program of speeches and entertainment in the high school auditorium. Among the committee members who planned the day of honor for Dr. Sogge were **Dr. J. H. Dokken** and **Dr. H. P. Basinger**, both of Windom.

Principal speaker at high school graduation ceremonies at Kenyon on May 31 was **Dr. H. J. Svien,** Rochester.

* * *

One of the speakers at a convention of the Minnesota Bankers Association in Minneapolis on June 14 was **Dr. Charles W. Mayo,** Rochester, who spoke on "The United Nations, Its Problems and Future."

* * *

Dr. William R. Bagley, Duluth, was the main speaker at a meeting of the Duluth Multiple Sclerosis Society at St. Mary's Hospital, Duluth, on May 25.

* * *

Dr. F. J. Plondke, who practices in Saint Paul, received an honorary degree of doctor of science at commencement exercises at Morningside College, Sioux City, Iowa, on May 30. Dr. Plondke is an 1895 graduate of the college.

* * *

Dr. and Mrs. A. H. Borgerson, Long Prairie, left in mid-May for a six-week tour of Europe. While in Geneva, Switzerland, Dr. Borgerson attended a meeting of the International College of Surgeons, following which he joined a group of surgeons on a month's tour of European medical centers.

* * *

"Observations of Metabolic and Anti-rheumatic Effects of Meticorten" was the title of a paper presented by **Dr. Howard F. Polley,** Rochester, at a two-day symposium preceding the annual meeting of the American Rheumatism Association in Atlantic City, New Jersey, on May 31 and June 1.

* * *

Dr. Donald C. Balfour, Rochester, has been named winner of the American Medical Association's distinguished service award for 1955. A retired member of the Mayo Clinic since 1949, Dr. Balfour was previously head of the clinic's division of surgery and director of the Mayo Foundation for Medical Education and Research.

* * *

Four Minneapolis physicians presented a paper at a combined meeting in Quebec, Canada, in mid-June held by pediatric societies from the United States, Britain and Canada. Entitled "Ventral Septal Defect in Infants and Children (Results of Direct Closure in Twenty-five Patients)," the paper was presented by **Dr. Paul Adams, Jr., Dr. Brian Kiely, Dr. Morley Cohen** and **Dr. C. Walton Lillehei,** all of Minneapolis.

* * *

Main speaker at commencement exercises at the Willmar School of Practical Nursing on June 26 was **Dr. W. E. Macklin,** Willmar. Eighteen practical nurses were graduated from the school, which is affiliated with the Rice Memorial Hospital at Willmar.

* * *

Dr. M. J. Shapiro, formerly of Minneapolis, was recently awarded a distinguished achievement medal by the University of Minnesota. Dr. Shapiro, who helped to bring about the building of the University's Variety Club Heart Hospital, left Minneapolis in 1951 to practice in Beverly Hills, California.

* * *

Dr. Bernard C. Halper, Hibbing, is now spending two days each week at Itasca Memorial Hospital at Grand Rapids, providing the hospital staff with diagnostic x-ray service.

* * *

Dr. William L. Benedict, Rochester, retired head of the section on ophthalmology at the Mayo Clinic, was awarded an honorary doctor of laws degree at commencement ceremonies at the University of Michigan, Ann Arbor, on June 11. Dr. Benedict, who joined the Mayo Clinic in 1917, has been retired since 1950.

* * *

Governor Orville Freeman has appointed **Dr. Magnus Westby,** Madison, to the state board of medical examiners to replace **Dr. J. F. DuBois,** Sauk Centre.

* * *

Dr. Robert J. Delmore has left Roseau for New Orleans, Louisiana, where he is specializing in the practice of surgery at Charity Hospital.

* * *

Dr. McKaig Day was held at Pine Island on June 26, when residents of the area and medical colleagues paid tribute to **Dr. Carle B. McKaig** for his nearly fifty years of medical and civic service to the community. Principal speaker at the afternoon program honoring Dr. McKaig was **Dr. Charles W. Mayo,** Rochester. Also appearing on the program were numerous other medical and civic leaders. The honorary day ended with an evening program featuring musical entertainment and a fireworks display.

* * *

Dr. Ivan D. Baronofsky, former associate professor of surgery at the University of Minnesota and director of surgical research at Ancker Hospital, St. Paul, has been appointed chief of thoracic surgery at the U. S. Naval Hospital, San Diego, California. Dr. Baronofsky entered military service last February.

* * *

Construction of the Albany Medical Center has begun at Albany, and completion of the clinic is expected early this fall. The center is being built by **Dr. F. H. Baumgartner** and **Dr. R. J. Salk,** who formed a medical partnership last January. The clinic will be 32 by 76 feet, with facilities for three physicians and their assistants.

* * *

Dr. Randall G. Sprague, Rochester, was presented with a merit award on June 10 by his Northwestern University graduating class of 1930.

* * *

Dr. Ralph T. Taubert, psychiatrist at the Rochester State Hospital for the past two years, has moved to California and become a member of the staff of Patton State Hospital, San Bernardino.

* * *

The Brainerd Clinic, founded in 1896, has moved from its original location into a new clinic building in west Brainerd. The clinic, which was founded by the late **Dr. J. L. Camp** and **Dr. John Thabes, Sr.**, is now operated by **Dr. John Thabes, Jr., Dr. G. I. Badeaux** and **Dr. Maurice Meller.**

* * *

Dr. W. W. Rieke, Wayzata, was the subject of a brief biographical sketch in the Town Toppers column of the *Minneapolis Star* on June 17. The column features biographies of outstanding citizens.

* * *

Dr. Eric P. Lofgren, Rochester, a medical missionary for the Lutheran Church, has left for Eket, Nigeria, West Africa, where he will head the Lutheran Synodical Conference Hospital. He was accompanied and will be assisted by his wife, Dorothy, a medical secretary, and Ethel M. Giziewski, a missionary nurse and former administrative pediatric supervisor at the University of Minnesota Hospitals.

* * *

The New Brighton Clinic, construction of which was recently completed in New Brighton, has opened its doors to the public. The new medical center is operated by **Dr. R. J. Houle** and **Dr. D. J. Dummer.**

* * *

Dr. N. E. A. Leppo, Duluth, spoke on the subject, "Children's Health Around the World," at a meeting of the Nettleton Parent-Teacher Association on April 21.

* * *

Dr. W. E. Park, occupational health physician with the Minneapolis Health Department, has been awarded a fellowship in the Industrial Medical Association in recognition of his contributions to industrial medicine.

* * *

The award of merit of the Ohio Valley Proctologic Society has been given to **Dr. Louis A. Buie,** senior consultant in the section of proctology at the Mayo Clinic, Rochester. The award was made during the recent annual meeting of the American Proctologic Society in New York City.

* * *

Three Duluth physicians participated in a television program on cancer over WDSM-TV on April 17. Taking part in the question-and-answer forum were **Dr. F. H. Magney, Dr. W. J. Ryan** and **Dr. F. J. Hirschboeck** of Duluth.

* * *

Three medical exhibits planned and produced by members of the staff of the Mayo Clinic were given top honors at the annual meeting of the American Medical Association in Atlantic City, New Jersey, early in June. The gold medal, highest AMA award for exhibit presentation, was awarded to "Esophagitis," an exhibit prepared by **Dr. H. W. Schmidt, Dr. F. H. Ellis, Jr., Dr. Arthur M. Olsen, Dr. J. H. Grindlay** and **Dr. A. H. Bulbulian.**

Receiving the certificate of merit in the section on general practice was the exhibit, "Congenital Dysplasia and Dislocation of the Hip," by **Dr. L. E. Harris** and **Dr. Paul R. Lipscomb.** The third winning exhibit, "Cushing's Syndrome: A Progressive and Often Fatal Disease," by **Dr. Randall G. Sprague, Dr. James T. Priestley, Dr. Waltman Walters, Dr. R. V. Randall, Dr. R. M. Salassa** and **Dr. D. A. Scholz,** was awarded the certificate of merit as best in the section on internal medicine.

NEW LOCATIONS

Dr. John Flatt, formerly of Fargo, North Dakota, has completed a residency at Philadelphia General Hospital and has moved to Wabasha, where he is a new member of the Community Clinic.

* * *

Dr. Harry F. Burich has moved to Rochester from LaCrosse, Wisconsin, and has been named to the staff of the Olmsted Medical Group.

* * *

Dr. Ralph F. Mach has returned to Pine City after two years in military service and has reopened his offices for the practice of medicine.

* * *

Dr. A. H. Thompson, formerly of Litchfield, has moved to Madelia and become associated in practice with **Dr. H. E. Coulter** and **Dr. W. J. Hruza.**

* * *

Dr. Jack K. Gardner, a native of Fairmont, who recently completed a surgical residency at Northwestern Hospital, Minneapolis, has opened offices for the practice of medicine at Ceylon.

* * *

Dr. Jack A. Guy, who has been with the Public Health Service at the White Earth Indian Hospital for two years, has returned to New London and reopened his practice there.

MINNESOTA BLUE SHIELD-BLUE CROSS

During the month of April, 1955, 12,461 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of April 30, 1955, to 1,007,886 participant subscribers.

Hospitalization expense for the month of April, 1955, amounted to $1,508,700.60, or a decrease of $257,103.30 over the previous month. Past statistics reveal that the month of March is usually the peak month for cases admitted and hospitalization expense incurred by Blue Cross subscribers. The April decrease of $257,103.30 though considerable is in accord with past years' experience.

It is interesting to note that the decrease in April over March, 1955, was attributed to a lower Blue Cross usage by members plus a reduction in the length of stay of each patient. A total of 13,615 Blue Cross subscribers required hospital care during April, 1955.

Commencing July 15, 1955, erection of 200 Scotchlite Blue Cross-Blue Shield highway signs will get under way throughout Minnesota.

The signs, 9 feet by 13 feet, will feature an exact replica of the Blue Cross-Blue Shield emblems in highly reflective scotchlite, emphasizing the slogan: "You Need Them Both."

Located at the bottom portion of each sign will be a courtesy panel whereby the exact distance to the next city or town will be shown in scotchlite.

The locations for the signs have been carefully selected so as to assure positions where the traffic count is high.

Blue Cross-Blue Shield realizes the necessity of constantly reminding the public of the importance of their benefits under both plans . . . and to those who are not members, the necessity of prepaid hospital, medical, surgical, maternity protection. It followed, that highway signs located in strategic positions throughout Minnesota, could augment this job most economically.

The objective of the program is twofold: The service feature of the distance-to-the-next-town, courtesy panel will be accepted with enthusiasm throughout the state. Also, the constant reminder theme emphasizing both Blue Cross-Blue Shield will supplement the excellent work already being done by the doctors and hospitals.

The program will be completed by August 15, 1955.

Of interest to all physicians are the improvements in both the Blue Shield contract and its schedule of payments, which have been approved by the Board of Directors for services rendered Blue Shield subscribers on and after July 1, 1955.

Briefly the improvements in the new contract provide as much as 120 days of in-hospital medical care or combined surgical and in-hospital medical care each 12 months for each unrelated eligible condition or illness instead of the 70 days previously provided.

Another highly important improvement in the contract is the addition of benefits for x-ray treatment. Both deep and superficial x-ray treatments rendered Blue Shield subscribers on and after July 1, 1955, for such conditions as cancer, blood diseases, female disorders and certain skin lesions, as listed in the schedule of payments, are provided in this revision of the contract.

In addition to the increased benefits provided in the contract, extensive revision of the schedule of payments makes available more liberal medical, surgical and other related service benefits for services rendered Blue Shield subscribers on and after July 1, 1955.

Among the important benefit increases are the addition of allowances for intensive in-hospital medical care in certain illnesses and electroshock treatment in psychiatric cases. Also, there are some 42 increases in benefits for surgical procedures and related services. Anesthesia services have been so changed that many subscribers will receive greater anesthesia benefits. Thirty-two new items which have not previously been listed in the schedule of payments have been added. Improvements in the wording and description of a number of items have been made to bring the schedule of payments up to date with the present-day practice of medicine.

All of these many improvements in both the Plan A and the Plan B contracts and schedules of payments are being made without any increase in the rates or cost of the contracts to the subscribers. These increased benefits mean several hundred thousand dollars more in benefits paid to doctors of medicine for subscribers' medical, surgical and maternity care each year, and are possible only as a result of the increased enrollment and the support and co-operation of the members of the medical profession.

CONTINUATION COURSES

(Continued from Page 524)

rection of Dr. John A. Anderson, head of the department of pediatrics. Registrants will also be invited to attend the meeting of the Northwest Pediatric Society which will be held on Friday, September 23.

The University of Minnesota announces a continuation course in *Office Psychotherapy in Psychosomatic Problems* from September 7 to 9. The course will be held at Douglas Lodge located deep in the woods of northern Minnesota on the shores of Lake Itasca.

The therapeutic management of psychosomatic patients will be the central theme, and the techniques of value to the general physician and to the non-psychiatric specialist will be emphasized. The course will be presented under the direction of Dr. Donald W. Hastings, professor and head of the department of psychiatry and neurology.

Sessions will be arranged to permit time for fishing, boating, and other recreational activities. The fee will be $130, which will include registration, tuition, meals and lodging, instructional material, transportation to Douglas Lodge and return, and fishing license. Further information may be obtained from the Director of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14, Minnesota.

A continuation course in *Clinical Chemistry for Physicians* will be presented by the University of Minnesota October 6 to 8. The two-and-a-half day session, which will be held in the Center for Continuation Study and the University of Minnesota Hospital Laboratories, has been designed to help the physician in setting up and supervising laboratory procedures in his own office or local hospital. Attendance will be strictly limited, and instruction will consist principally of actual experience at the laboratory benches. Dr. Ellis Benson, acting director of clinical laboratory medicine, will be in charge of instruction.

The University of Minnesota announces a continuation course in *Techniques in General Practice* which will be held at the Center for Continuation Study and the University of Minnesota Hospitals on October 20 and 21. The course will consist principally of demonstrations of certain techniques of interest and importance to the physician practicing general medicine such as tracheal intubation, tracheotomy, and exchange transfusion. Registration will be limited. This program will be presented on the two days immediately following the annual refresher course of the Minnesota Academy of General Practice which will be held in Minneapolis on October 19.

The University of Minnesota in conjunction with the Minnesota Tuberculosis and Health Association will present a continuation course in *Tuberculosis for Lay Persons* particularly interested in this field. The course will be held at the Center for Continuation Study on the University of Minnesota campus from October 10 to 12, and emphasis will be placed on the public health aspect of tuberculosis.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

A PRIMER OF ELECTROCARDIOGRAPHY. Third Edition. George E. Burch, M.D., F.A.C.P., Henderson Professor of Medicine. Tulane University School of Medicine; Physician-in-Chief, Tulane Unit, Charity Hospital; Consultant in Cardiovascular Diseases, Ochsner Clinic, Ochsner Foundation Hospital, Veterans Administration Hospital; Visiting Physician, Touro Infirmary; Consultant in Medicine, Hotel Dieu, Illinois Central Hospital, New Orleans; and Travis Winsor, M.D., F.A.C.P. Assistant Clinical Professor of Medicine, University of Southern California Medical School; Director of Nash Cardiovascular Foundation, Hospital of the Good Samaritan; Junior Attending Physician, Los Angeles County Hospital; Junior Attending Physician and Diplomate, Children's Hospital, Cardiac Department, Los Angeles. 286 pages. Illus. Price $5.00. (Philadelphia: Lea & Febiger, 1955.

DOCTORS IN THE SKY. Robert J. Benford, M.D., Colonel, Medical Corps, United States Air Force. 326 pages. Illus. Price $8.75. Springfield, Ill.: Charles C Thomas, 1955.

This is the story of the development of the Aero Medical Association. Louis H. Bauer, M.D., "the father of Aviation Medicine," founded the organization in 1929. Since that time there have been twenty-six annual meetings. The advance of aviation medicine is unfolded by relating the events of each meeting.

From the early medical problems involved in "blind" flying (now known as instrument flight) up to the present medical aspects of supersonic flight and space medicine, the reader is impressed by the strong alliance between medicine and aeronautics.

The rôle of Aviation Medicine in the Military, Civil Aeronautics Administration and in the commercial airlines is related in historical perspective.

Of special interest to Medicine in Minnesota are the important contributions made by two former Minnesota physicians, John P. Stapp, M.D. (Minn. '44) and W. Randolph Lovelace II, M.D. (Mayo Clinic '34-'41).

Dr. Stapp (Col., USAF) is at present conducting experiments in deceleration for the Air Force. Dr. Stapp has traveled up to speeds of 632 miles per hour on a rocket-powered sled, and he has withstood a deceleration force of 35 G.

Dr. Lovelace developed and tested for the Air Force oxygen bail-out equipment. He successfully parachuted to earth from 40,000 feet and, in conducting this experiment, he discovered that the opening shock of a parachute at high altitudes was extremely high. Previous to this time, it was thought that the opening shock would be less due to the decreased density of the atmosphere. Along with Drs. W. M. Boothby and A. H. Bulbulian (other Mayo researchers), Dr. Lovelace developed the now famous BLB oxygen mask.

I found the Aviation Medicine aspects of the book very intriguing; however, at times the narration becomes tedious when documenting the administrative side of the Aero Medical Association.

J. E. Power, Jr., M.D.

FIRST PROSECUTION UNDER PHYSICAL THERAPY LAW

(Continued from Page 510)

though there was no law in Minnesota whereby physical therapists could register as such, the defendant was informed that she was violating the Basic Science Law and could not legally continue her practice or maintain an office. She subsequently was employed as a nurse by a doctor of medicine in Rochester who stated that he had taken entire charge of her work, so she was not prosecuted at that time.

In 1951, the Legislature of Minnesota passed a law providing for the examination and registration of physical therapists by the Minnesota State Board of Medical Examiners. The law also provides that any person not registered as a physical therapist who in any manner represents himself as a physical therapist, shall be guilty of a gross misdemeanor. Miss Lundeen never submitted an application to the Board for registration as a physical therapist. In Minnesota, a physical therapist is defined as a person who practices physical therapy "under the prescription, and direct supervision of a person licensed and registered in this state to practice medicine and surgery and whose license is in good standing." Miss Lundeen, even if registered, could not maintain an independent office.

The records indicate that Edith Ingeborgh Lundeen was born in Sweden in 1898 and that after graduating from Red Wing City Hospital, in Minnesota, she became registered as a nurse in this state on December 1, 1923. The Minnesota State Board of Medical Examiners and the State Examining Committee for Physical Therapists wishes to commend Mr. D. P. Mattson, Olmsted County attorney, for the able and efficient manner in which he handled this case.

MINNEAPOLIS WOMAN SENTENCED TO SERVE TWO-YEAR TERM FOR ABORTION

Re: State of Minnesota vs. Alma Dora Peterson

On June 24, 1955, Alma Dora Peterson, sixty-six years of age, 4168 Bryant Ave. N., Minneapolis, entered a plea of guilty to an information charging her with the crime of abortion, before the Hon. Earl J. Lyons, Judge of the District Court of Hennepin County. On the same date, Mrs. Peterson also pleaded guilty to an information charging her with three prior convictions for felonies and thereupon the Court sentenced her to be imprisoned in the State Reformatory for Women at Shakopee, Minnesota, for a term of not to exceed two years. Judge Lyons stayed the execution of the sentence to July 5 to permit Mrs. Peterson to arrange her personal affairs. Because of her three prior convictions the defendant could have been sentenced to life imprisonment.

Mrs. Peterson was arrested on May 5, 1955, by Minneapolis police officers following the hospitalization in Minneapolis General Hospital of a twenty-three-year-old divorcee who was suffering from the aftereffects of a criminal abortion. The defendant admitted that she had performed the abortion upon the patient on April 30, 1955, by means of a catheter and wire, using a coat hanger for the wire. After doing the abortion, for which she was paid the sum of $50.00. Mrs. Peterson instructed the patient to take quinine tablets and castor oil.

The defendant, who has no medical training, has been sentenced three times in the past in Hennepin County District Court on abortion charges. On the first two occasions, on December 21, 1934, and February 21, 1944, she was placed on probation. However, on April 12, 1948, Mrs. Peterson entered pleas of guilty to two informations, one of them charging her with the crime of abortion and the other charging her with having two prior convictions for felonies. She was then sentenced by the Court to serve one year in the Minneapolis Women's Detention Home.

Original Contributions

Histoplasmosis

Preliminary Report of a Family Outbreak in Minnesota

RICHARD T. SMITH, M.D., and S. J. RAETZ, M.D.
Minneapolis, Minnesota

THE first recognized case of histoplasmosis in the United States was that of a Minnesota farm woman described twenty-eight years ago by Watson and Reilly.[13] Since then, investigative efforts in many parts of the country have established histoplasmosis as a fungus disease of widespread geographic distribution and a broad range of clinical manifestations.[1,2,3] The currently recognized endemic area has not included Minnesota with the result that scant attention has been given to the disease as a medical problem in this state. Recent experiences with a family outbreak and the studies thereby stimulated, however, suggest that this disease may be of greater medical importance in the state than previously appreciated. This report describes the clinical features of the outbreak and the preliminary epidemiological investigation. Further epidemiological studies will be presented in detail elsewhere.[11,12]

Case Reports

Case 1.—(D. H.) On August 27, 1953, a four-year-old Wright County farm boy was admitted to the University of Minnesota Hospitals, Pediatric Service, with a history of fever of one week duration. He had always been in excellent health and had developed normally until approximately two months prior to admission when he became somewhat listless, lost weight, and was noted to have night sweats. Fever was first recorded one week before admission. Examinations during that week at a St. Cloud hospital revealed irregularly spiking fever with no abnormal physical findings. Peripheral blood studies had revealed the white blood count to be 8,050 per cu. mm. with 64 per cent neutrophils, 29 per cent lymphocytes, 7 per cent eosinophils. Erythrocyte sedimentation rate, Westergren, was 20 mm. in one hour. The initial chest x-ray (Fig. 1a) was considered normal. He was transferred to University Hospitals for further diagnostic studies.

At the time of admission to the University Hospitals the temperature was 103.8° F., the pulse 120; respiratory rate was 30; blood pressure was 90/58. Physical examination revealed pallor, a barely palpable spleen and liver, but was otherwise normal. Laboratory studies gave the following results: white blood count 7,350 per cu. mm. with 69 per cent neutrophils, 24 per cent lymphocytes, 4 per cent monocytes, 3 per cent eosinophils; hemoglobin 12.3 gm. per cent; urinalysis negative; initial cultures of the nose and throat, blood, stools were negative for pathogens; tuberculin test—negative; chest x-ray negative.

Bone marrow biopsy revealed microorganisms morphologically identical to *Histoplasma capsulatum* in its yeast form in many macrophages (Fig. 2). Cultures of this material grew out of the yeast form of this fungus on blood agar plates incubated at 37° after nine days, and the mycelial form of the fungus on Sabouraud's media after seventeen days incubation at room temperature. Typical tuberculate chlamydospores were subsequently demonstrated. Complement fixation with whole yeast phase antigen revealed a 1-80 titer of circulating antibody.* Skin tests on four occasions with two lots of histoplasmin failed to give a positive result. The fungus was subsequently cultured from a venous blood specimen and the yeast forms were seen within polymorphonuclear leukocytes on serial examination of peripheral blood smears.

The clinical course in relation to certain laboratory findings, and treatment is shown in Figure 3. After establishment of the diagnosis on morphologic grounds,

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From the Laboratories of the Variety Club Heart Hospital, Department of Pediatrics, University of Minnesota, Minneapolis, Minnesota.

Aided by a grant from the Graduate School Medical Research School Fund, University of Minnesota.

Dr. Smith is Helen Hay Whitney Research Fellow, University of Minnesota; Dr. Raetz is County Health Officer, Wright County, Minnesota.

*The authors are indebted to Dr. Amos Christie, Vanderbilt University, Nashville, Tennessee, for performance of this test.

treatment with ethyl vanillate was started.** Figure 3 shows the dosage of ethyl vanillate employed and the blood levels achieved. Treatment was begun with a dose of 0.5 gm. per kilo; this was gradually increased to 0.9 grams per kilo, divided into twelve daily doses. This maintained the blood levels in the recommended range of 20 to 30 mg. per 100 cc. without significant difficulty until the time of death.†

In spite of treatment there occurred rapid enlargement of the liver and spleen, continued high, spiking fever, and progressive leukopenia and thrombopenia. The hemoglobin value fell from 12.3 gm. per 100 cc. on admission to 6.6 gm. with the appearance of many reticulocytes and normoblasts in the peripheral blood. Whether this was an effect of the drug, or hemolytic anemia resulting from the disease could not be determined. Treatment was ineffective in controlling the rapidly increasing numbers of yeast forms in the bone marrow, or the extent of growth of cultures of the material. Marked respiratory distress accompanied by chest infiltrations demonstrable by x-ray (Fig. 1b) marked the last few days of life. He expired on September 10, twenty-five days after onset of fever. No post-mortem examination was allowed.

Morphologic, serologic, and cultural studies established the diagnosis of disseminated histoplasmosis beyond doubt, in spite of a negative skin test on several occasions. Failure to react to the skin test antigen in the presence of overwhelming infection has been noted by others.

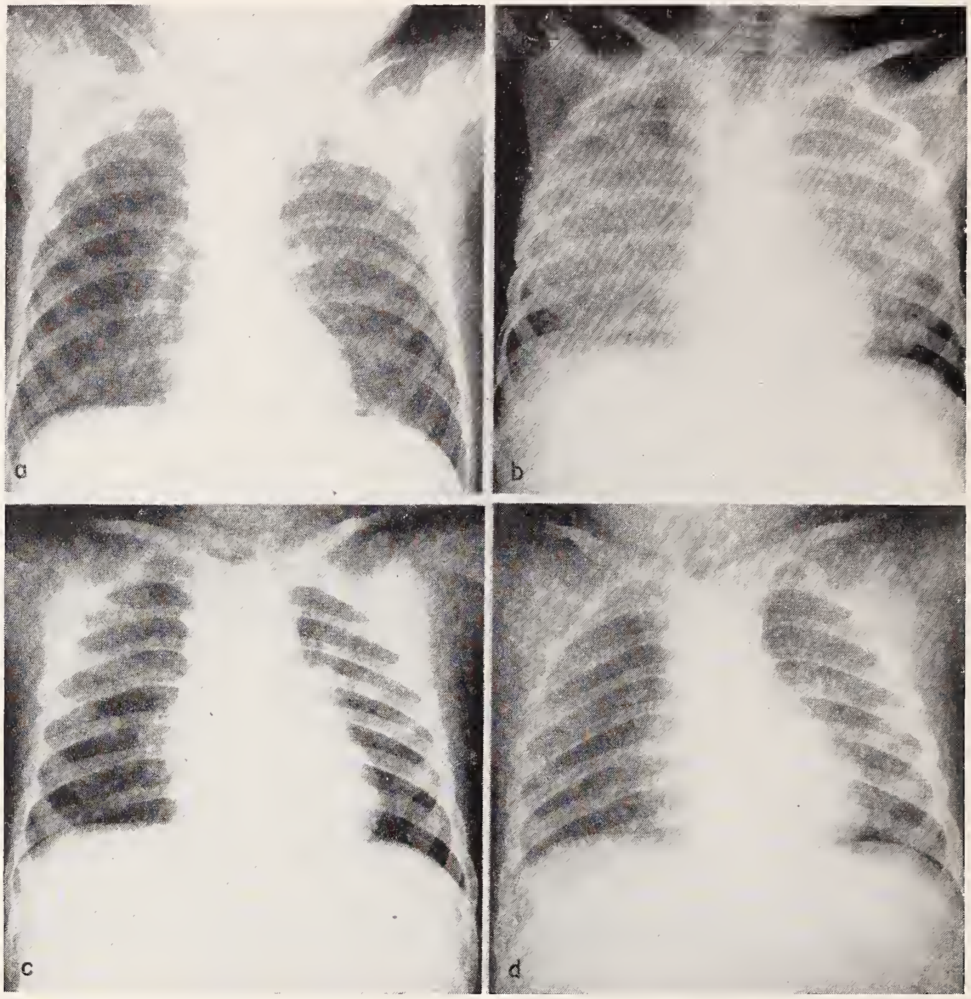

Fig. 1. Chest x-rays illustrating serial changes which occurred in members of H. family involved in the Maple Lake, Minnesota, outbreaks of histoplasmosis 1949-1953.

(a) Case 1, D. H., August 23, 1953, at onset of fever-negative.

(b) Case 1, D. H., September 10, 1953, after beginning of respiratory distress—shows diffuse bilateral interstitial pneumonitis.

(c) Case 2, A. H., June 16, 1954, two weeks after onset of illness—shows peribronchial infiltration bilaterally with a pneumonic process in the left upper lobe subtended by what appears to be an enlarged hilar lymph node.

(d) Case 2, A. H., July 17, 1954, six weeks after onset of illness. Clearing of the left upper lobe process, and the peribronchial infiltration is shown.

**Ethyl vanillate was generously supplied by E. R. Squibb & Sons, New York.

†Determinations of blood levels of ethyl vanillate were performed in co-operation with R. J. Salmon.

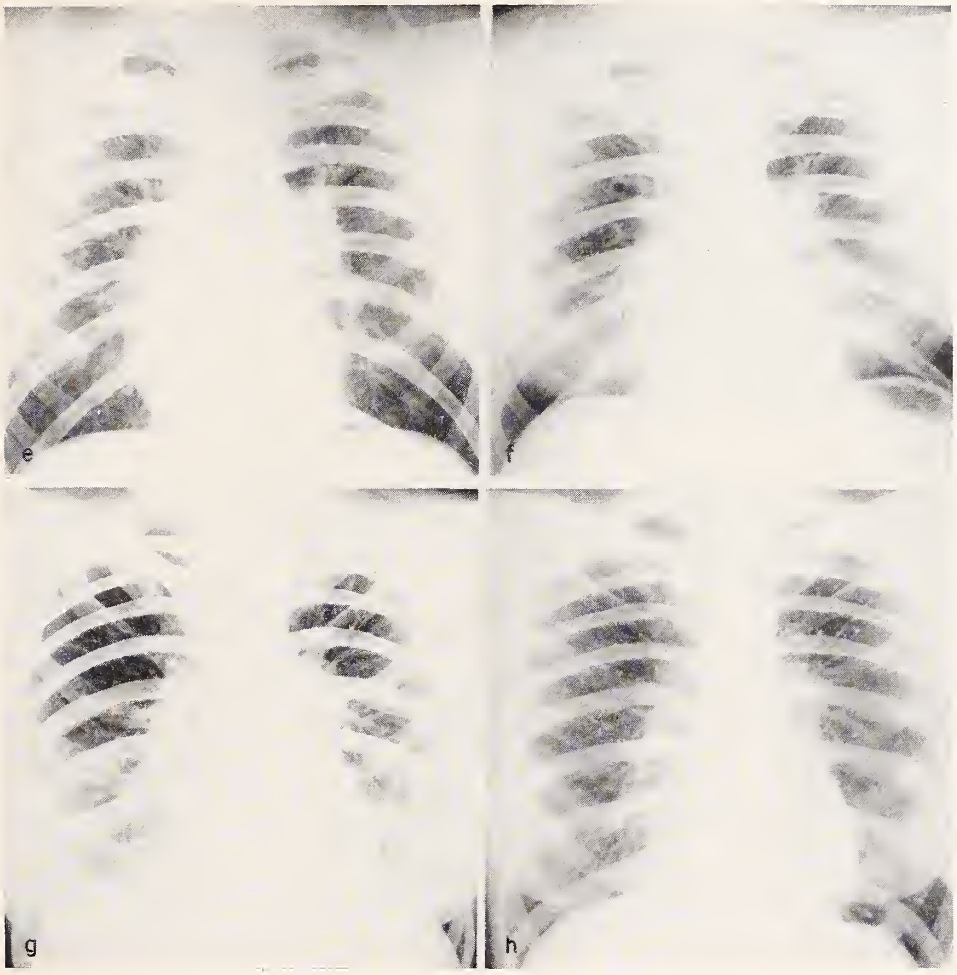

Fig. 1. (e) Case 3, C. H., February 25, 1949, two days after onset of symptoms. Nodular densities and infiltrations in both lung fields with marked increase in hilar shadows.

(f) Case 3, C. H., June 21, 1954, five years after illness, asymptomatic. Fine nodular parenchymal and hilar calcifications are shown.

(g) Case 4, L. H., March 8, 1949, two weeks after onset of symptoms. Shows soft nodular and linear densities in both lung fields with increased size of hilar shadows.

(h) Case 4, L. H., April 27, 1953, four years after illness, asymptomatic. Shows extensive bilateral nodular parenchymal calcifications.

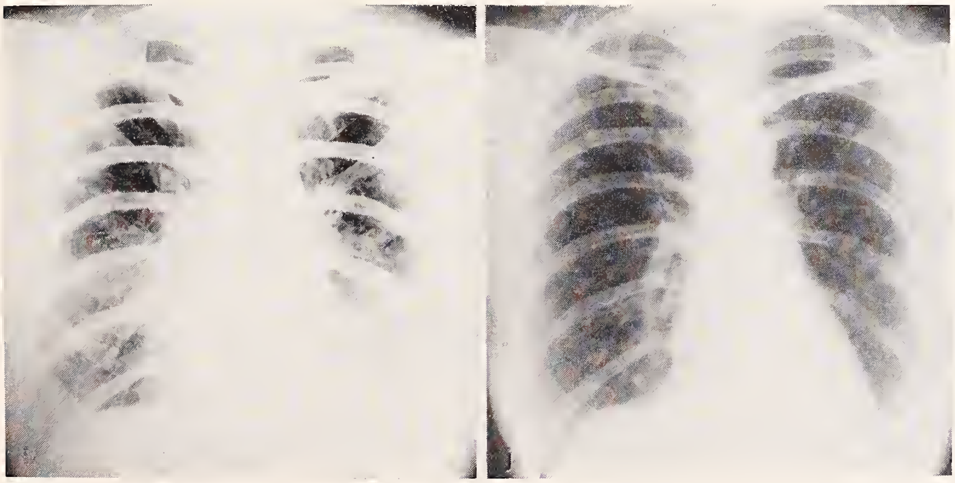

Fig. 1. (i) Case 5, M. H., February 25, 1954, shows bilateral linear and soft nodular densities with marked increased size of hilar shadows.

(j) Case 5, M. H., May 29, 1954, five years after illness, asymptomatic. Bilateral nodular parenchymal calcifications.

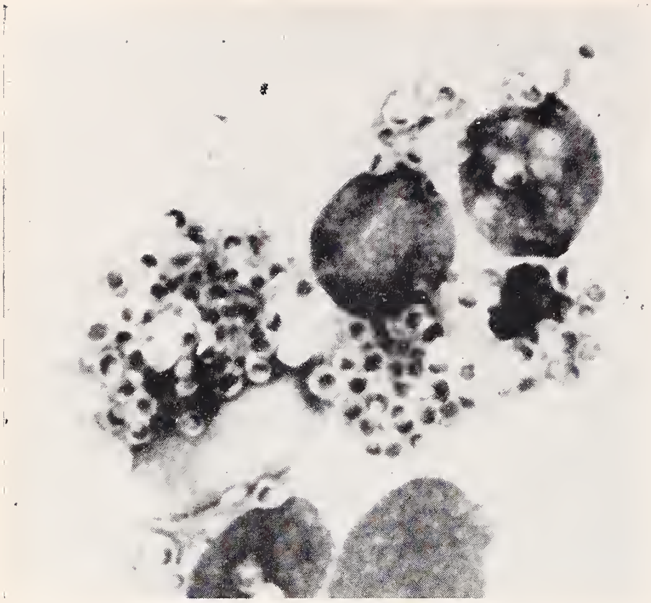

Fig. 2. Sternal bone marrow smear from D. H. (Case 1) taken eight days after onset of illness, showing typical yeast phase *H. capsulatum* within macrophages. Wright-Giemsa stain, X 1200.

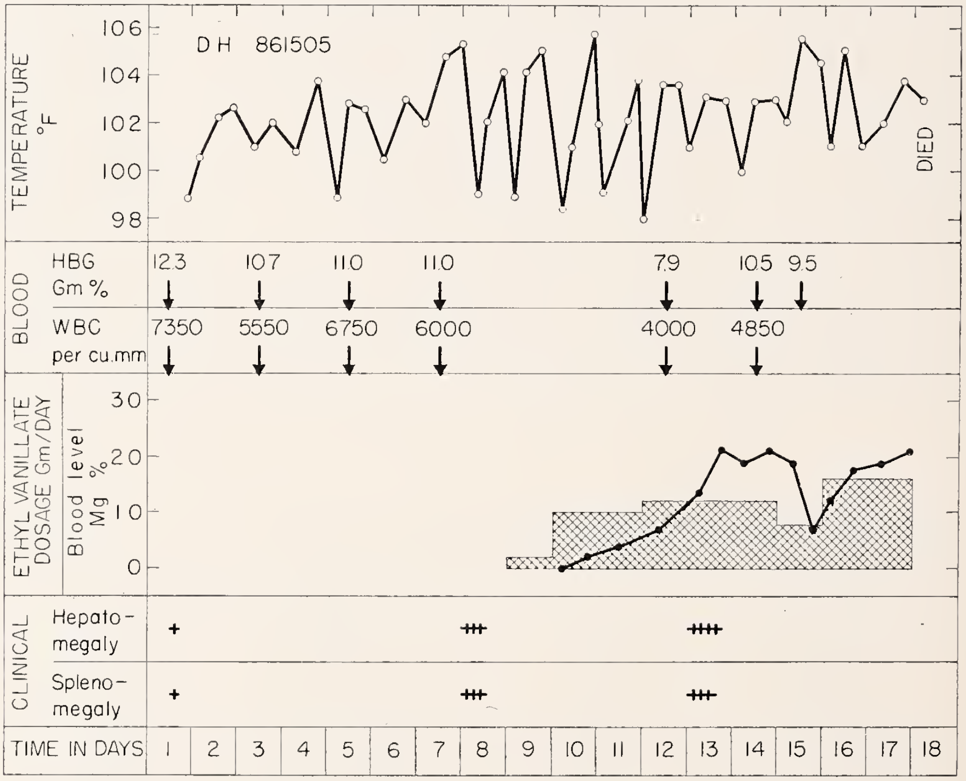

Fig. 3. Chart summarizing various clinical and laboratory features of the illness of D. H. (Case 1).

Case 2.—(A. H.) The two-year-old male sibling of D. H. was well until June 4, 1953, when he developed fever, lethargy, cough, and rapid respirations. He was hospitalized one week later, on June 11, 1953, because of persistence of these symptoms, and failure to respond to antibiotic therapy. At that time physical examination was negative, the white blood count was 11,000 per cu. mm. with 39 per cent neutrophils, 48 per cent lymphocytes, and 3 per cent eosinophils. Roentgenographic examination of the chest (Fig. 1c) revealed infiltrations in the left upper chest with some enlargement of hilar lymph nodes bilaterally. During hospitalization he continued to run low grade fever with no response to the administration of antibiotics. The infiltrations seen in the chest roentgenograms increased during the first two weeks of illness, then gradually disappeared over a three-month period (Fig. 1d). An erythema multiform type rash appeared approximately thirty days after onset and lasted two days. By the time D. H. was admitted to the University Hospitals this patient was clinically well, having no further cough, fever, or other symptoms. At that time a histoplasmin skin test was performed with a strongly positive result. Complement-fixing antibodies were present in the serum in a 1-32 titer. Bone marrow and sputum smears and cultures were negative for *Histoplasma capsulatum.*

Case 3.—(C. H.) The twenty-eight-year-old father of D. H. was ill four and one half years earlier. On February 22, 1949, he developed fever, chills, headache, cough and profound prostration. Because of these symptoms he was hospitalized. The white blood count was 12,150 with 53 per cent neutrophils, 43 per cent lymphocytes, 1 per cent monocytes, 3 per cent eosinophils. Chest x-ray (Fig. 1e) revealed soft nodular densities in both lung fields with increased size of the hilar shadows. His temperature during six weeks of hospitalization never exceeded 100.6 degrees F.; however, he had fre-

TABLE I. MAPLE LAKE OUTBREAK, 1949-1953. SUMMARY OF CLINICAL DATA

Initials	Age	Sex	Date of Onset	Symptoms	Duration	Calcification on Chest X-ray	Histoplasmin Skin Test	Complement Fixing Test Titer
DH	4	M	Aug. 22, 1953	Fever, sweats, weight loss, hepatosplenomegaly, positive bone marrow. Died.	4 weeks	—	—	1-80
AH	2½	M	June 4, 1953	Cough, fever, malaise, erythema multiforme, negative bone marrow	8 weeks	+	+	1-32
LH	27	F	Feb. 22, 1949	Cough, fever, chills, malaise, severely ill.	8 weeks	+	+	1-8
CH	28	M	Feb. 23, 1949	Cough, fever, chills, chest pain, prostration, severely ill.	6 weeks	+	+	neg
MH	55	F	Feb. 18, 1949	Fever, chills, nausea, severely ill.	4-5 wks.	+	+	neg
TH	58	M	Feb. 18, 1949	Cough, fever, malaise, mild illness.	4-6 wks.	—	+	1-8
RH	24	M	Feb., 1949	Cough, fever, malaise, mild illness	1-2 wks.	+	+	neg
BH	55	M	Feb. 23, 1949	Fever, malaise, cough	2 weeks	+	+	neg

quent chills, and sustained a 35-pound weight loss. Approximately two months were required for recovery of vigor after this disease. In 1953 at the time of D. H.'s illness he was found to have a strongly positive histoplasmin skin test, hilar and parenchymal calcifications in the chest (Fig. 1f) but a negative complement-fixing antibody test.

Case 4.—(L. H.) This twenty-seven-year-old woman, mother of D. H., developed fever, chills, cough, dyspnea, and chest pain on February 22, 1949. The severity of her illness was such as to require two weeks of hospitalization. X-ray examination of the chest at that time revealed numerous linear densities in both lung fields with small, soft, nodular densities in the lung parenchyma bilaterally (Fig. 1g). There were no changes in hilar shadows and the appearance by x-ray suggested the diagnosis of bilateral atypical pneumonia at the time. White blood count was 5,300 with 67 per cent neutrophils, 30 per cent lymphocytes, 2 per cent monocytes and 1 per cent eosinophils. Improvement was very gradual over an eight-week period. However, complete recovery was very slow over the following eight-month period. It is of unusual interest that she was pregnant with D. H. at the time of her illness; although the possibility may be raised that this circumstance played a role in the subsequent illness of D. H., it is purely speculative at the present time. In September, 1951, a routine prenatal x-ray examination of the chest showed multiple nodular calcifications. Because of the appearance of these multiple calcifications, a histoplasmin skin test was performed. This was strongly positive. In October, 1953, complement-fixing antibodies for *Histoplasma capsulatum* were present in a titer of 1-8. A recent chest x-ray showed bilateral nodular calcifications (Fig. 1h). At the present time she is in good health except for occasional chest pain.

Case 5.—(M. H.) The paternal grandmother of D. H., aged fifty-five, became ill on February 18, 1949, with fever, chills, nausea, and severe cough lasting approximately three weeks. At this time she was hospitalized where infiltrations of the chest were demonstrated by chest x-ray (Fig. 1g). She too failed to respond to antibiotic therapy. There was a gradual reduction in fever from a high of 102° F. over a period of two weeks when it became normal. White blood cell count was 10,700 per cu. mm. with 55 per cent neutrophils, 43 per cent lymphocytes and 2 per cent monocytes. The total duration of illness was approximately five weeks. X-ray of the chest in 1954 revealed multiple calcified nodules throughout the chest (Fig. 1h). A histoplasmin skin test was strongly positive and complement-fixing antibodies were absent from the serum in October, 1953.

Case 6.—(T. H.) The paternal grandfather of D. H., aged fifty-eight, became ill in February, 1949, on approximately the same day as M. H., with fever, cough, and malaise. The symptoms were never severe, but lasted four to six weeks. No x-ray of the chest was taken at that time, and hospitalization was deemed unnecessary. The x-ray of the chest at the present time reveals no calcification. Skin test for histoplasmin is strongly positive. In a serum specimen taken in October, 1953, complement-fixing antibody was present in a dilution of 1-8.

Case 7.—(R. H.) An uncle of D. H., aged twenty-four, was ill during February, 1949, within a few days of the other members of the family, although the exact date was not recalled. His illness was mild, consisting of fever, malaise, and severe cough lasting approximately two weeks. No chest x-rays or other studies were performed at that time. At the present time he is well, has a strongly positive skin test and, except for some hilar calcifications, a negative x-ray. Complement-fixing antibody was absent in an October, 1953, specimen of serum.

Case 8.—(B. H.) A paternal great uncle of D. H. aged fifty-five, became ill on February 23, 1949, with a non-specific febrile respiratory illness consisting of cough and malaise. No hospitalization was required; the duration of illness was approximately two weeks. At the present time he is clinically well, has a strongly positive skin test; roentgenograms of the chest show hilar calcification.

The symptoms, clinical course, and laboratory findings in each of the patients described above are summarized in Table I. Roentgenograms of the chest taken at various times during the course of illness in each of the patients are illustrated in Figure 1. From Table I it may be seen that histoplasmosis in the H. family occurred as two distinct groups of cases, the first occurring in February, 1949, and the second during the summer of 1953.

Evidence that the illness of the family in 1949 was symptomatic pulmonary histoplasmosis, is principally inferential. The microorganism was not isolated from any of the patients, and no changes in antibody titers were detected which might provide more direct evidence that the 1949 illness was due to *Histoplasma capsulatum*. Nevertheless, the presumptive evidence that the 1949 outbreak was indeed pulmonary histoplasmosis is quite impressive. Almost simultaneous onset of illness in each member of the family suggests that exposure was simultaneous and that the etiology of their infection was similar. This occurred in the epidemic outbreaks of histoplasmosis which have been studied elsewhere.[3,5,6] Serial changes in the chest x-ray which are available for C. H., L. H. and M. H., show evolution of a pulmonary lesion from soft, parenchymal infiltrations to the miliary nodular calcifications which characterize their latest chest x-rays, and which is considered to be typical, if not diagnostic, of previous infection with *Histoplasma capsulatum*. In addition two of the persons ill in 1949 still have low complement-fixing antibody titers. At the present time all have positive histoplasmin skin tests. Of three persons for whom serial chest roentgenograms are not available, two have pulmonary calcification at present.

Direct evidence of the etiology of infection in one of the boys is available in the form of culture of the microorganism from bone marrow and blood. Evidence, while presumptive, is good that the other boy had pulmonary histoplasmosis. Although no organism was isolated, a high complement-fixing antibody titer, a positive skin test, and a serial x-ray change resulting in calcification over a year's period provide evidence of the etiology of his pulmonary infection.

All of the persons involved in the outbreak in 1949 have lived on or in the vicinity of the H. family farm. Those who did not live on the farm itself worked frequently in and around the house, barn, or fields. No single specific episode of exposure at which all of the persons who became ill in 1949 could be recalled. The short interval within which symptoms began in all those involved suggests, however, that they were exposed simultaneously to a source of spores.

It was learned that the H. family had moved on this farm approximately two months prior to the onset of their illness. Each had participated extensively in the cleaning and renovation of the farm buildings and residence house during that period of time. It is recalled by all persons involved that upon many occasions during the cleaning process, clouds of dust were raised in various rooms of the house. No contact with areas in which pigeons or other fowl had been roosting is recalled. As it was winter and there were 2 to 3 feet of snow on the ground, the family infrequently ventured forth from the house as a group. This tends to implicate the house itself as the source of infectious spores. Attempts thus far to isolate *Histoplasma capsulatum* from many samples of dust, soil, and other materials collected around the farm area during October, 1953, have been unsuccessful. Further attempts to isolate the fungus are underway.

The illness of the two children in the family can be less definitely ascribed to a point source of spores. Because of the vague onset of symptoms in the four-year-old boy, no probable time of exposure could be established. For the younger child, the specific date of onset was known. The only known event which might have provided an occasion for massive exposure to infectious spores occurred approximately two weeks prior to onset of illness in the younger boy. At that time a large amount of old hay and straw fell on both of the boys while they were in the main barn area. They were covered with the dust which fell with the straw. Culture of dust from this area six months later failed to reveal infectious spores, however.

From data available in the literature[8,9] it might be assumed that the incidence of histoplasmin reactors in this part of Minnesota would be very low, perhaps 1 to 5 per cent. In order to make a preliminary determination of the incidence of skin reactivity to the histoplasmin in the immediate area of the farm, a survey was conducted on October 3, 1953.*

For this survey a group of approximately 100 volunteers gathered on the farm. Injections of 0.1 ml. histoplasmin (2-1000 dilution of lot H-KC-5) were made intradermally in the forearm.

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*Dr. M. L. Furcolow, Dr. Phillip Danufsky, Dr. Raymond Fagan, and Mr. David Sachs of the U.S.P.H.S. Kansas City Field Station, and Dr. Dean S. Fleming of the Communicable Disease Division, Minnesota Department of Health, collaborated in this phase of the investigation.

The reactions were read forty-eight hours after application, an area of induration 5 mm. in transverse diameter or greater being interpreted as a positive test. Blood specimens were taken from the reactors for precipitin and complement-fixing antibody titers. As the population tested consisted of volunteers and not a random sample of the population, the results provided only a preliminary indication of the incidence of histoplasmin sensitivity in this area. More extensive studies of skin reactivity to histoplasmin and tuberculin in Wright County will be reported subsequently.[11]

Table II gives the results of this preliminary survey of volunteers. The data is arranged in two categories; those persons tested who have had previous contact with the site of the outbreak, and those who had never had contact with the site. The data indicate that 10.8 per cent of those never on the premises before the time of the survey were reactors, whereas 50 per cent of those who had visited or worked there were reactors; 26.3 per cent of the entire seventy-six persons from whom information as to previous contact with the farm had been obtained were positive reactors. None of the positive reactors had significant complement-fixing antibody titers.

In addition, a number of animals on the farm were tested for histoplasmin sensitivity in accordance with the method of Menges.[7] Of thirty-three cows tested, five were found to be reactors. Of three dogs tested, one was found to be positive, and of three rabbits tested, none was positive. The precipitin test in two of the cows was positive in a 1-5 and 1-10 dilution, respectively.

This series of cases illustrates the broad spectrum of clinical manifestations which may result from infection by *H. capsulatum.* Case 1, D. H., illustrates the disseminated systemic, usually fatal type of the disease in which the yeast form of the fungus multiplies rapidly within the macrophages of the reticuloendothelial system. The fungus spreads throughout the body, producing enlargement of the liver, spleen, lymph nodes, high spiking fever, dissemination through the blood and eventually death. This form of the disease, in contrast to past notions, appears to be the rarest manifestation of infection and occurs principally at the extremes of life or in debilitated patients. The pulmonary form of the disease is well illustrated by Cases 2, 3, 4, 5, and 6. In these patients there was abrupt onset of fever, chills, cough, and other symptoms of pulmonary involvement of varying severity. In each there was no response to antibiotic therapy, and the illness lasted for a prolonged period of time, clinically resembling atypical pneumonia. Less severe symptomatic forms of pulmonary involvement is illustrated by Cases 7 and 8. Their infections would have passed unnoticed had not the other members of the family been so severely involved. Their infection did, however, result in sensitivity to the fungus antigen and pulmonary calcification. Other types of histoplasmosis which have been reported, including local forms[14] and chronic pulmonary histoplasmosis resembling tuberculosis,[4] were not seen in the epidemic.

TABLE II. RELATIONSHIP BETWEEN EXPOSURE TO SITE OF MAPLE LAKE OUTBREAK AND SKIN SENSITIVITY TO HISTOPLASMIN

Exposure History	Histoplasmin Skin Test Result		
	Number Tested	Number Positive	Percent Positive
Never on premises at site of outbreak	46	5	10.8
Visited or worked on premises	30	15	50.0
Total	76	20	26.3

This outbreak emphasizes the fact that histoplasmosis occurs in Minnesota. One previous outbreak has been reported by Ruteledge[10] and later by Furcolow.[3] This epidemic occurred in Detroit Lakes in 1948, and was similar in many ways to that reported here. In the adult members of a family, simultaneous onset of a diffuse pulmonary disease with slow recovery occurred. Spores were isolated from beneath the kitchen floor. The occurrence of these two epidemics should alert the Minnesota physician to the possibility that some of the puzzling unexplained types of pneumonitis occasionally encountered in his practice might be due to *H. capsulatum* infection. It also indicates an urgent need for more incisive information than is currently available on the prevalence of the fungus, the disease, and skin test reactors in the state.

Summary

The clinical and epidemiological aspects of an outbreak of histoplasmosis occurring in rural Minnesota have been described. Six adults and two children manifested a broad range of clinical

(Continued on Page 582)

"Ideal" Socialized Medicine

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BECAUSE the general trend of socialized medicine is insidious and because so many proponents of such a scheme for the United States frequently cite the Scandinavian countries and Great Britain as examples of apparent success, it has become evident that even in the light of a current dormancy in legislative attempt, the medical profession as a whole must be informed of the salient features of these various programs. For this reason I have summarized the results of my recent observations for your consideration and cogitation. In 1952 I was fortunate to be in the position of practicing medicine in Great Britain under the panel system, a report of which was made in 1953.[1,2] This experience gave me the opportunity of learning the hidden and often serious problems to look for in terms of care of the patient, standards of practice, encroachment upon freedom, and preparation for the future, as in training and research. With this background and with numerous relationships having been established, I returned in 1954 to Great Britain where a progress report was undertaken. Following this several weeks were spent in Norway, Sweden, and Denmark. The general approach was to interview the following groups: (1) organized medicine in the form of the medical association, (2) government medicine which included the director of the program and his associates, (3) the practicing general physician in both urban and rural areas, (4) the specialist both in the hospital and in the private practice, (5) the medical student and graduate student, (6) the beginning practitioner, (7) the patient, and finally (8) the press. It was my feeling that each had an "axe to grind" and that only in talking with all of them could I get a relatively true picture without actually participating in the practice. In addition, by being alone with the various individuals and by proper rapport (including judicious use of the "juice of the grape") I found them more ready to discuss freely and to criticize constructively the programs. The survey was conducted with co-operation from the American and the World Medical Associations, together with the Norwegian, Swedish and Danish medical associations and government agencies.

With this as a background as to the experience and mode of approach established for this study, I should like further to point out briefly some other factors which contribute markedly to the reason for such programs having evolved. Historically one must remember that the United States grew from infancy as a democracy. Conversely, the European countries have struggled from the dominance of the feudal system and it is fairly evident that changing an adult is more difficult than training a child. Thus different measures were necessary. As a result, though existing as democracies, the respective countries themselves are different. Tradition plays an extremely important role also—one which is difficult for us to understand in light of the fact that a change from tradition could produce more efficient effect in many instances. The population and size of the countries together with communications, transportation and finances also point out the fact that in *no* way can any program be compared with nor suggested for the United States. Whereas the United States has forty-eight states and 160 million people, Great Britain has a population of about 50 million and is the size of Minnesota; Norway has 3.3 million and compares in square miles with Arizona; Sweden's size is analogous to that of California and there are 7.1 million people; and Denmark is the size of Maryland with a population of 4.3 million. Their problems, then, are those of a single state. There are about 35,000 physicians in Great Britain, 3,500 in Norway, 5,500 in Sweden, and 4,300 in Denmark. In each instance there is no more desirable patient-doctor ratio than in the United States and in some instances, less.

Another significant fact to remember is that in

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each of the countries described the general practitioner is *not* permitted to care for his patients in the hospital but must refer them to the specialist who then takes over the care of the patient and returns him to the general physician upon discharge. This of course disrupts the continuity of care and does not encourage the stimulation of the general physician.

Two arguments noted were that the general men in these countries did not miss the continuity and that the system in the United States resulted in the general physician overstepping his training or ability. In refutation I noted that younger men in the countries were against this old tradition; that Dr. Charles Fleming, the Principal Medical Officer for Scotland, wrote an extensive treatise regarding the need for that change and others corresponding to United States practices;[3] and finally, that in both instances it depends upon the integrity of the man and his recognition of his own limitations.

Doctor Collis of Dublin wrote similarly of this need in his citing the United States in encouraging ideal medical education and practices.[4] Insofar as medical training and postgraduate training are concerned, one can say generally that the students in each country are of high calibre and well trained. The medical care, training, research, and programs in the teaching hospitals were comparable to the United States; however, in general, the equipment and facilities, though adequate, were not. This is undoubtedly temporary as a result of the war and in certain instances they were as good. There are very few private hospitals (usually religious or Red Cross), and in Great Britain a good number of nursing homes are used by the general practitioner and his consulting specialist. Surgery and obstetrics are done there, but very few, if any, laboratory procedures. Thus the hospitals are government owned and administered. Therefore you can see that there are two basic segments to medicine in Europe, the general physician in home care without the use of a hospital and the specialist in hospital care. The effect this has played in the socialization schemes will be noted.

In order to give the full picture in detail, this paper would have to become a book; therefore only the significant points will be covered and you must keep in mind that there are always exceptions which, for lack of space, may be omitted. One decided general impression is that there is a great deal of good along with the bad and potential dangers in each of the schemes, but that one must see the needs and application to the country involved to understand its evolution. In no instance can any phase of any country be compared with the United States.

Great Britain

In Great Britain socialized (or nationalized, as they prefer to call it) medicine really began in 1911 as the panel system, whereby the government began paying physicians for caring for some patients. For many years this included only those who earned less than 400 pounds a year. (This was equivalent to $2,000 but possessed a greater buying power.) The program was carefully thought out and further extensions were planned. The Conservative party, not the Labor party as we have believed, was the originator of the program and was in the process of consultation (admittedly slow) with the Britain Medical Association, when the Labor party through its Bevan made the program a political football and, with votes in mind rather than British health, thrust it through. This was greatly enhanced by the poor co-operation of the voting conservative population. The cost was estimated in 1948, when the plan was begun, at 510 million dollars. The mechanism was to be simple and it is, for the basic plan had been in effect for thirty-seven years. However, the cost in 1951 was 1.3 billion dollars and about 1.5 billion dollars for 1952. The Conservative government has set a 1.2 billion dollar limit per year thereafter. However, one can see that some of the problems are being met, but slowly. Thus, as I have been told, "when we get the rough edges off and are used to the scheme, it will not be so bad."

The plan is compulsory for all and is directly under the control of the government with local subdivisions. Physicians are *not* in control. The plan is payed for either directly to the government or by the usual deduction if the person is on a payroll. It is included in the fee for all social benefits which, if calculated by population, will not in itself run the program in the black. The panel system involves the general practitioner. The general physicians are not centralized, but rather tend to care for the patients in their own area. Thus, it was simple to continue this tradition, and in becoming a panel member, the patient merely signs up for a year with the doctor of his choice. The number of patients is limited to 3,500 per doctor (having been cut from 4,000) and includes children. The doctor cannot see any more than this according to the system. Thus there is some restriction to patient choice; however, could you as a general physician care for 3,500 people as often as they desire in a year? With better than 90 per cent of Britain now gainfully employed, some patients, who formerly had time to wait their turn, now will pay to be seen. With this increase in private practice the doctor is supposed not to show discrimination, but as one man said "human nature frequently prevails." The adjustments made in the past years include that of an increase in fee and decrease (by 500) in the number patients per physician. Currently the pay is $2.38 per head per year for the first five hundred; $3.78 for the next one thousand and $2.38 for the remaining two thousand (total about $9,700). If a doctor has a certain number of patients, he can now obtain through the Health Service an assistant who receives a straight salary of about $2,000.

Some of the original problems appear to be combated on the surface, but in place of them there are others. With the increase in fee and decrease in patient load to the general practitioner, the specialist group was up in arms for an increase. The training of students and paying of salaries of about $5,000 to what corresponds to our senior resident has increased the number of specialists to the point where there are not enough hospital positions and they will not be accepted in general practice. The increase in employment has worked to the advantage of private paying practice as noted. With the bureaucracy of the Health Service, as I was told by a physician high in government service, several major problems have resulted: (1) Because of the disadvantage in being a general physician, most students intend to specialize (this I noted personally while a member of the University of Edinburgh faculty). (2) The general physician now has little opportunity to specialize, for he finds it impossible to get a position in training. (3) The general physician may *not* change jobs as he desires. He must apply to the Executive Council (not doctors) of a given community to set up practice there. This he cannot do unless the council feels the area is underdoctored. Thus the freedom of practice location is no longer free! (4) In addition, it is noted that there are fewer general physicians striving for advance degrees—a fact particularly true in the past. A gradual decline of general practice was predicted, with the one redeeming note being that an Academy of General Practice has been founded.

There are some general physicians, particularly in industrial areas, who enjoy the Health Service remuneration, for they did not have it in the past. Most with whom I talked at length felt they had compromised themselves.

The specialist is not paid in the same manner as the general physician, but rather generally has an appointment to one of the hospitals where he is paid according to the number of sessions he works—a total of eleven sessions are possible at $560 each. In addition he may make up to fifty domiciliary calls at $12 each (a total of about $6,800). However he may and does see private patients in consultation. In addition, 35 per cent of the specialists receive "merit awards" each year ranging from $1,400 to $7,000. Reputedly there is a fair manner of selection, but there is also the human equation!

From another authority I learned that the established specialists were faring remarkably, but that the newer men were receiving positions on a straight salary only and that the intent of the government circles is to place them all under civil service, which then would negate what few bargaining rights are left. Medical facilities, equipment and the like are improving, but this is due more to the relaxing of government control on rationing material and construction with resultant new building.

The specialists' problems, besides those noted above, include the fact that there are too many for the number of positions. As a result, in Great Britain as well as in the Scandinavian countries, the most junior staff man is usually in his forties. Two other examples of bureaucracy are these: in attempting to get a man for a position in a hospital, the chief may not hire whom he desires, but must show first the need, and then take it through the chain of command of hospital administrator, executive council, district council, regional council and finally, headquarters. Another is that should a physician obtain funds for a research project and then note that it will not succeed, he cannot divert the remainder to another phase of research. He must return it. You can guess what occurs.

One must remember that in previous years hospital appointments were without salary, but rather for the prestige (and referrals). Now the specialist is being paid for what had been done gratis.

Hospitals are now getting caught up with the old backlog of elective procedures after almost six years of extended waiting lists. This is true because patients are over the novelty stage and because so many use private insurance schemes to have more personal care in private rooms at Nursing Homes. In addition a very few of the better nursing homes (originally left out of the plan) are being taken in. Moreover, the Royal College laboratories for out-patient or nursing home use, which were not supported by the government, now are back in use. This makes far more desirable the care in the home or nursing home (although the average number of laboratory procedures done by physicians in Scotland is only twenty-seven in a year).

The patient pays for nothing in the hospital including drugs, laboratory, et cetera, and is discharged to the care of his personal physician. Dentistry is free but restricted—it being an advantage financially to pull all teeth and make dentures. Prostheses are free if necessary for livelihood, the wig and toupee now being charged for.

Compensation to the worker is separate from the health service. Public health measures, particularly tuberculosis control, have improved in the past two years, but are far from adequate. Only 7 per cent of expenditure goes to these public health needs. Another problem has arisen in the form of free legal counsel and as a result the Health Service is being sued along with the physician (since he is employed)—even for actions that occurred years earlier. This results in further expenditures, if only for defense.

Presently a study commission is examining the problems and needs of the Health Service and the country. It does not have physicians in its number!

There are many opinions and reasons given by the numerous proponents, opponents, and acceptors of the plan in each of the categories of patient, doctor, government and press, which I shall not elaborate upon. However, there is much dissatisfaction, and this, even in the face of satisfaction to many, indicates that the plan still has not improved adequately even to the British.

Norway

In Norway the initial plan was begun in 1912. This was altered through the years until 1930, when the present general program was brought into effect. This of course was disrupted because of the German occupation. Currently the program is compulsory for the employed. The others may use it electively, although in years gone by there was a ceiling to the income a person could have beyond which he was not permitted to enter into the plan. Now it is available to all. About 80 per cent participate in the insurance scheme.

The plan consists of a number of private insurance companies throughout the country which insure the persons themselves; however, these insurance companies are controlled by the government in that all the policy planning for each of the companies is established through a committee within the government. The health plan is supplanted by taxation for some aspects which will be mentioned later. The manner in which the insurance is paid for is as follows: the employes pay 6/11th of the entire cost; the employer pays 2/11th; the community government, whether it be city or county, pays 2/11th; and the federal government pays 1/11th. This is deducted if the person is an employe; otherwise the entire amount is paid directly to the insurance company by the person involved. Therefore, in effect, he personally pays more. The plan costs approximately $7.00 per year (not to be compared in purchasing power with the United States). The patient has a free choice of physicians.

There are some other factors, however. Approximately 400 districts are located within the boundaries of Norway. These are similar to what we would call a public health district and within this district there will be at least one physician, who acts as a public health officer. The more sparsely populated districts will have just one man, whereas in the more populated districts there will be more than one. In the sparsely populated districts the man, who is the public health officer, is responsible for the care of the needy and indigent but also may have general practice; conversely his position is full time in a municipal area. He is given a basic salary for this and therefore is selected by the government. However, another physician may come into this district and practice without any government salary but merely by being paid by the insurance company or by the patient himself. If a patient desires to see a specialist, he must consult a general practitioner first, who in turn will refer him to the proper specialist. There is a limit to the number of the referrals the general physician may make. As was mentioned, the district physician receives a basic salary. In order to attract some of the men to the rural areas a higher basic salary may be paid in the relatively more undesirable districts. As the officer grows older he may be advanced to the better districts if he so desires. Many prefer to continue in the challenge of the fjords and difficult terrain.

In private practice, there is a charge with a recommended scale. The doctor will see a patient, and then according to the scale, which has been prepared by both the government and the Norwegian Medical Association, he will charge a given amount for a procedure. The patient then pays this particular bill and takes the receipt to the insurance company where he will receive a refund for a portion of what he had been charged. This runs to approximately 75 per cent of the minimum charge. A wealthy person (of which there are few) can be charged more, but is refunded only the percentage of the recommended scale. The charge varies with the procedure and varies in general practice and in the specialty. The specialist will follow the same routine and will charge according to the recommended scale, which is similar to our Blue Shield, but his charges will be higher even if it is the same procedure. This program involves the specialist who is in private practice; however, most specialists attempt to have full-time hospital appointments. Usually they are on a basic hospital salary and are permitted to have approximately six hours a week private practice for which they are paid as noted above.

The patient's choice of the doctor is free and doctor's choice of the patient is free. The patient may change doctors any time just as in the United States. If a patient requires hospitalization, he is then placed in one of the public hospitals owned by the state. He is given full and complete care without charge. The hospitals are all of the ward type. Part of this is paid for by the insurance company; however, the amount it costs the hospital per patient is considerably more, so the remainder is paid for from the general taxation funds. Should the patient desire to go to one of the few private hospitals, he will have the same amount paid by the insurance company (about $3.50 a day), but there is no taxation to make up for the remainder as in the public hospitals. Therefore, if the cost is eight or nine dollars a day, as it is in many instances, the patient must make up the difference. If the patient can prove that there was no bed available in a public hospital, then the private hospital charge will be paid in full also by the government.

All laboratory procedures are without charge in the hospital. Outside of that they are not all covered, although there are some special procedures which are partially paid for, such as the x-ray. There is no charge whatsoever for so-called "life saving" drugs as the antibiotics, insulin and any of the drugs which are necessary to maintain one's life. The remainder of them are paid for by the patient. Dentistry is only partially covered. This involves only some reparative procedures and the dental care therefore is lacking to a point. Certain of the prosthesis are paid for, such as hearing aids. This depends upon whether they are necessary for livelihood. During illness the patient will get a percentage of his salary in compensation. This depends upon the type of insurance he has taken out; therefore he must pay more in order to get greater compensation, it being a sliding fee.

There is a good program of public health in Norway. There has been concerted effort to increase the public health measures, and as can be seen by using the district health officers, there is a good grass roots approach. In addition, the state pays separately for the survey and care needed in the control of tuberculosis and of mental disease. Persons requiring hospitalization for these diseases are cared for entirely by the state from a different fund of taxation.

Insofar as our general observations were concerned, the care is relatively good. Certainly the program prevents the catastrophe of losing of all of one's funds; however, there are some problems. The general practitioner does not have available to him hospital or laboratory facilities, so that the care under a general physician is restricted even though he is doing a very good job under the circumstances. If these were to be increased, costs would rise markedly. The doctor status is restricted to a certain point by the government control but it is relatively free. With the centralization of authority, a potential danger of poor administration exists, though excellent now. The fees are established for the doctor, which is a potential problem. They are quite fair, however, compared to the other charges throughout the country for other services. The doctors have no objection in that regard; they feel that their particular lot is a good one. However, when one looks at the total comparative picture of Norwegian socialization, one can understand why the doctor feels this way. Currently there is freedom insofar as the total program is concerned. The total effect on medical care has been further spread of medical care including the rural areas. It has helped increase the specialization care but the general practitioner should be given more opportunity.

There are some miscellaneous factors which I think are extremely important to consider. First of all, there is a relative dictatorship by the director's office of the Health Service. He has drug control under his direction. For the sake of finances it has been established that the best drug for the least money is the one which is used. However, there will not be two drugs of the same type made by two different companies. Thus competition is gone. This currently is not a major problem except that it suggests what could happen if a man who did not have the country's welfare at heart were in charge. In addition, the director's office selects three people, from whom one is chosen for any vacancy that occurs in government medicine. Here again a threat, because of the centralization, could arise. There is none at the moment, but the possibility is real. Cheating on time off seems to be a problem which has arisen. In this the doctor is inclined to give a patient an extra day or two off in order to keep him happy. This keeps him as a patient; otherwise he might go to another doctor. This doesn't happen too often but occurs sufficiently frequently for me to have heard it from many men.

There is a shortage of training in Norway for some of the specialties, particularly ophthalmology, by reason of the fact that there are only two medical schools in Norway, one in Bergen, established since the war, and one in Oslo. In Oslo there is an emergency center, so that there is a physician ready and available twenty-four hours a day. The one problem that has arisen from this is that many people who are not true emergencies come to the emergency center due to the fact that they can get a specialist more readily without a referral.

The attitude toward the plan by the patient is good; the patients really seem to enjoy it; they think it is a good plan and hold the doctor in high esteem. The government of course believes it is good but they would like to set up a basic salary for *all physicians.* This was very soundly trounced by the Norwegian Medical Association, however, and for the moment has been set aside. The physicians feel that they have good freedom in their care of patients and look to the plan as being adequate, but with the potential dangers mentioned; the press has nothing to pick at in general. They feel that the plan is very good. In summary, one would feel that this, in so far as a socialized plan is concerned, shows considerable freedom and more possibilities of leverage, and that the only main complaint by the physicians is the centralization within the government.

Sweden

The Swedish plan had a period of indoctrination from the turn of the century until 1930, when a rather inclusive program was incorporated. This was rather close to that of Norway in most respects. There were changes involving drugs and compensations introduced in 1946, but the total compulsory plan was not initiated until January 1, 1955. Thus the effect of this recent innovation will not be noted for some time. Sweden has distinct economic advantages in the Scandinavian sphere through its historic relationships and its attempted neutrality in the wars. These have contributed considerably toward its having better facilities in general than the other countries. Much more could be said in this regard. In summarizing the main features in this paper, I shall have to cover the experience noted in the past twenty-four years of the plan and merely comment on the new compulsory factor. There are a few minor changes, but in reality the inclusion of the entire population is the main one. This was done primarily to get a greater assured total operating budget, but the greater centralization does present possible further dangers. With the new feature the total freedom, of course, is further impaired. It is interesting in this regard to note that the Scandinavian countries have combined to publish a book entitled "Freedom and Welfare." Some criticisms by the medical association, besides those noted above include (1) the compensation features in which some patients can receive more money by not working; (2) the poor timing of a total plan, for there are too few doctors and too few beds, which results in too many patients per doctor per day; (3) the fact that it costs less for the patient in the hospital and therefore there are too many admitted.

Before the new ruling about 75 per cent of the country was covered by health insurance. This was (and still is) operated under the same type of plan as that of Norway in which private insurance companies are controlled by a committee within the government. The approximate breakdown of cost is 44 per cent paid by the employe, 27 per cent by the employer and 29 per cent by the local government. The cost per year is approximately $6.00. This again cannot be compared with the United States, for the greater buying power, lower salaries, et cetera, make it impossible. Again one must note that just as in Norway this insurance pays only for a part of patient care, that the drugs, prosthesis, major portion of individual hospital care, tuberculosis hospitals, mental hospitals, et cetera, are all paid for by the government from taxation. Thus the real cost to the patient is hidden.

The patient may select whatever doctor he chooses, but in contrast to Norway, he may go directly to a specialist. In Stockholm this has resulted in better than 95 per cent of the doctors being specialists or partial specialists. Great Britain physicians in observing this have written a word of warning to their general practitioners.[6] This trend has worried the Swedish Medical Association. The patient upon being billed for service by either a general practitioner or specialist then pays the sum and brings the receipt to the insurance company where he is refunded a portion of the bill (as in Norway about 75 per cent of the recommended scale). This scale is established by the government in conjunction with suggestion by the Swedish Medical Association.

In addition, the district health officers (there are 600 districts in Sweden) are employed by the government as in Norway and may have private practice in the rural districts. These men must care for the indigent without charge as a result of the basic salary. Government medicine is interested in increasing the number of districts, but the Swedish Medical Association believes this another centralization danger. There is good reason for fear, for although the present medical director is an excellent man, the one who preceded him was not. In fact, he was praised by the Communists and criticized by the Western bloc physicians who knew him. He has since been removed from another position in India. This has alerted the medical association to potential dangers of centralization.

In Sweden polyclinics have sprung up. These are large clinic facilities staffed by specialists on a salary. They are generally owned by the municipality. The patient may go directly to these clinics, but has no personal relationship. In addition the out-patient departments of the hospitals which are generally large (1,200-1,800 beds) will care for patients. It was most amazing to see the tremendous lines waiting, but understandable when I was informed that in one department alone two surgeons would see 200 patients per day. The morning was for men and the afternoon for women. This figures out (including forty new patients) to as many as thirty patients per hour. Need I say more?

The care of a patient requiring hospitalization is excellent and without charge. He receives drugs, laboratory work, et cetera. As in Norway also he is in wards only. The same financing of insurance plus government funds pays for it. He may be referred back to his general physician, but often returns to the specialist and out-patient department. There are few private hospitals. Those that I visited were excellent but are not covered other than by insurance—unless the patient can prove that no government hospital bed was available.

The life-saving drugs are paid for; those that are essential but not live-saving are partially covered; and the remainder are not paid for. Prostheses are covered if essential for livelihood. Dentistry of a reparative nature is the only type covered and compensation varies with the job and the insurance. Public health measures, including special funds for tuberculosis and mental hygiene care, are excellent.

General summary of effect of the plan would be that the care is good under the circumstances but that there are too many patients seen in too short a time. The intimate doctor-patient relationship is diminishing, but hospital attention and facilities are excellent. The dotcor is in high esteem and receives very adequate recompense. His freedom in private practice is essentially unimpaired, but if he is a specialist and on a staff, he is salaried. The usual patient has learned to wait in view of the superspecialization in the major centers, but a considerable number miss the personal touch. Those who have sufficient income can still get what they want, however. The government and the press currently are satisfied, but the former appears to encourage more salaried physicians. The medical association is not pleased with the compulsion, the great number of free and partially free drugs, the relatively inequitable compensation during illness in some cases, the over-reimbursement to the patient of certain procedures, the superspecialization and trend toward polyclinics, and finally the overcrowded hospitals when convalescent hospitals would help relieve the load considerably. It may be noted also that the number of students will stay high because of prestige, but that the young physician in a specialty will be a junior man for many years and on a salary.

Denmark

Perhaps the most complicated of the socialized plans in Scandinavia is that of Denmark. This stems primarily from the long history and growth of the plan, but also is due to the population distribution and geographical history. In actuality farmers began forming "Sick Clubs" in the 1850's which were set up in the manner of insurance companies. In the 1880's parliament discussed subsidization of the program to a degree, and in 1892 the first government plan was introduced. The current program was established in 1932, at which time all social laws were co-ordinated including sickness compensation. The German occupation of course interferred with the total picture of the country, but currently the scheme has two distinct modes of implementation—one type involving Copenhagen (about one quarter of the entire country's population) resembles the British scheme—the other is like that of Norway and Sweden and covers the remainder of the country. The plan is compulsory for the employed, the manner of payment being the same as in the other two Scandinavian countries. Here also the employe pays about half the cost ($9.00 a year—again not comparable, for it includes all children under fifteen years). There is a limit of income beyond which one may not enter into the government scheme. Presently about 75 per cent of the population is covered by this plan. The insurance companies are government controlled; however, there is also subsidization from taxation.

The general plan, exclusive of Copenhagen, is like the other Scandinavian schemes in that the patient has a free choice of doctor and the physician is paid on a fee for service basis. One major difference is that the doctor fills out one of the dozens of colored forms for a particular insurance company and submits them monthly for full scheduled payment. The patient must see a general physician first, thus needing a referral to consult a specialist. There is a limit to the number of referrals a general physicians can make in a year. A person covered by private insurance may go directly to the specialist but the company will pay general practitioner rates.

The country is divided into twenty-two districts where there is at least one public health officer and sometimes two to three assistants. Government medicine desires these men to be specialists in the field, but the Danish Medical Association wants them to be general physicians as most of them are. They have the duty of public health measures and care of the indigent, but also may have a private practice, thus the usual procedure is to buy one—a difficult problem for young men. The hospital system involves a central hospital in each county (250-400 beds) with a few mixed hospitals (90-100 beds). Occasionally there may be some specialties lacking in central hospitals. The mixed hospitals include medicine and surgery —with the surgeon always being the director. The laboratories of all the hospitals (and a central laboratory in Copenhagen) are open to the general physicians. All life saving drugs are paid for; those determined to be necessary or important are covered ⅔ and the remainder are paid for by the patient. All hospital expenses are paid through the Sick Club, the local government and the state. Dental care is not complete, being only reparative. Prostheses are paid for by insurance if needed, although hearing aids (e.g.) are paid for by the government.

The chief of a specialty in a county or municipal hospital may see private patients by referral as well as receive the basic salary for his hospital work. The assistants are permitted to see patients on their time off, but not at the hospital. The government has established a maternity and child care program for which the general physician receives fee for service. This includes three free obstetrical examinations preceding delivery for any pregnant woman and free pre-school and school examinations. In addition, he is paid for making out health certificates.

The Copenhagen program (includes Fredricksberg) is completely different. In this instance the Sick Clubs appoint city district physicians who then establish a panel. Other physicians are permitted to practice, but there is no source of patients—thus, in effect, all are employed by the Sick Clubs. All must be members of the Danish Medical Association. There is a free choice by the patient within the district, but this limitation plus the fact that the patient signs up for a year on the doctor's panel hinders liability. The doctor receives about $3.50 per head per year for 2,000 patients, but must care for those in the family under fifteen without charge. He may have another 500 patients, but is paid only about $.30 per head for these. The general physician does *not* make calls between 7:00 p.m. and 7:00 a.m. for there are special "night doctors" employed by the Sick Club. This group is drawn upon for any "day doctor" vacancies that may occur in a district. The newest man in a district gets what is left over from the others—in other words the "rejects." Fortunately the usual cooperation amongst patients is quite good.

The specialist has the same situation as his associate in the county hospitals except for the eye, ear, nose and throat specialists who are assigned 30,000 people (whom they may never see) at a rate of about $.15 per year per patient.

This series of apparently confused adjustments has many minor sidelights, including the fact that some physicians still collect 10 to 70 dollars in advance per year from private patients for tending their needs that year. Some comments and observations to be noted were that in the per capita plan the general physician tended to send too many to the hospital to get them out of his hair, whereas in the fee for service program he tended to see them too often. There are mechanisms of check and counter check in this regard.

The tuberculosis, mental and child and maternal care are paid from tax sources in Denmark also. There is governmental pride in the extreme narrowing of net income range within the country and some of the doctors think "it is nice that the people do not pay." Currently the people are satisfied, having known nothing else; the doctors are complacent except for a few; and the government is currently considering proposing full compulsion. A commission of twenty men (including three from the medical association and two from government medicine) is meeting in studying this possibility. In reality the medical association-government-press relationships are excellent.

As one doctor said, "We are far closer to a true communist state than the Soviets."

Summary

Summarizing the total trend of socialized medicine in Europe, one must keep in mind that the historical, geographical and traditional factors are far different from ours. In addition, the needs, habits and finances are completely at variance. Therefore one cannot begin to compare with nor apply their experience to these United States. We in effect are the equivalent of forty-eight countries from the standpoint of size, population, complexity and administration.

There was a distinct need for further medical care, otherwise the various programs would not have come into effect. The only true full blown political maneuver evidenced was that of Great Britain. But even in that instance the initial program was thirty-seven years old. It was interesting to note that similar schemes for the employe group are present in every country I visited—Holland, Switzerland, France, Italy, Greece, et cetera and that Germany (the originator) still has a program. In East Germany I found the Russian plan woefully inadequate, with physicians slipping to the West regularly. This fact, together with the very definite turn of the world to the United States for leadership in medicine, indicates that many routes are sought to attain the same level of medical care enjoyed by our country.

Thus one notes a difference in attitude and acceptance, plans and facilities of medicine in each of the countries. However, one singular fact of full freedom remains in that private insurance for private care beyond each national program is definitely utilized and in many instances has increased markedly!

It has become quite evident that free enterprise has brought better general medical care, facilities, and future to all peoples in this country than is present in any of the "ideal socialization schemes," *but* we have not a sufficiently comprehensive program to offset medical financial catastrophe

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Radical Treatment of Brain Abscess

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TREATMENT of brain abscess in the preantibiotic era was necessarily governed by the physician's inability to combat directly the most important aspect of the patient's disease, the microorganism concerned. Only the patient, through the antibacterial mechanisms of his own body, could discourage and destroy the pathogenic organism. Thus, in a very real sense, the patient had either to overcome his own disease or to die. Under these circumstances the treatment of brain abscess was one of the more dismal aspects of neurosurgery.

The accepted mode of treatment was to wait until the abscess had become chronic and then, and then only, to evacuate its contents by drainage.

If drainage was undertaken too soon, the surgeon encountered not a firm, well-defined abscess wall but an area of diffuse cerebritis and necrotic tissue. Mechanical interference in such a situation resulted only in spreading of the infection, meningitis and death. On the other hand, the increased intracranial pressure resulting from cerebral edema in the hemisphere harboring an abscess frequently resulted in prolonged suffering, loss of vision and even death before the abscess was deemed ready for drainage. The selection of the proper moment for drainage was a problem requiring the nicest clinical judgment.

A number of technical methods were devised to facilitate the drainage of brain abscesses. Dandy's air studies (pneumoencephalography and ventriculography) permitted more accurate localization of the lesion, which in turn permitted the use of minimal skull openings in approaching the abscess. The use of a large craniotomy, as might be the case when tumor rather than abcess was suspected, permitted bulging of the brain with formation of hernia cerebri and almost always ended in disaster. Neurosurgical opinion came to favor the placing of the approach to an abscess in a clean field rather than through an infected area such as the mastoid.

Most surgeons used drains of one sort and another extending from the abscess cavity to the surface. Dandy pointed out the traumatic potentialities of the draining process itself and advocated single or repeated tapping through a brain cannula as sufficient in most instances. Others (Grant), devised atraumatic methods of introducing drains. Such efforts were greatly facilitated by the suggestion of Kahn that a radiopaque material (Thorotrast) be introduced into the abscess cavity following its initial tapping. Serial x-ray studies then permitted following the lesion's course with respect both to its size and position. It was seen that even a deeply placed abscess would migrate toward an overlying defect in the skull provided a sufficiently large opening was made.

King developed a more aggressive drainage technique in which an area of cerebral tissue between the abscess and the surface was removed and the abscess itself was uncapped. The rest of the abscess cavity was then everted by the pressure of the brain behind it. This marsupializing technique did not achieve wide employment because it involved such extensive trauma to the brain (and probably also because it was so time consuming).

Finally, it may be noted that many surgeons had had the occasional experience of removing an encapsulated intracerebral lesion, presumably a tumor, only to find upon sectioning the mass that it was actually an old chronic brain abscess. Ballance, as early as 1912, suggested that this was the ideal treatment for brain abscess. Others, including Dandy, felt that the procedure was potentially too dangerous to be deliberately undertaken.

The end results of treatment of brain abscess by these methods left much to be desired. Many patients succumbed to their infections before surgical drainage could safely be undertaken and

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half or more of those operated upon died as well. Surgeons have exhibited an understandable reluctance to publish their over-all results but many have admitted their inability to match the results of Macewen of Glasgow, who in 1893 reported survival of eighteen of nineteen patients in whom an abscess was drained. This is perhaps a reflection of their lack of Macewen's fortitude in waiting out the development of a truly chronic stage of the abscess before drainage.

Less generally appreciated was the high incidence of neurologic residues, particularly of convulsive seizures, after the "successful" treatment of brain abscess. After an abscess has been drained and heals, there remains a mass of fibrous and necrotic tissue embedded in the brain and frequently connected with cortical areas which is practically a classical example of an epileptogenic scar. The development of fits has been reported to occur in one-half of those who survive drainage of a brain abscess.

This residual core of the healed abscess was also a potential source of reinfection and many ostensibly cured patients returned a few months later with a recurrence of activity in their abscesses.

With the arrival of effective antibiotic drugs, dramatic and welcome changes occurred in the incidence, behavior and treatment of brain abscess, as in so many other fields of medicine. Most striking has been the great reduction in the incidence of brain abscesses through the effective control of their precursors, namely, sinusitis, mastoiditis, pulmonary infections and infected craniocerebral wounds. This hardly requires further elaboration.

The use of antibiotic drugs has also considerably altered the clinical picture of brain abscess in that the symptoms of the infection are often held in check and the patient no longer presents the toxic appearance formerly seen. Bucy early reported a group of patients in whom drug therapy alone resulted in a subsidence of symptoms and presumably in healing of the abscess. That such an improvement of the patient's clinical status is an unreliable indicator of the chemical cure of a surgical lesion is illustrated by the following experience:

Case 1.—A thirty-four-year-old woman had had a mastoidectomy following scarlet fever at age five. A post-auricular abscess developed and had drained intermittently since. Repeated suggestions that the lesion be eradicated by radical surgery were refused. The patient was admitted to the hospital by her obstetrician in January, 1954, complaining of severe headache. Her temperature was elevated. Two weeks previously a large post-auricular abscess had been drained. She was at term but not in labor. Under chemotherapy the headache and fever subsided, and the patient was discharged from the hospital after three days.

She was readmitted a day later complaining again of severe headache. Her temperature was 100 degrees F., and there was post-auricular drainage. Under chemotherapy the headache and fever again subsided and the patient was again discharged.

She was readmitted in labor nine days later and delivered a normal infant. On the second post-partum day her temperature rose and headache returned. By the fourth post-partum day her headache had become intense. At this point her condition deteriorated rapidly and within a few hours she was unresponsive, her pupils dilated and fixed. There was no choked disc.

At this point mastoidectomy was carried out and a large temporal lobe abscess was drained through the mastoid wound. The patient died a few hours later. Post-mortem examination showed the ventricles to be filled with purulent material. The brain abscess had apparently ruptured before drainage.

Of greatest interest and value to the neurosurgeon has been the wider latitude of intracranial maneuvers rendered possible by the use of antibiotic drugs. Whereas formerly much of the surgeon's effort was directed at avoiding spreading of the infectious process into uncontaminated areas he now finds himself able to open the skull widely and to deal with the abscess much as he would with a tumor. The object of surgery has become the total removal of all infected cerebral tissue rather than simple drainage of the abscess.

Early efforts at deliberate complete excision of brain abscess were carried out in two stages. Vincent tapped the abscess in a subacute stage and then several weeks later returned and enucleated the entire unopened mass. Kahn allowed the unopened mass to migrate toward an overlying decompression and later drained or removed it. Groff and Grant devised a modification of Vincent's method in which the abscess was tapped and then immediately enucleated if found to be thick-walled. If the abscess was thin-walled its removal was postponed until a later date. In all of these methods it was planned to remove the abscess as an intact, unopened mass.

Fincher appears to have been among the first to advocate resection of the whole infectious process and of any remnants of involved brain tissue at an initial operation which was terminated by primary wound closure. He reported five cases successfully treated in this manner in 1946. These patients were, of course, all subjected to intensive chemotherapy both before and after the surgical procedure.

LeBeau also has demonstrated that brain abscess of all stages of development can be successfully attacked and removed in a piece-meal fashion and the wound tightly closed. Such a method, when successful, is not only curative of the abscess, but results in a marked shortening of surgical morbidity as compared with more traditional drainage methods. It seems reasonable to assume that there will also be a significant reduction in neurologic sequelae, particularly convulsive seizures.

Two cases treated last year illustrate the application of this method.

Case 2.—Three months prior to his admission to St. Barnabas Hospital a 17-year-old boy, while sliding, had been struck in the region of the left eye by the limb of a tree. Marked ecchymosis and edema of the eyelids developed and as this subsided diplopia was noted on gazing to the left. Improvement followed under chemotherapy. One month prior to admission the boy began complaining of left-sided headache, fatigue, and uncinate seizures (olfactory hallucinations). When he began to vomit he was referred to a neuropsychiatrist as a functional problem.

Under observation in the hospital the patient developed blurring of his optic disc margins, a febrile course and a moderately stiff neck. The spinal fluid was under increased pressure and contained 1,000 cells per cubic millimeter, 90 per cent being polymorphonuclear. The sedimentation rate was 56 millimeters in one hour. He was placed on intensive chemotherapy.

On April 4, 1953, a ventriculogram indicated a mass in the left temporal region and craniotomy was immediately undertaken. As is our usual custom, a circular craniotomy was carried out employing a hole saw of 1½-inch diameter. Because the brain was under considerable tension, the abscess was first drained through the unopened dura. This resulted in sufficient reduction of pressure to permit wide dural opening and the exploration of a tract through the brain down to the abscess itself, which was found as an irregularly nodular sac measuring about 2 by 4 centimeters and having a stalk-like extension which ran forward to the region of the superior orbital fissure, that is, the posterior margin of the orbit. Apparently a twig had penetrated not only the orbit but the brain itself through the superior orpital fissure. The mass was mobilized and removed in one piece following which all of the softened brain about it was sucked away. The wound was then closed without drainage. The free bone flap was replaced so as to leave the patient without a defect in his skull. Cultures of the brain abscess yielded a coagulase positive strain of Staphylococcus aureus which was sensitive to terramycin and aureomycin. The patient was placed on intensive therapy with these drugs plus penicillin.

The patient became afebrile in nine days and chemotherapy was discontinued in fourteen days. He was discharged on the sixteenth postoperative day at which time his wound was well healed and he was free from all complaints except a mild residual diplopia on left lateral gaze. He has remained in good health.

Case 3.—A sixty-three-year-old flour miller with bilateral chronic ear infections of many years' standing suddenly developed fever and right ear pain. Under treatment with aureomycin he improved but a week later the pain returned and the patient had a generalized convulsion. Upon admission to Northwestern Hospital under the care of Dr. R. Priest, the patient was obviously ill and appeared mentally confused. The right ear canal was draining purulent material profusely. The spinal fluid was under normal pressure and contained 79 cells per cu. mm., 85 per cent being mononuclear.

On the following day radical mastoidectomy was performed on the right side. A wide area of dura was exposed but no evidence of dural involvement by the infection was seen.

Following this procedure the patient was much improved but continued to exhibit apparent confusion which was actually the result of a receptive aphasia. Neurologic examination gave normal findings. It was felt that there was insufficient evidence to justify a diagnosis of brain abscess and continued chemotherapy was advised.

Under observation the patient became disoriented as to time and place. Re-examination of the spinal fluid showed its cell count to have returned to normal levels (3) and to be still under normal pressure.

On June 10, 1953, a pneumoencephalogram indicated a mass in the left temporal region. By this time the left ear canal was also draining pus profusely. Left radical mastoidectomy was performed on June 12 and again the dura was not obviously diseased.

At the conclusion of the mastoidectomy the temporal lobe was explored through a separate incision. A 2-inch circular craniotomy was made with a hole saw. The exploring cannula encountered a soft area just above the petrosal ridge from which 45 cc. of foul-smelling pus was aspirated. Following this the dura was opened widely and soft brain tissue was sucked away until a poorly defined abscess capsule was encountered. This was attached to the inner surface of the dura just above the mastoid. All of the purulent material and obviously softened brain tissue were sucked away.

Following this the wound was closed without drainage, the free bone flap being replaced in the wound. The patient was given aureomycin intravenously during the operation, and postoperatively received penicillin and streptomycin also. Aerobacter aerogenes was cultured from the left ear canal, the left mastoid and the brain abscess.

The patient made a satisfactory recovery without complication of any sort, his wound healing promptly. His aphasia improved progressively but was still apparent when he was discharged from the hospital on the ninth postoperative day. Chemotherapy was continued at home for two weeks. After several months his aphasia had improved sufficiently to permit a return to his regular work. The patient has remained well.

A number of clinical aspects of brain abscess in the present day are illustrated by the foregoing cases.

In two instances the abscesses developed as complications of chronic ear infections, but in each case the symptoms of the abscess were at first so minimized by the use of antibiotics that the diagnosis of brain abscess was not seriously entertained until the patient was in a rather critical state, much too critical in the first instance. The chronically draining ear remains a serious threat to the patient and deserves appropriate surgical as well as medical treatment.

In the third case the rather unusual circumstance of penetration of the brain through the orbit by a foreign body, a twig, led to a temporal lobe abscess. Here again chemotherapy so altered the clinical course of the disease in its early stages that when it became chronic, its symptoms were at first attributed to functional mechanisms rather than to the seemingly minor injury.

In both instances in which the abscess was approached through a clean field and the entire infected area of brain, together with the abscess, was radically removed, a happy result followed. With the patients under intensive systemic chemotherapy before, during and after operation, healing of the wound was prompt in each instance and there has been no hint of recurrence in over a year. We have not felt it wise or necessary to instill drugs locally in these wounds since some of the drugs are irritating to the brain, in high concentrations, and since effective blood and spinal fluid levels are produced by systemic administrations.

The relatively brief and uncomplicated postoperative courses of these patients is in sharp contrast to the prolonged period of drainage and dressings often encountered under former methods. The prompt wound healing in these cases is the more remarkable in view of the replacement of free bone flaps in wounds which were almost certainly infected. Such a maneuver in earlier days would almost inevitably have led to osteomyelitis of the flap requiring its subsequent removal finally to achieve wound healing. The absence of disfiguring scars and of skull defects, while not of prime importance, is certainly a worth-while consideration. Finally, one can hardly doubt that these patients have much less cerebral scarring than do those in whom a drained abscess is allowed to heal and remain in the brain.

The over-all results of such a radical method of treating brain abscess can, of course, not be judged from a few cases. At present brain abscesses are encountered so seldom as to make the collection of an adequate series of treated cases a difficult matter for a single surgeon. A recent report from the Massachusetts General Hospital gives some indication of the superiority of similar methods. Thirty-one patients with brain abscess were treated during the years 1936-1940, with twenty-five deaths (80 per cent). In the years 1946-1950, however, only ten of twenty-nine patients died (34.5 per cent), and none of these from spread of infection at time of operation. Many of these patients were treated by excision of the abscess.

Summary

The intensive systemic administration of suitable antibiotic drugs permits more radical surgical treatment of brain abscess, the aim of which is the complete excision of the infectious intracranial process followed by primary closure of the wound. Such a method not only cures the abscess but results in a significant reduction of morbidity and should result in a reduction in such sequelae as convulsive seizures and recurrent abscesses.

The advent of such a method may finally permit our approaching Macewen's optimistic opinion voiced in 1893 that " . . . there is no cerebral affliction more amendable to surgical treatment, and none which offers better results."

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A Study of Cancer Mortality in Minneapolis, Minnesota

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CANCER ranks second only to diseases of the heart as a cause of death throughout the United States. In 1953 it accounted for one out of each six deaths reported for Minneapolis, taking a toll of 973 city residents—nearly four times as many as were killed in accidents that year.

The crude death rate from cancer continues to rise even though early diagnosis with prompt and adequate treatment could prevent many of these deaths and the suffering preceding them. The Public Health Service cancer morbidity surveys in 1937-1939 and again in 1948-1949 for ten large cities in the United States—Altanta, New Orleans, Birmingham, Dallas, San Francisco, Denver, Chicago, Detroit, Pittsburgh and Philadelphia—showed that in these cities only half of the new cases were diagnosed while the cancer was still localized and that two out of five patients with newly diagnosed cancer would die within one year unless earlier diagnoses are made.[1]

While information on the incidence of cancer in Minneapolis is not available, the Minnesota Health Department Cancer Study[2] showed that, in 1950, 68.7 per cent of the hospital admissions for treatment of cancer were newly diagnosed cases. If this statewide proportion held true in Minneapolis that year, there would have been 1,390 new cancer cases among the 2,023 known to have been hospitalized for treatment of this disease. On this basis, in 1950 eleven new cases would have been diagnosed here for each seven deaths from cancer.

Local Study

Purpose.—In order to determine in general what progress is being or could be made in the control of cancer in Minneapolis, a study of local cancer mortality data was undertaken. This analysis was planned to find out how much of the apparent increase in mortality from this disease is real; what changes have occurred within the past ten years in the death rates at various ages and for various sites of cancer; and what occupational groups are principally affected here.

Sources and Methods.—The information used in this study was limited to that from the death certificates. Resident data are used whenever available to show the present status of cancer mortality here. Since information on cancer deaths before 1944 is available only by place of occurrence, comparisons with previous years is limited to these data which will include some non-residents brought here for terminal care.

The standard international classification of causes of death was used throughout. Insofar as is known, the comparability of the total figures for malignant neoplasms was little affected by the various decennial revisions until the sixth revision which was adopted for use here in 1950. In this revision for the first time leukemias and lymphosarcomas, including Hodgkin's disease, were made part of the neoplasm group. This would have increased the number of deaths from cancer by about 5 per cent. However, the change in the method of selecting the underlying cause of death would have reduced the number reported as due to cancer. The net effect of the classification changes was a reduction of about 1 per cent.[3]

Findings

General Trend.—The over-all death rate from cancer for residents and non-residents in Minneapolis nearly trebled between 1900 and 1940—rising from 59.2 deaths per 100,000 population in 1900 to 171.6 in 1940.

During the past ten years the cancer mortality rate for city residents rose from 149.0 in 1944 to 183.4 in 1953, an increase of 23 per cent. Much of this increase is due to the increased number of aged persons in the population. Be-

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TABLE I. CANCER DEATH RATES BY GEOGRAPHIC AREA IN 1950

States in the Geographic Area	Cancer Death Rate per 100,000
New England	176.3
Middle Atlantic	170.4
East North Central	152.1
West North Central*	149.1
Pacific	139.6
West South Central	108.7
South Atlantic	106.5
Mountain	104.8
East South Central	102.8

*Includes Iowa, Kansas, Minnesota, Missouri, Nebraska, and North and South Dakota.

TABLE II. CANCER MORTALITY BY AGE, MINNEAPOLIS RESIDENTS, 1951-1953

	1951-53 Cancer Deaths		Annual Cancer Death Rate per 100,000 Pop.		Per Cent of Deaths From All Causes Due to Cancer	
	Male	Female	Male	Female	Male	Female
All Ages	1346	1414	180.5	172.5	14.9	20.1
Under 20 yrs.	23	17	10.8	7.9	3.4	3.7
20-29	14	17	10.5	11.5	9.2	19.5
30-39	26	50	25.1	43.2	10.9	29.8
40-49	87	122	92.5	110.8	15.7	35.4
50-59	217	284	232.1	273.6	16.9	36.9
60-69	547	554	1433.3	1087.8	14.2	14.5

tween 1940 and 1950, while the entire population here increased 6 per cent, the number of persons sixty years and over gained 36 per cent, and those under fifteen years, 18 per cent. A very small part of the increase may be due to the change in classification of causes of death.

The United States and Minnesota increases in cancer mortality are similar but more gradual than those for Minneapolis. The United States rate has increased from 64.0 per 100,000 in 1910 to 144.7 in 1953. During that period Minnesota rates rose from 67.4 to 158.5

Urban-Rural Differences.—The cancer death rates have been markedly higher in urban areas of the United States—particularly cities of 100,000 or more population—than they have in rural communities. In 1940 if the differences in the age distribution of the population are removed, cancer death rate for the urban areas was 137.6 compared with 95.5 in the rural communities.

Geographic Differences.—The cancer mortality rates differ widely in the various sections of the country. Rates are highest in the northern states, particularly those in New England, where in 1950 there were 176.3 cancer deaths per 100,000 population—and lowest in states in the east south central section with a rate of 102.8, as indicated in Table I.

While part of these differences may be due to variations between regions in the proportion of adults in the population, recognition and reporting of the disease must be important in producing such widely differing death rates as the 69.4 for New Mexico and 179.6 for Massachusetts.[3]

Comparison with Other Large Cities.—In general, among the twenty-five largest cities of the United States with populations over 400,000 in 1950, those in the north tended to have higher cancer death rates than those in the south. The only exception to this was Detroit, a northern city, with the fairly low rate of 144.5. The rates ranged from 199.3 in Seattle to 122.5 in Dallas. There was no apparent tendency for the larger cities to have the higher cancer death rates. Minneapolis ranked seventeenth in the population and sixteenth in the size of the cancer death rate among these cities.

Age on Death.—Cancer mortality in Minneapolis increases rapidly with age from twenty years on for males and thirty years on for females. During the years from 1951 through 1953, the average annual cancer death rate for Minneapolis male residents ranged from eleven deaths per 100,000 for boys under twenty years of age to 1,433 deaths from this cause among men seventy years of age and older (as shown in Table II). The rates for females ranged from eight deaths per 100,000 girls under twenty years of age to 1,088 deaths per 100,000 women twenty years of age and over.

The rates for women are less than those for men, particularly from sixty years of age on, but slightly greater in the ages from thirty to fifty-nine years. The differences in these rates, however, were so small that they could easily be due to chance alone.

Since the death rate from all causes shows a similar increase with age, the proportion of these due to cancer shows more clearly the relative impact of this disease with age. For men of all ages, fifteen out of each 100 deaths were from cancer compared with twenty out of each 100 deaths among women. Except for the very young and the very old, women appear more likely—if they die between the ages of twenty and fifty-nine—to die from cancer than do men.

TABLE III. CANCER MORTALITY BY AGE IN 1941-1943 AND 1951-1953.
ALL CANCER DEATHS OCCURRING IN MINNEAPOLIS

	No. of Cancer Deaths		Annual Cancer Death Rate*	
	1951-1953	1941-1943	1951-1953	1941-1943**
Males				
All Ages	1541	1433	206.6	203.7
Under 20	57	39	26.3	19.3
20-29	14	27	10.4	22.7
30-39	34	33	32.6	30.8
40-49	92	105	97.8	97.0
50-59	213	249	227.9	280.6
60-69	472	425	688.2	882.6
70 years on	659	555	1727.1	1782.8
Females				
All Ages	1678	1498	204.8	193.6
Under 20	51	27	23.5	13.2
20-29	26	23	17.5	15.4
30-39	69	71	59.4	56.2
40-49	163	204	147.9	173.5
50-59	327	328	314.9	379.7
60-69	433	365	592.8	694.5
70 years on	609	480	1195.5	1286.1

*Rates per 100,000 population. Deaths include residents and non-residents.
**If the 1951-1953 age specific rates were applied to the population in this period, the adjusted rate for men would be 181.6 per 100,000 population and for women 174.9.

If these rates were adjusted to eliminate the sharp increase in the proportion of children and persons sixty years of age and older in the population between 1940 and 1950, the cancer death rate is found to have decreased 10 per cent in the past decade—a significant drop not likely to be due to chance alone. Applying the 1951-1953 cancer death rates for each age group to the 1940 population, we find that if the 1951-1953 rates had existed in the earlier period, only 182 men per 100,000 population would have died from this disease instead of the 204 who did and 175 rather than 194 women per 100,000 would have died from cancer.

As shown in Table III, the death rates for both men and women decreased slightly in the ten-year period for persons fifty years of age and over. The only significant decrease, however, is that for men aged sixty to sixty-nine years.

TABLE IV. PRIMARY SITE OF CANCER.
DEATHS IN MINNEAPOLIS FROM MALIGNANT NEOPLASMS,
1941-1943 AND 1951-1953

	Number of Cancer Deaths		Annual Cancer Death Rates Per 100,000 Pop.			Per Cent Change from 1941-43*
	1951-53	1941-43	1951-53 Rates*		1941-43	In Age Adj. Rates
			Crude	Age Adj.		
All Sites	3219	2931	205.7	178.0	198.4	−10.3
Buccal Cavity	61	60	3.9	3.2	4.1	−21.9
Digestive System	1253	1253	80.1	67.0	84.8	−21.0
Respiratory System	249	165	15.9	13.8	11.2	+23.2
Breast	334	283	21.3	19.2	19.2	0
Genital Organs (Cervix, Uterus, Prostate, etc.)	456	503	29.2	25.3	34.0	−25.6
Urinary Organs	180	178	11.5	9.7	12.0	−19.2
Other Sites and Lymphoblastomas	468	355	29.9	26.8	24.0	+11.7
Leukemias	218	134	13.9	13.0	9.1	+42.9

*The crude or unadjusted 1951-1953 rates are based on the 1950 population, the age-adjusted rates were obtained by applying the age specific 1951-1953 rates to the 1940 population.

The proportion of male deaths from cancer increases steadily from 3.4 per cent for boys under twenty to a peak of 18.9 per cent among men aged sixty to sixty-nine years. Among women, the proportion of all deaths reported as due to this disease increased from 3.7 per cent for females under twenty to a high of 36.9 per cent among those dying at ages fifty to fifty-nine years.

In comparing the cancer death rates for the 1941-1943 period with those for the 1951-1953 period, the total number of deaths occurring in the city were used and leukemia and Hodgkin's disease included with the malignant conditions for both periods. The crude death rate from cancer for males on this basis in 1951-1953 was 206.6 per 100,000 population compared with 203.7 in 1941-1943. Among women the rate increased from 193.6 in 1941-1943 to 204.8.

National Office of Vital Statistics reports show that in the United States from 1900 to 1935 there has been a steady increase in the cancer death rates for persons of all ages, the greatest being for those over forty-five years with relatively smaller gains in the younger ages. Since 1935 there has been a leveling off of the age-adjusted death rates which indicates that the increase in the total cancer death rate in recent years is due to the growing proportion of persons in the older age groups.[3]

Primary Sites of Cancer.—The digestive system is the most frequent primary site of cancer. Among Minneapolis resident deaths during the past three years, 39 per cent of those from cancer were ones in which the malignancy was reported as having started in the digestive system—usually in the stomach or large intestine. Next in order were malignant neoplasms of the genital organs (uterus, prostate, et cetera) which accounted for 16.3 per cent of all cancer deaths; breast, 10.5 per cent; respiratory system, 8.5 per cent; urinary organs, 5.4 per cent; leukemias, 5 per cent; and all other sites, including lymphoblastomas, 14.2 per cent.

An improvement is evident in Minneapolis during the past ten years in the age-adjusted cancer death rates for all sites except leukemias, lymphoblastomas and malignancies of the respiratory system (Table IV). There were marked decreases in the rates for the buccal cavity, digestive system, urinary organs, and genital organs.

Early detection and treatment are essential if any real progress is to be made in the control of this disease. The survival of the patient depends largely on the stage of disease at the time of diagnosis. The more accessible the site to direct examination, the greater the opportunity for early discovery. Public Health Service surveys in the ten large cities in the United States showed that, excluding cancer of the skin, 48.3 per cent of all cancer in females and 22.6 per cent in males is located in accessible sites.

Occupation.—Occupational cancers are almost the only known cancers for which a definite cause is known and for which control is possible through elimination or modification of the carcinogenic substance or protection of the worker.

During the past 179 years many different carcinogenic substances affecting the skin, lung, bladder and/or bone marrow have been identified. According to Hueper,[4] these include in order of discovery:

soot
arsenic
paraffin oil
shale oil
pitch and tar
some crude oils
sun (ultra violet rays)
aromatic rays
roentgen rays
radium
Schistosoma hematobium
anthracene oil
betel nut
extreme dietary deficiency
creosote
benzol
uranium
radioactive rays
chromates
nickel carbonyl
asbestos
tar fumes

Breslow[5] has found a high incidence of lung cancer among certain occupations in which there is a continuing exposure to metallic particles, fumes and products of metallic combustion.

The increasing use of many of these substances in industry means that greater numbers of persons are being exposed and may develop cancer unless adequate precautions are taken to prevent excessive exposure.[6]

TABLE V. USUAL OCCUPATION FOR ALL RESIDENT CANCER DEATHS IN MINNEAPOLIS, 1953

Type of Occupation	Number of Deaths		Death Rate Per 100,000 in Occ. Gp.	
	Male	Female	Male	Female
All Types	366	423	147.2	154.9
Laborers	38	1	405.5	207.4
Skilled Trades	85	6	271.9	373.1
Semi-Skilled	31	23	115.5	176.0
Managers, Proprietors (with farm)	70	6	368.7	185.1
Professional, Technical	35	16	224.3	141.2
Private Household Employees	0	4	—	107.5
Other Service Employees	34	10	308.1	81.4
Sales	38	6	257.7	79.2
Clerical	20	23	126.8	71.9
Children Under 20	12	6		
Housewives	0	321	12.5	178.0
Invalids	0	1		
Unknown	3	0	—	—

Information obtained from death certificates for Minneapolis residents who died in the city from cancer during 1953 showed some significant differences in the cancer death rate for the various occupational groups (Table V).

Among men in Minneapolis, the mortality rate ranged from 405.5 per 100,000 in the laboring class to a rate of 115.5 for the semi-skilled group and 12.5 for unemployed males under twenty years of age. The highest rate for women was among those in the skilled trades—373 per 100,000 women employed in these trades—while the lowest rate was 71.9 for the clerical group. The death rate of 178 for housewives not in the labor force was slightly greater than the average rate of 154.9 for all women.

Respiratory cancer was found more frequently among persons employed in the skilled or semi-skilled trades or as laborers, while fewer housewives died of this type of cancer than would be expected. Cancer of the breast and genital organs was found more frequently among housewives than among the women of other occupational groups in this study.

Summary

The cancer mortality rates in the United States tend to be higher both in urban areas with more than 100,000 population than in smaller communities or rural areas and higher in the northern than the southern states. Consequently, it is to be expected that the cancer death rate in Minneapolis will be greater than those in the United States as a whole or the remainder of Minnesota.

This study of cancer mortality in Minneapolis showed that:

1. During the period from 1941-1943 to 1951-1953, the increase in the cancer mortality rate here, is apparently due largely to the increase in the number of aged in the population and in a very small part to the change in classification of the cause of death.

When the correction is made for the increase in population of children and persons over sixty years of age, the total cancer death rate decreased 10 per cent from 198 deaths per 100,000 population in 1941-1943 to 178 per 100,000 in 1951-1953.

If the population correction is not made, the total cancer death rate increased slightly from 198 to 206.

2. The death rates during the past ten years for both men and women decreased for those fifty years of age and over. The only significant drop, however, is among men aged sixty to sixty-nine years.

3. The death rates (age-adjusted) for cancers of the buccal cavity, digestive system, genital organs and urinary organs are from 19 to 27 per cent below those for these sites ten years ago. Little change is evident in the death rate from cancer of the breast, while rates for cancer of the respiratory system, other sites and lymphoblastomas and leukemias have increased 23, 12 and 43 per cent, respectively.

4. More than one-third of all deaths for women forty to fifty-nine years of age are caused by cancer.

5. Cancer of the digestive system—principally the stomach and large intestine—is still the major problem, with a death rate more than 2.5 times that for any other site. Following in order of magnitude are cancers of the genital organs, breast, respiratory system, leukemia and the urinary organs.

6. Markedly higher death rates were found among certain occupational groups for 1953. For men the occupational groups with the highest cancer death rates in Minneapolis during 1953 were laborers, with the semi-skilled having the lowest rate. Among women, those in skilled trades had the highest rate, while the clerical group had the lowest rate.

Every physician's office should be a cancer detection center. Since the survival of the patient depends largely on the stage of the disease at the time of diagnosis, early detection and treatment together with continuous education of the public regarding cancer are essential if real progress is to be made in cancer control. From the United States Public Health Service studies it has been found that, excluding cancer of the skin, 48 per cent of all cancer in females and 23 per cent in males is located in accessible sites in which early detection could be possible.

Occupational cancers are a group for which the cause is or could be definitely known and for which control is possible through modification in use of carcinogenic substances or protection of the worker from them. The increasing use of known or possible carcinogenic substances in industry makes this a potential problem. Consequently, it is important for physicians attending cancer patients to obtain information regarding possible exposure to carcinogenic agents.

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The follow-up is of more importance to the cancer patient than any other service following treatment.

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The most common sites of neoplasms in childhood are: Intracranial tumors; cutaneous and soft somatic tumors; bone tumors; eye and orbital tumors; lymphoid and hematopoietic tumors; retroperitoneal tumors.

The granulosa cell tumor is the most common functioning ovarian tumor; the majority of reported cases have been in women who have passed the menopause.

* * *

The occurrence of postmenopausal bleeding associated with an ovarian tumor is very suggestive of a granulosa cell tumor.

Physical Therapy for Painful Shoulder

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PAINFUL shoulder," a general term applied to a substantial group of closely related conditions, may severely cripple a shoulder and cause considerable disability. "Periarthritis" is another term frequently applied to shoulder conditions characterized by pain and limited motion.[1] Pain and disability are often severe and may last for days, weeks or months.

The purpose of this discussion is to describe the proper use of various forms of heat, massage and exercise in the treatment of painful shoulder. Ghormley,[2] Coventry,[1] Smith[3] and others have stressed the need for physical therapy in this condition. The tendency toward spontaneous recovery should not detract from the necessity of adequate and prompt treatment. To be truly effective these agents must be administered in the proper manner, under direct medical supervision. In addition, home physical therapy is mandatory since progress is slow and few patients can afford the prolonged professional treatment that is required, or such treatment may not be available to them.

In order to make clear the purpose of the treatment it is necessary to review some of the prevalent ideas which serve to explain the development of painful shoulder. Present concepts emphasize lesions of the so-called musculotendinous or rotator cuff of the shoulder. Calcareous deposits or partial or complete tears of the cuff may all be the result of degeneration, as first elucidated by Codman.[4] De Palma[5] has described changes in all soft-tissue components of the shoulder joint. Smith[3] and Rose[6] have pointed out that the pain which is produced by the changes in the supraspinatous tendon causes spasm of the muscles and immobilization of the shoulder. While this situation is favorable to healing and repair, if it persists over a long period it leads to muscular atrophy, adhesions and osteoporosis. The frozen shoulder represents culmination of this vicious cycle. If the spasm and pain do not persist long enough to produce the unfavorable factors, the cycle is reversed and spontaneous recovery may occur.

Coventry[1] has described personality factors which may have an adverse effect in some patients and prevent spontaneous recovery or retard improvement. He has stated further that these patients are hyperemotional and show hyperactive vasomotor responses. They expect someone else to get them well. The periarthritic personality is, according to Rome,[7] a result of "poised indecisiveness," an inability to express tension.

Although lesions of the rotator cuff may be a common cause of painful shoulder, other conditions must be considered. Inflammatory conditions such as rheumatoid arthritis or tenosynovitis of the long head of the biceps or traumatic lesions from direct injuries or chronic irritation related to certain occupations, such as heavy labor, may initiate painful shoulder. Ghormley[2] has stated that painful shoulder may result from any condition requiring prolonged fixation of the shoulder joint. Coventry[1] has pointed out that the initial cause of shoulder pain leading to painful shoulder may be irritation of a cervical nerve root either by cervical osteoarthritis or by a protruded cervical disk. Periarthritis of the shoulder may appear with coronary heart disease although the relationship is not clear, as reported by Scheifley.[8]

Heat and Massage

For the relief of pain the various types of heat and massage can be used. An effective heating device is a simple clamp lamp, as described by Krusen,[9] which may be used in the home very conveniently on a prescription basis. Shortwave or microwave diathermy is especially helpful in the treatment of painful shoulder. The former

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is applied either by the use of a cable electrode coiled around the shoulder and insulated from the skin by toweling or by means of a drum electrode. No information is available to indicate which technique is superior. However, in the department with which I am associated, the cable wrap is used most commonly, mainly because it can be applied so that the whole shoulder girdle is heated more evenly, whereas, with the drum electrode, more of the heat is concentrated over the tip of the shoulder.

Microwave diathermy may be applied to the shoulder with any of the three smaller directors directed over the tip of the shoulder joint.[10] The duration of the heat should be for thirty minutes. Occasionally, diathermy aggravates the pain in this condition. However, if the intensity of the heat is kept at a low level, the patient may derive considerable benefit without aggravation of his discomfort. Recent unpublished data indicate that pulsed ultrasonic energy applied to the shoulder by the stroking technique is nearly as effective as microwave diathermy when massage and exercise are used.[11] Occasionally, it may be necessary to substitute some other form of heat for diathermy if the patient is unable to tolerate the deep heat. This is particularly true in the early stage of the disease. Instead of diathermy one may use radiant heat or hotpacks. There seems to be general agreement that x-ray therapy frequently is effective in relieving pain in the acute phase of painful shoulder.

The use of sedative massage augments the effects of heat. Moderately firm stroking and kneading movements are used to promote relaxation of the muscles and to relieve pain. The patient is enabled to move his shoulder more easily. Massage may cause pain in the acute phase, if tenderness is marked. Otherwise, it is useful in all phases of painful shoulder. A member of the family can be trained to administer massage in this condition. However, it is our belief that whenever heat is used it is well to follow it with sedative massage.

Restoration of Motion

Restoration of motion by exercises is the most important aspect of treatment in this condition. In the acute phase, motion may be limited to a few degrees by spasm and pain. If the pain can be relieved either by the use of heat and massage or by x-ray therapy or by some orthopedic procedure, such as needling or injection of compound F (hydrocortisone), motion may be restored rather rapidly, perhaps in one or two weeks, merely by active exercises. If the pain is severe in the acute phase, it may be necessary to put the shoulder at rest by the use of either a sling or an abduction splint. The need for rest in this condition has been stressed by Rose.[6] He reasoned that because of the spasm there is adoption of the position of adduction and internal rotation of the arm, and the contracture of the shoulder cuff and the shoulder in this position leads to the development of frozen shoulder, a condition which is usually extremely resistant to treatment. As a general rule, the more intense the pain, the greater the need for rest of the part. Indeed, rest in bed may be necessary for some patients with the arm maintained in the position of slight abduction and midway between internal and external rotation. The arm must be removed from the splint or the sling several times a day for general, passive, relaxed, reciprocal range-of-motion exercises of the shoulder joint. As the pain subsides, the exercise program can be increased in order to hasten recovery.

According to Watson-Jones,[12] limitation of motion by muscle guarding rapidly progresses to limitation by adhesion formation and in severe cases by generalized adhesions of the whole paracapsular tissue, leading to a frozen shoulder. Neviaser[13] has expressed the belief that there are adhesions of the folds of the inferior aspect of the relaxed capsule in addition to the adhesions of various other surfaces about the head of the humerus. Mosely[14] has expressed the same opinion and has added that probably other adhesions are present in the region of the superior aspect of the head, perhaps underneath the insertion of the rotator cuff. According to Jampol,[15] Sir Colin MacKenzie was of the opinion that a stiff shoulder did not necessarily mean that adhesions in the joint were binding the head of the humerus to the glenoid cavity. He maintained that the two factors concerned are adduction contracture which is especially favored by gravity and the weakness of the muscles which elevate the limb.

It is very important to have the confidence of the patient in regard to exercises. Otherwise, the treatment will be ineffectual. The initial exercises which are used are the so-called pendulum exercises. The body is bent forward and the affected limb hangs like a pendulum. Motion of the limb is encouraged so that it will swing in circles. Progression of these exercises can be accomplished by increasing the size of the circle which the hand describes, as well as increasing the number of repetitions. No muscular action of a shoulder girdle is required and the muscles are relaxed. A 3- or 4-pound weight may be placed in the hand to increase the effectiveness of this exercise. As Jampol[15] has correctly pointed out, the shoulder capsule must be stretched in order to mobilize the head of the humerus which is held high in the glenoid fossa either by adhesions of the shoulder capsule or by spasm of the muscles.

The next step is to proceed with exercises designed to increase the range of rotation, particularly external rotation which is usually very limited because the arm is held in a position of internal rotation and adduction. The patient is instructed to rotate the arms externally with the elbows held close to the body. When this is done actively the internal rotators of the shoulder are stretched and the external rotators are strengthened. When there is considerable tightness it will be necessary for the physical therapist to assist in this motion, thus performing active assistive exercises. There are many variations of this type of movement. For example, the patient holds his hands clasped behind his head and his elbows forward. He then pulls his elbows backward. When this motion is extremely painful, it should be done against resistance, given manually by the physical therapist. This makes use of the reciprocal innervation principle and the patient often finds that the exercise is less painful.

The next group of exercises is designed to increase the range of internal rotation. It is important that the patient be able to place his hand behind his trunk and this requires considerable range of internal rotation. The hand is placed behind the back, the forearm is supinated and the patient attempts to raise his hand as high as possible toward his scapulae. He may assist this motion with the other hand or he may use an assistive device such as an overhead pulley or stick or wand held in both hands.

Increasing the mobility of the shoulder girdle is the next step because the whole shoulder complex is limited in motion. These exercises of necessity include exercises for abduction and adduction. The patient brings the arm up forward and attempts to get it up over the level of his head. The same exercise is done bringing the arm up laterally with the hand supinated as much as possible in order to rotate the humerus to permit full abduction. It may be necessary for the therapist to provide considerable assistance in both of these motions; in addition, the therapist will prevent elevation of the scapula. The use of a shoulder wheel, overhead pulley and finger ladder is of great help in these movements. It is possible for the patient to provide an overhead pulley and finger ladder in his home. The treatment must be continued until the patient has recovered full range of motion.

Summary

Physical therapy under medical supervision is useful in all phases of painful shoulder. In the acute painful phase, the various types of heat including diathermy are used to relieve pain and spasm. If the pain is severe, the use of a sling or splint is indicated for a short period. As the pain subsides a progressive program of exercise is added in order to preserve or increase the range of motion of the shoulder joint. Some of the exercises described can be performed unaided by the patient while some require help by a physical therapist or some member of the patient's family. When the condition is more severe than usual and a more strenuous exercise program is needed, assistive devices for exercise are required.

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(Continued on Page 587)

Can We Further Decrease Infant Mortality?

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A FEW months ago a survey of infant deaths at the Virginia Municipal Hospital was started. During this time, a study of neonatal and stillbirth rates in the State of Minnesota for 1952[1] showed that we ranked third best in neonatal deaths and seventh best in stillbirths among twenty-six hospitals of similar size in Minnesota. This rating seemed rather good. In going over our deaths it became obvious that our rating could be improved. The purpose of this paper is to show that although our fetal mortality rates may be acceptable, or even good, we should not complacently ride along because we are doing so well. As you will see, there is ample room for improvement.

This study extends from April 1, 1951, to April 1, 1954. There were a total of 2,393 live births. The total death rate was 31 per thousand. The still birth rate was 14.6 per thousand. The neonatal death rate was 16.2 per thousand. There were 165 prematures with 51 deaths among the prematures. In other words, prematures which made up 6.8 per cent of the total deliveries, had 69 per cent of the deaths. Death rate among prematures was 31 per cent.

Only infants beyond twenty weeks' gestation were considered in this study. The autopsy rate was 43 per cent.

I have broken down the figures according to the relative time of death. I have not listed prematurity among the diagnoses. I feel that prematurity is only a sequelae of some other pathological condition.

If you will refer to Tables I through IV you will note that the deaths which occurred at a time most remote from the time of delivery were the deaths to which we were least able to contribute a cause; that is the macerated stillbirths. As we approach the time of delivery in the recent and intrauterine deaths, the intrapartum deaths and especially the neonatal deaths, we note that controllable obstetric factors become more and more prominent as the causative or contributing factor in the death of the infant.

By glancing at Table V which is a listing of the total number of each complication among the entire group of infant deaths, you will note that a great majority of deaths were associated with a few complications. These were, in order of decreasing frequency: premature separation of the placenta, multiple pregnancy, toxemia of pregnancy and congenital anomalies. You will note also that there were seven cases in which no particular cause of death was listed. Several of these cases had charts which were so incomplete as to make it impossible to deduce any diagnosis.

Next, the cases were picked out in which I thought there were errors of commission or omission in the treatment. I do not maintain that each of these errors caused the death of the baby in each instance. I do maintain, however, that the errors were not in the best interests of the patient. Table VI is a listing of these errors. When reading over this list, keep in mind that these things occurred in a hospital which has quite a low infant death rate compared to other similar hospitals in the state.

Next, I have grouped cases according to obstetrical diagnoses. I will mention only the cases which, in my opinion, might have been prevented or in which something grossly wrong was done or in which something important was omitted in the treatment. The first of these comes under the heading of indiscriminate use of oxytocic drugs. There were seven of these cases. In four, oxytocics were used without medical or obstetrical indication, apparently for the sake of convenience:

1. Probably not preventable. Birth weight 1 pound 2 ounces. Intramuscular pitocin near the end of first stage.
2. Term. Castor oil and quinine. Hard, driving, short labor. Prolapsed arm. Autopsy, bloody subarachnoid fluid, adrenal hemorrhage.
3. Powdered ergot induction. Birth weight 4½ pounds. Persistent posterior. Autopsy, hemorrhage in the meninges, 7½ gr. nembutal three hours before delivery.

Presented at a meeting of Minnesota Society of Obstetrics and Gynecology in Minneapolis, December 4, 1954.

TABLE I. DIAGNOSES ASSOCIATED WITH STILLBORN MACERATED FETUSES

Stillborn macerated fetuses	12
Prematures	7

3	One of a set of twins. Small fibrotic placenta.
3	No particular cause of death. Small fibrotic placenta. Many white infarcts.
3	Congenital.
1	Rh sensitization—37 weeks.
1	Ulcerative colitis—7½ months.
1	Acute toxemia near term.

TABLE II. DIAGNOSES ASSOCIATED WITH RECENT INTRAUTERINE DEATHS

Recent Intrauterine Deaths	11
Prematures	7

3	Premature separation of placenta.
2	Chronic nephritis? Twins.
2	Acute toxemia.
1	Neuritis?
1	Chart useless. No diagnosis possible.
1	Rupture of section scar.
1	Premature separation of placenta—fatal auto accident.

TABLE III. DIAGNOSES ASSOCIATED WITH INTRAPARTUM DEATHS

Intrapartum deaths	12
Prematures	8

4	Premature separation.
3	No particular cause—(1 driving labor; 1 pitocin, 1 pound 2 ounces).
2	Breech.
1	Face presentation (pitocin).
1	Premature rupture of membranes with prolapsed cord.
1	Pitocin induction—F.H.T. stopped during induction. ? Rh sensitivity.

4. Second of twins which were undiagnosed. Intramuscular pitocin at 6 cm. along with 7 cc. delvinal.

The next three cases show the improper use of oxytocics. These include:

1. Pitocin induction. Possible Rh sensitivity. Membranes not ruptured. Separation of placenta followed induction.

2. Pitocin without rupturing the membrane. Premature separation.

3. Face presentation, chin anterior. Position not known. Pitocin because of lack of progress of presenting part.

In three of the cases where there was no medical indication for their use, oxytocic drugs must be held responsible for the death of the baby since they were of viable size and there was no other serious complication. In two of the cases where there were other complications it is quite probable that the oxytocic drugs used improperly were responsible for the death of the baby. If we discard the eight deaths in infants who showed congenital anomalies incompatible with life, these five cases in which oxytocic drugs were used indiscriminately would account for 7.5 per cent of the infant deaths during this three-year period.

TABLE IV. DIAGNOSES ASSOCIATED WITH NEONATAL DEATHS

Neonatal Deaths	39
Prematures	29

9	Premature labor with twins (1 pitocin for convenience). (1 congenital anomaly).
7	Premature separation (1 diabetic).
5	Placenta previa.
4	Congenital anomalies.
4	Toxemia (1 diabetic).
2	Rh sensitization (1 death due to regurgitation and aspiration).
2	Induction of labor for convenience.
2	Premature labor—no cause.
1	Breech.
1	Persistent posterior (attempted unsuccessful version).
1	Lobar pneumonia.
1	Apparently normal pregnancy and delivery. Probable aspiration in nursery.

TABLE V. TOTALS OF DIAGNOSES ASSOCIATED WITH DEATHS IN THE ENTIRE SERIES

15	Premature separation.
11	Multiple pregnancy.
9	Toxemia (One of these was multiple pregnancy).
8	Congenital anomalies.
7	No particular cause—several useless charts—1 pitocin.
5	Placenta previa.
4	Rh—2 of these probably not due to Rh (1 pitocin and 1 aspiration).
3	Breech.
3	Acute infection (pneumonia, colitis and neuritis?).
2	Induction of labor.
2	Premature labor—cause undetermined.
1	Version extraction on posterior.
1	Face—chin anterior.
1	Rupture of section scar.
1	Prolapsed cord.
1	Regurgitation and aspiration. Normal OB.

TABLE VI. ERRORS

Obstetric	
74	No record of anesthesia.
45	No serology.
38	No Rh typing (mother).
33	Consultation not obtained in bleeders, toxemias, malpositions, compound presentations.
8	Toxemias with delayed and inadequate treatment.
7	Charts very incomplete.
6	Large amounts of respiratory depressants too close to delivery.
4	Oxytocic drugs without indication.
3	Oxytocic drugs with improper indication or without precautions.
1	Prolapsed cord. Closer attention to F.H.T.
1	Spinal anesthesia to mother in shock for delivery.
Pediatric	
39	No history or physical on any live birth chart.
18	No consultation.
10	Cyanosis, labored respirations and in oxygen—babies fed and watered.
8	Over 2½ pounds. Tracheal tug. No attempt to clear airway.
1	Alpha lobelin to an apneic baby—no airway or oxygen.
1	CO_2 only to an apneic baby—no other respiratory stimulant.
1	Cyanotic premature—bath in water.
1	Regurgitation—aspiration—Nothing done until doctor arrived.

This is too high a price to pay for someone's convenience. It is time for those who have the patients' welfare at heart to take a stand against those who glibly advocate labor and delivery at a time of their own choosing through the use of oxytocic drugs.

The next group is that of the toxemias. These number seven. Some of my comments written while going over these charts have been noted:

1. Twin pregnancy. The only thing noted on the entire chart was a mention of albuminuria during this pregnancy. No physical examination. No blood pressure during the prenatal course. No blood pressure on the patient's chart.

2. Acute toxemia. Inadequate therapy. No abnormalties noted in the prenatal care. No record of attempt to control fluid balance.
3. Patient responsibility.
4. Treatment inadequate. Eclampsia developed. Toxemia subsided, but labor was not induced.
5. Multiple pregnancy. Moderately severe pre-eclampsia. Toxemia of several weeks' duration. Procrastination.
6. No record of prenatal blood pressure. Procrastination.

The chief errors in these cases seem to be in failure to institute adequate medical therapy soon enough and failure to induce labor once the toxemia is stabilized.

The next group, that of premature separation, numbered six. Again some of my comments on the cases are recorded:

1. Labor was induced with intramuscular pitocin. Membranes were not ruptured, and no pelvic examination.
2. Patient responsibility.
3. Patient bled for six hours in the hospital without any attempt at diagnosis or treatment.
4. Intramuscular pitocin without rupture of the membranes.
5. No pelvic examination. Membranes were not ruptured, and intramuscular pitocin was given.
6. Septate uterus. Placenta was found in one horn and infant in the other. Membranes were not ruptured. Vaginal delivery was done.

Along with these there were two cases of placenta previa in which the treatment was questionable. One was a seven months' pregnancy. With due precaution this pregnancy might have been prolonged in the hospital to a period of viability. In the other case, the patient bled nine hours in the hospital without treatment, finally went into labor and delivered spontaneously.

In these bleeders, then, we have the use of oxytocics without rupturing the membranes and lack of any treatment whatsoever. Cesarean section was not considered in at least one case where it should have been, that of the septate uterus.

The next group is that of malposition. These number four:

1. An occiput posterior, weight 7 pounds 12 ounces, in a primigravida. No pelvimetry or pelvic measurements were recorded on the chart. Attempted version extraction, jack-knifed the baby, got stuck, then called for help. Postpartum pelvimetry revealed a slightly contracted pelvis.
2. A frank breech in a para 3, gravida 4. Weight 8 pounds 1¾ ounces. The second stage lasted two hours. Fetal heart tones were heard until just before delivery. Infant was stillborn. Tentorial tear and bilateral adrenal hemorrhages were found.
3. A prolapsed arm in which labor was induced for convenience in a primagravida. Forceps delivery with no episiotomy was done. Baby was live born, breathing very poorly, and was not given suction. No oxygen was used. Autopsy, bloody subarachnoid fluid and one large adrenal hemorrhage were found.
4. Frank breech, weight 4 pounds 9¼ ounces. Premature rupture of the membranes occurred. Death was apparently due to inexperience—did not recognize the position, did not call for consultation until the body had delivered, unable to deliver the head.

There should be practically no deaths from malposition if recognized and properly treated.

Under Rh sensitivity there were two cases which should not have been lost:

1. Induction by rupture of membranes. Infant delivered at thirty-six weeks. The infant was doing well so far as the Rh was concerned, but regurgitated and aspirated. The nursery personnel left the infant to summon the doctor by telephone without applying suction.
2. Rh negativity of the mother was not noted during pregnancy. Typing was apparently wrong. This was not discovered until the baby developed jaundice on the second day of life. Infant was not transfused. Baby lived four days.

Under miscellaneous:

1. A premature rupture of the membranes and a prolapsed cord were not noted until delivery. Fetal heart tones were heard one-half hour before delivery. Closer observation of the fetal heart tones would have alerted one to this possibility.
2. Spontaneous rupture of an old section scar was the diagnosis which was missed for a period of six to eight hours before laparotomy. Patient went into shock. Spinal anesthesia was used.

We then find four groups of cases in which most of the errors of commission or omission occur:

1. In the *toxemias* the chief difficulty seems to be inadequate treatment and procrastination.
2. In the *bleeders* we have lack of adequate treatment and the use of oxytocics without the rupture of the membranes.
3. In the *malpositions* it appears to be a matter of inexperience, inability to make a proper diagnosis, or complete lack of judgment.
4. In the *indiscriminate use of oxytocics* the chief difficulty seems to be the indiscriminate use of oxytocics.

It is quite obvious that our infant salvage could have been improved. In a few concise statements, how could we have improved it?

(1) By being constantly on the alert for toxemias; treating them adequately, and inducing labor when the toxemia is stabilized. (2) By treating bleeders after making a diagnosis. If induction of labor is selected, the membranes should be first be ruptured. (3) One should always make a diagnosis of the position during labor by whatever means necessary. One should not attempt operative maneuvers beyond his capability. (4) The oxytocic drugs should not be used for the convenience of the patient or the physician. If there is an indication for their use, the recognized prerequisites and precautions should be taken. (5) Newborn infants who are cyanotic or having difficult respirations should receive nothing by mouth and should not be bathed. Stimulants should not be given to a newborn infant unless a clear air-way is assured and an adequate supply of oxygen is being given. Carbon dioxide in an apneic infant is not a respiratory stimulant. Nursery personnel should be instructed in, and told to use the emergency procedures in case of regurgitation and possible aspiration.

These sound like dry old text-book statements, which they are. However, had the physicians responsible in these cases been familiar with, and used, these dry old statements, there would have been quite a few more live children today.

I am sure that similar, and even worse, situations exist throughout the country. If such conditions exist in a hospital which has a quite low infant mortality rate, one can imagine the state of chaos that must prevail in the institutions with a high mortality rate.

Some of the findings of this study have been presented to our medical staff. Many improvements in the charts have already been made.

The amount, type, and duration of anesthesia are now fully recorded. Immediate postpartum examinations and discharge examinations on the infants are now being fully recorded. A labor history is included in the infants nursery record including the amount of sedation, anesthesia, pathological conditions in labor and immediate condition of the infant at birth. A pediatrician has been placed in charge of the nursery. Most important, we are starting a permanent fetal mortality study. The group studying this includes a pediatrician and a pathologist. We feel that all infants, whether stillborn or live born, should be included in this study. We feel that there should be a high degree of uniformity in these studies throughout the state and for this reason we are using a form which requires essentially the same information as the Hennepin County Fetal Mortality Study Committee. We definitely feel that these studies should be carried on at local level.

Addendum

It is of interest to note in relation to the indiscriminate use of oxytocic drugs that on this same program, in a round-table discussion, it was brought out by Dr. J. L. McKelvey that in an entirely independent study, the indiscriminate use of oxytocic drugs was the third most common cause of maternal mortality in Minnesota. The percentage of maternal deaths (approximately 7 per cent) attributed to the oxytocic drugs was essentially the same as stated by this author (7.5 per cent) in relation to infant mortality attributed to oxytocic drugs.

Reference

1. Minnesota Department of Health, Section of Maternal and Child Health and Section of Vital Statistics: Infant and Neonatal Mortality and Stillbirth Rates by Hospitals, 1952. p. 3.

Discussion

E. C. Maeder, M.D., Minneapolis: I wish to congratulate Dr. Pierce on his fine presentation of this timely subject, also on his interest in the formation of an Infant Mortality Study Committee in Virgina. The Hennepin County Neonatal Mortality Study is now completing its third year, during which period about 1,000 cases have been reviewed.

There is no question but that neonatal or infant mortality can be reduced by more intensive effort on the part of all physicians. Such studies should be carried out by each hospital's own staff. A team consisting of a pediatrician, obstetrician and pathologist should be appointed to appraise all neonatal deaths. The Hennepin County group has now added an anesthesiologist to its committee, as the importance of maternal anesthesia and analgesia as contributing to neonatal depression and anoxia is well recognized.

Every single fetal death should be analyzed, no matter how immature or obvious the cause. Gross fetal anomalies inconsistent with life are still being overlooked.

Dr. Pierce has stressed the importance of good complete records. They are a must for any good survey. Particular effort must be made to find the mothers who have a history of premature births, repeated abortions and miscarriages.

In the 1952 Hennepin County report of 320 mothers and 333 offsprings, obstetrical complications were associated with 114 infants or 34 per cent. With bleeding late in pregnancy, often an expectant management, with sensible precautions, does not compromise the mother's safety. Prolonging gestation to the relative safe thirty-six weeks and beyond is the greatest possible step in the

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Fractures of the Pelvis

A Report of Eighty Cases Treated at University Hospitals

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THE pelvic ring contains within its confines the pelvic viscera and forms the floor of the abdominal cavity. This ring is a semi-rigid structure formed of three large bones bound together with strong ligaments. To fracture or to separate the components of this ring requires considerable force. This force may be exerted in the body generally, producing injuries unrelated to the pelvic fracture, or it may be concentrated locally, producing injury to the pelvis and the abdominal viscera. *Two-thirds of the patients in the group reported here had other injuries occurring in association with an injury of the bony pelvis.* It is self-evident that the responsibility of the physician in ministering to these patients does not end with the establishment of a diagnosis of a fractured pelvis, but must include a thorough search for other, commonly more serious, injuries.

This report is based upon experience gained in the treatment of eighty patients (fifty-one male, twenty-nine female) who were treated at the University Hospitals in the years 1947 through 1954. The ages of the patients ranged from three to ninety-three years and were evenly distributed over the life span. One-third of them (twenty-six) had isolated fractures of the pelvic ring. Two-thirds of them (fifty-four), however, had multiple injuries in addition to fractures of the pelvis. Sixteen of these patients had visceral injuries. The remainder of the injuries varied in severity and were disseminated throughout the body (Fig. 1).

The manner in which the injury was incurred will frequently suggest the coincidence of visceral and other injuries. Was the patient crushed between two objects? Did a wheel pass over the trunk? Did he fall from a height? Was he struck, tumbled or rolled? Corroborative physical signs such as tire marks on the skin, the location of abrasions and confusions, the presence of fractured ribs, will all aid in reconstructing the course of the forces producing the injury. The initial physical examination must be thorough, with special attention to the chest and abdomen, since these findings form a base line against which all changes in the patient's findings during the succeeding hours can be evaluated.

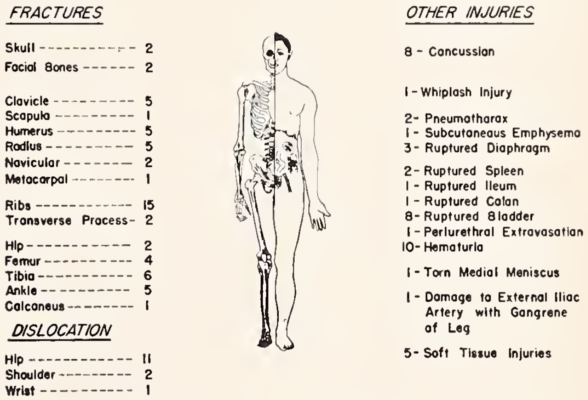
FRACTURES
Skull ---------- 2
Facial Bones ------ 2
Clavicle --------- 5
Scapula --------- 1
Humerus --------- 5
Radius --------- 5
Navicular ------- 2
Metacarpal ------- 1
Ribs ----------- 15
Transverse Process- 2
Hip ----------- 2
Femur --------- 4
Tibia ---------- 6
Ankle --------- 5
Calcaneus -------- 1
DISLOCATION
Hip ----------- 11
Shoulder -------- 2
Wrist ---------- 1
OTHER INJURIES
8 - Concussion
1 - Whiplash Injury
2 - Pneumatharax
1 - Subcutaneaus Emphysema
3 - Ruptured Diaphragm
2 - Ruptured Spleen
1 - Ruptured Ileum
1 - Ruptured Colon
8 - Ruptured Bladder
1 - Periurethral Extravasation
10 - Hematuria
1 - Torn Medial Meniscus
1 - Damage to External Iliac Artery with Gangrene of Leg
5 - Soft Tissue Injuries

Fig. 1. Injuries associated with fractures of the pelvis (80 cases).

The initial blood loss in a patient with a fracture of the rami of the pelvis may be considerable. This can be easily demonstrated on the cystogram by the displacement of the bladder away from the site of fracture by the hematoma. Shock occurring shortly after injury is usually associated with local blood loss and can be effectively treated by transfusion. Shock occurring after twelve to twenty-four hours in a previously stabilized patient is of grave import and should signal immediate exploratory laparotomy. In our cases late shock has always been associated with a ruptured abdominal viscus.

There were eight deaths occurring after fractures of the pelvis, a mortality of 10 per cent. One of these, occurred in a hospital patient with renal failure associated with congenital polycystic kidneys. One week prior to death, the patient

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From the Department of Surgery, Division of Orthopedic Surgery, University of Minnesota.

fell and sustained an undisplaced fracture of the pubic rami on one side. Another patient died of cardiac arrest during anesthesia administered for the performance of a supra pubic cystostomy in the treatment of a ruptured bladder. Four patients died as a result of complications arising from injuries to abdominal organs. One patient died of undetermined causes after leaving the hospital. One patient died of amyloidosis three years after injury as a result of chronic pelvic suppuration arising initially in an open fracture of the pubic rami and a ruptured bladder.

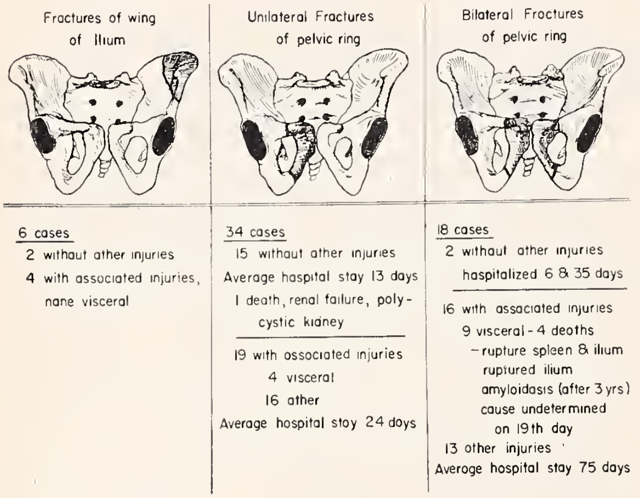
Fractures of wing of Ilium
Unilateral Fractures of pelvic ring
Bilateral Fractures of pelvic ring
6 cases
2 without other injuries
4 with associated injuries, none visceral
34 cases
15 without other injuries
Average hospital stay 13 days
1 death, renal failure, polycystic kidney
19 with associated injuries
4 visceral
16 other
Average hospital stay 24 days
18 cases
2 without other injuries hospitalized 6 & 35 days
16 with associated injuries
9 visceral - 4 deaths
- rupture spleen & ilium
ruptured ilium
amyloidosis (after 3 yrs)
cause undetermined on 19th day
13 other injuries
Average hospital stay 75 days

Fig. 2.

There were only three late complications directly attributable to the pelvic fractures. These were an ischio-rectal absess, a urethral structure, and a myloidosis. There were no complications of fracture healing. Bony union of the pelvic fractures of stabilization of the symphyses separations occurred in all cases.

The fractures of the pelvis in these patients have been divided into two groups: those requiring special treatment, and those requiring no treatment except supportive care.

Figure 2 outlines our experience with isolated fractures of the ilium, and fractures of the pubic rami, bilateral or unilateral. These cases make up about three-fourths of the total number of patients. The treatment of the fractures in this group consisted of good supportive care and the institution of activity as soon as the patient was able to move about. The patients were actively encouraged to turn, to sit up in bed, to be up in a chair and to walk with or without the support of crutches as soon as possible. The only limitation on their activity was pain. As can be easily seen, the more extensive the injury, the longer the period of hospitalization required. However, a large portion of the morbidity can be attributed to the presence of other associated injuries. Plaster dressings were not used in any of these patients in the treatment of the pelvic fractures. No complications attributable to the regimen were encountered.

Figure 3 contains information regarding those cases requiring specific treatment for their fractures of the pelvis. Fractures with displacement of the hemipelvis, fractures of the acetabulum and symphyseal separation accounted for about one-fourth of our cases.

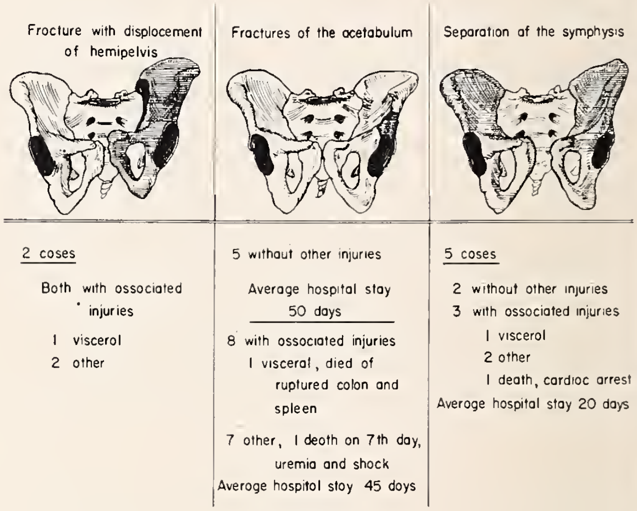
Fracture with displacement of hemipelvis
Fractures of the acetabulum
Separation of the symphysis
2 cases
Both with associated injuries
1 visceral
2 other
5 without other injuries
Average hospital stay 50 days
8 with associated injuries
1 visceral, died of ruptured colon and spleen
7 other, 1 death on 7th day, uremia and shock
Average hospital stay 45 days
5 cases
2 without other injuries
3 with associated injuries
1 visceral
2 other
1 death, cardiac arrest
Average hospital stay 20 days

Fig. 3.

Fractures of the pelvis with displacement of the hemipelvis upward were treated by strong skeletal traction through the distal end of the femur. An attempt was made to pull the displaced portion into a normal relationship with the opposite side. Traction was maintained for three to six weeks until the position of the fragments were stabilized. The patients were then encouraged to resume normal activity as soon as possible.

Fractures of the acetabulum were associated with dislocation of the hip (twelve posterior, one central) in all cases. The primary treatment consisted of reducing the dislocation and maintaining the reduction by traction on the leg. When the reduction was unstable, operative fixation of the acetabular fragment was carried out. The end result in these cases depended not upon the

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Hyperpotassemia and Hyperglycemia

Treatment with Insulin and Invert Sugar

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WHEN hyperpotassemia develops, it represents a critical complication.[7] This condition is not observed as frequently as hypopotassemia, but when it does occur its correction must be made within a few hours lest sudden death from heart stoppage supervene.[10] Cardiac arrest usually occurs in diastole in this condition.[7]

The presence of hyperpotassemia can be clinically suspected in the presence of oliguric uremia,[14] unexpected cardiac irregularities and when the patient experiences undue weakness or flaccid paralysis.[7,12] It can be definitely identified only by serum potassium determinations or by electrocardiographic findings.[14]

A number of methods[2,6,8] for the control of hyperpotassemia have been suggested. We have observed the effect of treating this condition with large doses of intravenous invert sugar and large doses of insulin on the basis that when the metabolism of glucose is forced, the potassium ion is removed from the blood stream in the process of glycogenesis.[5,8]

Case Report

J. R., aged sixty-six, 51 kg., entered the hospital January 15, 1954, with symptoms suggesting ruptured sigmoid diverticulitis. He appeared dehydrated and he was acidotic (CO_2 18.2 mEq./L). Because of the critical nature of his illness it was decided to treat him conservatively for the time being. His heart action was irregular. He was weak and listless. He was oliguric though not anuric. He was given 1,900 cc. of 5 per cent glucose, 5 per cent amino acids and 5 per cent alcohol daily for two days. During this two-day period the patient received a total of 108 mEq. sodium, 36 mEq. potassium and 164 mEq. chloride.

The results of blood chemical determinations made two days after admission are illustrated in the graphs (Figs. 1 and 2). It was evident that he was suffering from an electrolyte imbalance, the most critical aspect of which was the high serum potassium. His blood sugar likewise was elevated.

As soon as these findings were observed, he was given 50 units of insulin subcutaneously and 250 units of insulin intravenously in 2,000 cc. of 20 per cent invert sugar during the next twelve hours. The amount of insulin and invert sugar administered on subsequent days is also illustrated in the graphs.

Blood chemistries were then determined every few hours (Fig. 1).

Seventy-eight mEq. of sodium in the form of sodium lactate was given on the 15th and 16th in an effort to aid in the correction of the acidosis. No other electrolytes were administered during this period of observation. Balance studies were conducted (Fig. 3).

Five days after he had recovered from the electrolyte imbalance, as illustrated in this study, he developed a strangulating, incarcerated incisional hernia which required emergency operation. The diagnosis of ruptured sigmoid diverticulitis associated with a diffuse fibrinous exudative peritonitis was confirmed. He made a satisfactory recovery from this operation without complications.

An analysis of the studies which were made during this critical period is of interest.

Blood Sugar

It can be observed that the blood sugar dropped rapidly during the period when insulin was given on the 15th. The patient was semi-comatose at the outset of this treatment when the blood sugar was 296 mg. per 100 ml. He was responsive when the blood sugar was 146 mg. and he was in insulin shock when the blood sugar was 57 mg.

The intravenous invert sugar and insulin were then discontinued for nine hours. The blood sugar rose spontaneously to 232 mg. on the morning of the 16th. Treatment was again instituted with smaller doses of insulin and 20 per cent invert sugar. Again the blood sugar promptly came down to a low level and treatment was discontinued when the blood sugar was found to be 61 mg.

During the second interval of twelve hours in which no insulin or sugar was given, the blood sugar again rose to 304 mg. Invert sugar and insulin were given again in smaller doses until

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This study has been supported in part by a grant from the Baxter Laboratory, courtesy Robert P. Herwick, M.D., Medical Director.

the patient was able to eat on the afternoon of the 19th.

This erratic reaction of the blood sugar appears to illustrate the disturbance in the carbohydrate metabolism associated with the alarm reaction similar to that observed by Abbott and others. The bromsulphalein tests done on the 15th and 16th revealed 60 and 50 per cent retention, respectively. Glucose tolerance test done on the 27th (ten days after these studies) revealed a disturbance in the carbohydrate metabolism (blood sugar fasting 104, one-half hour 203, one hour 278, two hours 308, three hours 286).

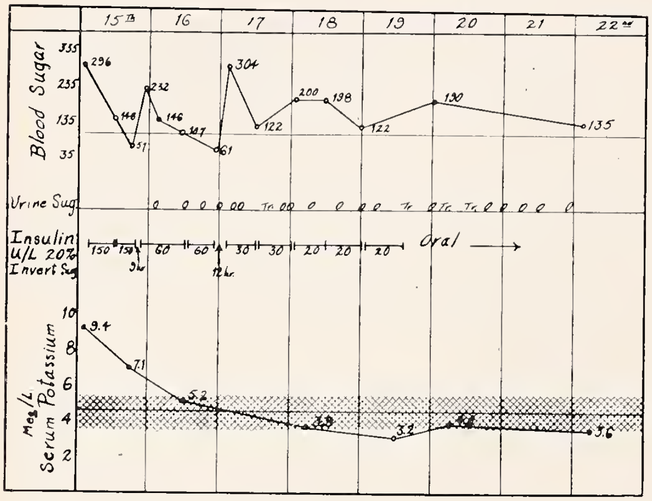

Fig. 1. Note simultaneous drop of blood sugar and serum potassium on 15th during administration of 300 units of insulin. Blood sugar rose spontaneously on two subsequent occasions (16th and 17th) when insulin and invert sugar were discontinued.

Subsequent glucose tolerance tests done four months later suggest that the carbohydrate disturbance was not diabetic (Table I).

The blood phosphate was of no value in excluding a diagnosis of diabetes.[11]

TABLE I

1954 Date		Fasting	Gm. of Glucose	½ Hr.	1 Hr.	2 Hr.	3 Hr.	Urine Sugar
1/27	Bl. Sugar	104 mgm.	100	203	278	308	286	Trace
5/19	Bl. Sugar	95 mgm.	100	160	195	125	105	0
5/26	Bl. Sugar	100 mgm.	50	141	175	125	75	0
5/26	Bl. Phos.	4.08 mgm.	50	4.24	3.95	4.16	4.16	0

The Serum Potassium

The graph illustrates the rapid drop in the serum potassium which occurred concomitantly with the administration of large doses of insulin and invert sugar. It is believed that this occurs during the process of carbohydrate metabolism as potassium is bound and presumably stored in the liver[3,4,5,8] though the potassium balance figures (Fig. 3) indicate that 411 mEq. of potassium were excreted in the urine during the three days in which the serum potassium dropped from 9.4 to 3.9 mEq.

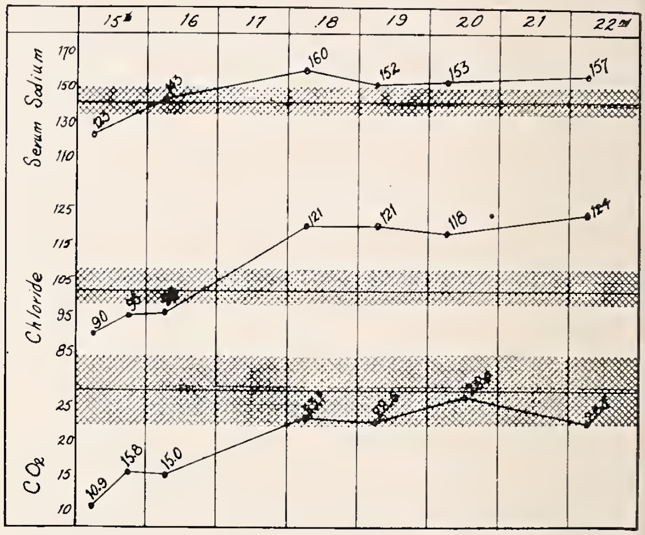

Fig. 2. Serum sodium, chloride and CO_2 combining power were all low at onset of treatment as illustrated in Figure 2. This graph illustrates the spontaneous correction of the electrolyte pattern of the blood which occurred during the forced metabolism of carbohydrate by the administration of insulin.

The Balance Studies

The intake, output and balance determinations clearly illustrate (Fig. 3) that the correction of the electrolyte pattern of the blood was not accounted for on the basis of the electrolyte intake.

There was only slight positive sodium balance on the 15th and 16th, insufficient to account for the subsequent rise in the serum sodium which occurred on the 18th and 19th even in the face of a negative sodium balance.

There was also a negative chloride balance as the blood chlorides rose.

These findings suggest a shift of the sodium and chloride from their intracellular position or from other stores in the body.[1]

There was a considerable loss of potassium in the urine which, since there was an absence of potassium intake, could have accounted for the rapid drop in the serum potassium level.

The patient was in positive caloric balance which averaged 125 per cent of his caloric requirements. All of these calories were provided from invert sugar.

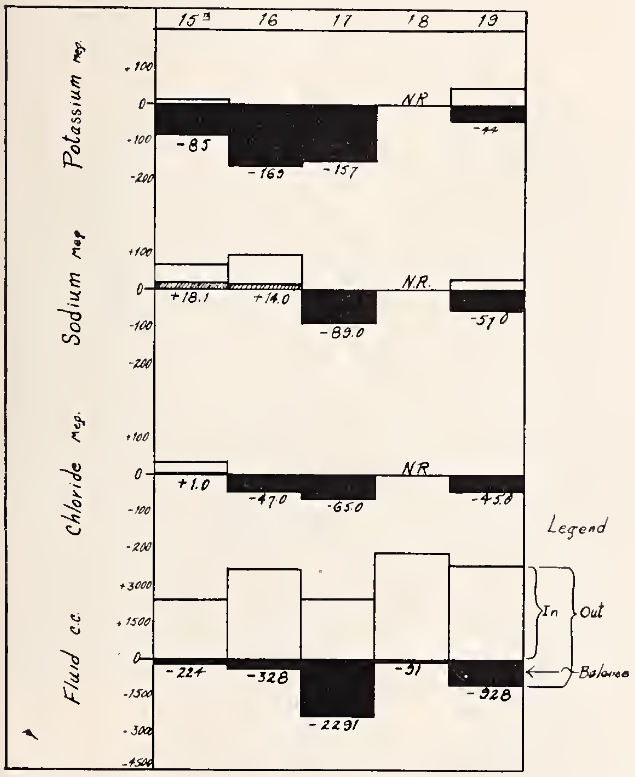

Fig. 3. Balance studies during the period of observation. There were no electrolyte intakes except for a small amount of sodium on the 15th and 16th. The potassium output could have accounted for the drop in the serum potassium as illustrated in Figure 2. Despite the negative sodium and chloride balances, and no intake, the serum sodium and chloride levels rose as illustrated in graph II. These observations seem to suggest a physiological shift of the electrolytes associated with the metabolism of invert sugar as induced by large doses of insulin.

There was a negative nitrogen balance amounting to an average of -17.51 grams per day. This would be expected in that no nitrogen was administered during this period of observation.

Diuresis began on the 15th and continued through the 19th (Fig. 3). Clinical dehydration disappeared during this period of treatment despite the negative water balance. This was no doubt due to the shift of fluid.

Comments and Conclusions

This case illustrates the effectiveness of the treatment of hyperpotassemia associated with hyperglycemia by the administration of invert sugar and insulin wherein a rapid drop in the serum potassium was observed when 300 units of insulin was given in conjunction with 400 grams

The spontaneous recurrence of high blood sugar levels on two subsequent occasions, when the gar levels on two subsequent occasions, when the administration of sugar and insulin was temporarily interrupted, suggests a disturbance in the carbohydrate metabolism. This is also indicated by the glucose tolerance test done while in the hospital and the bromsulphalein retention. The subsequent finding of a normal glucose tolerance test indicates that the patient was not a diabetic.

The other electrolytes which were in imbalance appear to have been spontaneously corrected in the period during which carbohydrate metabolism was forced with insulin. This leads us to speculate that any severe electrolyte disturbance might be benefited by forcing, with the administration of insulin, the metabolism of large quantities of carbohydrate.*

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*Subsequent studies have demonstrated this, the results to be reported in a future publication.

Bilateral Glenoid Hypoplasia

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FAILURE of development of the ossification center for the inferior rim of the glenoid is a rare anomaly. The patient described in this report presented this finding in association with bilateral flattening of the humeral heads, limitation of abduction, external rotation of the arms, and webbing of the axillae.

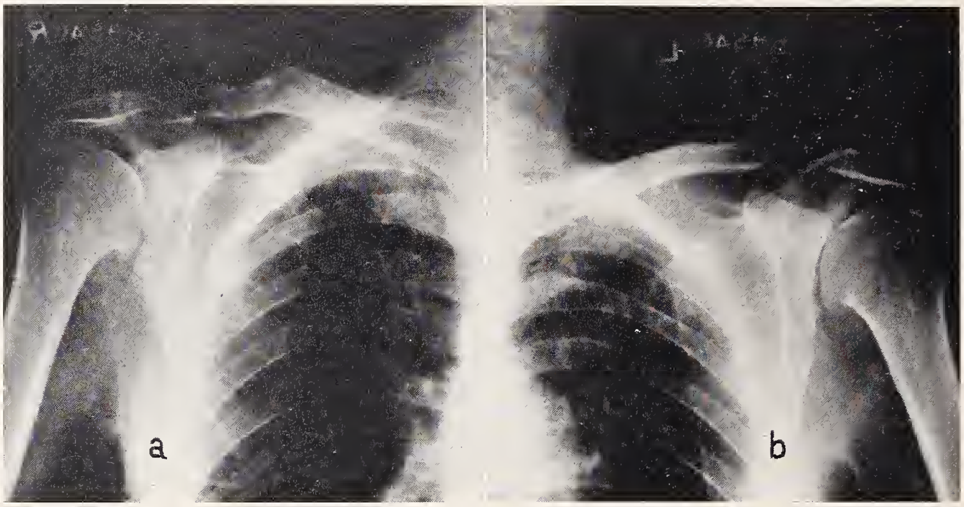

Fig. 1, a and b. Antero-posterior projections of the right and left shoulders, showing the absence of the inferior rims of the glenoid, with secondary hypertrophic changes of the lower glenoids and heads of the humeri. Flattening of the humeral heads is also apparent.

Case Report

This patient was an eighty-seven-year-old retired Navy man, whose presenting complaint was intermittent leg pain. A diagnosis of arteriosclerosis and normocytic anemia, of undetermined cause, was made, and the patient responded in a satisfactory manner to medical therapy. On routine examination of the chest it was observed that there was underdevelopment of the glenoids bilaterally, manifested primarily by absence of the inferior glenoid rims. The humeral heads showed flattening; there were secondary degenerative arthritic changes involving the bones of the shoulder joint (Fig. 1, a and b). Axial views revealed normal alignment of the heads of the humeri in the glenoid fossa (Fig. 2).

Physical examination revealed a marked limitation of external rotation of the humerus. Abduction was limited to 90°. The most striking feature of the examination was webbing of the axillae when the humeri were abducted (Fig. 3). This webbing was most prominent in the posterior axillary folds. A skeletal survey revealed no other congenital variations. No history of birth trauma or bilateral injury to the shoulder joints was elicited. At the age of twenty-five the patient sustained a fracture of his right clavicle. Radiographs were obtained of the shoulders of one son, and two male grandchildren; all appeared normal.

The patient had been aware, all of his life, of a limitation of motion of his arms; this had not hindered his ability to work. The patient's son had never noticed the father's disabilty.

Anatomic Considerations

The ossification of the glenoid is from three centers. The floor is formed by the body of the scapula, which ossifies during the eighth week of intra-uterine life. The upper margin and the coracoid process are formed from a secondary center; this appears at about the tenth year. The inferior rim is derived from a center appearing in the thirteenth year. Normally, the pre-cartilage of these secondary centers is present in utero.

In our case, and in the cases of Owen (1953), the ossification for the inferior rim failed to develop. We believe that this represents a congeni-

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tal anomaly. The flattening of the humeral heads, limitation of range of motion, axillary webbing, and degenerative arthritic changes all appear secondary to the congenital alteration at the glenohumeral joint.

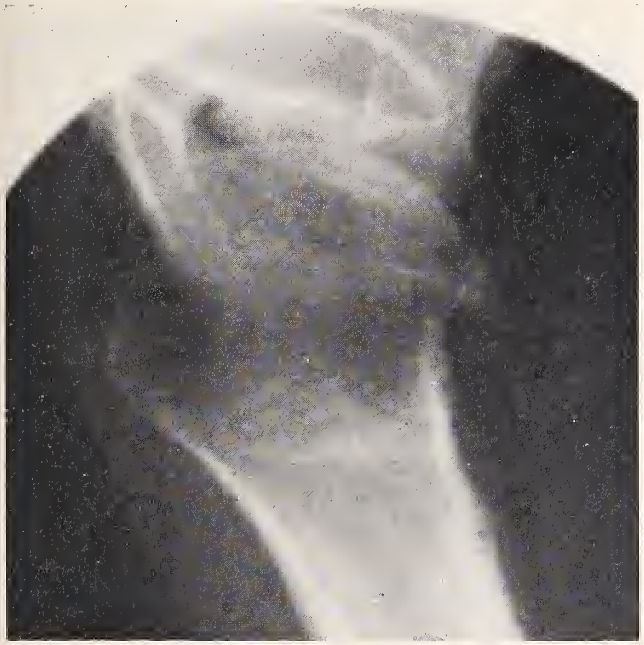

Fig. 2. Axial view showing normal relationship of the head of the humerus to the glenoid. Arthritic change is again noted along the articular surface of the humerus.

Discussion

Birth trauma to the shoulders may result in a deformity that is somewhat similar, though several differentiating points may be made. Scaglietti (1938) reviewed 199 cases of obstetrical injuries to the shoulder, and found the disability to be due to joint trauma in the majority of cases, rather than from nerve injury. He demonstrated fractures through the epiphyseal plate of the proximal humerus. Roentgenograms taken immediately after birth frequently failed to show evidence of fracture, the injury being apparent only on later examinations, after callus had been formed. This type of trauma resulted in shortening of the humerus, deformity of the head, limitation of motion at the shoulder joint, and partial posterior dislocation of the head of the humerus. This latter deformity is due to inward and backward rotation of the head fragment, following the epiphyseal separation. Frequently in these cases dysplasia of the inferior portion of the glenoid becomes noticeable at three to four years of age. In our case bilaterality of involvement, lack of posterior dislocation, absence of evidence of old epiphyseal separation, and normal length of the humeri all tend to rule out birth trauma as the etiologic factor.

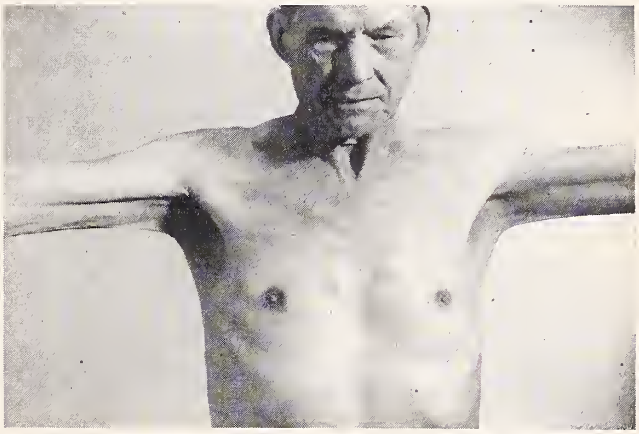

Fig. 3. Frontal view of the patient, with the arms abducted to their fullest extent. The webbing of the posterior axillary folds is well shown.

Fairbank (1947) described twenty cases of dysplasia epiphysalis multiplex, a congenital abnormality of multiple epiphyses resulting in dwarfism. In this condition the epiphyses show mottling and irregularity. Several roentgenograms included in his paper show deformity and apparent absence of the inferior glenoid rim. Fairbank does not believe this disease is hereditary or familial.

Five cases of bilateral glenoid hypoplasia have been reported by Owen (1953). He considered these cases congenital in origin, with no hereditary or familial factors involved.

Two cases of congenital abnormalities of the shoulder were described by Andreasen (1948). In these patients the humeral heads were flattened, and the articular surfaces of the glenoids were convex. He also reviewed six cases reported by Lewin, Muller and Brailsford, all quite similar, with concave humeral heads and convex glenoids. He believed the primary defect to be failure of normal development of the humeral capital epiphysis.

Brailsford (1948) reported three cases, showing a bilateral defect in the formation of the glenoid. These occurred in two women and one nine-year-old girl. In these cases, the glenoid was flat and had no lower lip. He believed the etiology to be failure of development of the epiphysis for the lower rim of the glenoid. His

(Continued on Page 596)

Current Concepts

SURGICAL TREATMENT OF CORONARY ARTERIAL DISEASE

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Since an editorial is, by definition, an article giving the views of its author, it would appear permissible to write one on the surgical treatment of disease of the coronary arteries. At the present state of knowledge, it would not appear advisable that a definitive report be written concerning this subject. In my opinion, the data available with regard to operative procedures in both experimental animals and man are not sufficient to allow final conclusions as to the efficacy of operations for coronary arterial disease. This is not to deny the merits of the publications made by the various workers who have contributed so greatly to this field; it is merely to emphasize that conclusions concerning the value of operations for coronary arterial disease must be considered tentative in the light of the incompleteness of the material so far available.

Coronary arterial disease may be considered as a form of segmental arterial occlusion in which the occlusion affects the vital left and right coronary arteries or their branches. These occlusive changes, either acute or chronic, result in ischemia of portions of the cardiac muscle. Secondary myocardial changes may stem from this deprivation of blood supply. Although the myocardial changes may be severe or even fatal, the occluded coronary artery itself remains the basic problem. Therefore, surgical efforts in this disease must be directed toward improvement of the blood supply to ischemic parts of the myocardium if they are to be done with a view to cure. Palliative surgical measures performed in the hope of relieving distress must not be confused with procedures aimed at producing an increased blood supply to the heart.

Many methods have been used by surgeons throughout the years in an attempt to improve blood flow to the heart in patients who have disease of the coronary arteries. Attempts have been made to stimulate the ingrowth of myriads of extremely small vessels by the production of pericardial adhesions or of adhesions between the heart and such organs or tissues as the pectoral muscle, the omentum or isolated loops of intestine. The implantation of the internal mammary artery into the ischemic myocardium recently has been proposed as a more direct approach to the problem of bringing arterial blood into the cardiac muscle. Anastomosis between the aorta and the partially occluded coronary sinus is another method of attack on the same problem. As the result of experimental studies, little doubt remains concerning the ingrowth of new vessels from either adhesions or implanted vessels.

A critical question remains: Do these procedures truly increase the blood supply to the myocardium and by virtue of this increased blood supply relieve the pain of angina pectoris and prolong life? Opinions that these operations do effect such changes can be found expressed in the literature. It is of great importance to the surgeon to arrive at his own opinion as to the merits of these operations. He must constantly see patients with coronary arterial disease who seek relief, even though it be by a major operation, from the burden imposed by the occlusive disease of the coronary arteries. It is not sufficient that the surgeon offer an operation to the patient. He must, indeed, advise the patient either for or against a surgical attempt to improve the blood supply to the myocardium.

It is to be admitted that the evidence to date is not conclusive as to the value of such operations. The surgeon must balance this against the known mortality rates of the various operative procedures. If in his opinion the chances of obtaining significant benefit from the operation justify the mortality rate involved, he may wish to advise patients under his care to submit to such operations. If, on the other hand, he remains unconvinced from the evidence presented that operations for disease of the coronary arteries are significantly prolonging the life and increasing the comfort of the patients receiving this type of treatment and particularly if he feels that the mortality rates from these operations are higher than are acceptable for a procedure that is not of proved value, then he probably will advise patients not to have operations for coronary arterial disease at the present time but to seek management of the disease by other methods of approach. Research into the surgical treatment of this common affliction continues and further evidence may cause the surgeon to alter his own opinions on this matter.

From the Section of Surgery, Mayo Clinic, Rochester, Minnesota.

Laboratory Aids

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George G. Stilwell, Editor

BLOOD COAGULATION

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Much research has been devoted to the process by which blood coagulates. Because the techniques and vocabulary of coagulationists are not familiar to many clinical pathologists and to most practitioners, publications in this field remain practically unnoticed in specialty journals. When the pathologist does set out to perform tests for "prothrombin consumption," "prothrombin utilization" or "thromboplastin generation," or "P and P" tests, he finds the reagents difficult to prepare and unstable once prepared, and he finds the methods capricious. So he returns to the performance of clotting and bleeding times and the "prothrombin-time" test of Quick, thus offering no more than he did fifteen years ago. This is unfortunate, since the characterization of coagulation defects appears to be on a sounder basis than ever before. Recognition of these defects is usually not too difficult, and the patient's need for fresh or stored blood or for vitamin K often can be anticipated by the laboratory.

First Step of Clotting

Clotting may be divided into four steps. The trigger mechanism apparently is an interaction between certain substances from platelets and several factors in plasma. Hemophilia and its clinically indistinguishable variants lack this triggering device. In hemophilia, a specific plasma protein (antihemophilic globulin, or AHG) is lacking or is blocked by an inhibitor. Coagulation is inefficient in either case despite normal concentration of the other clotting factors. In PTC deficiency, the missing factor is "plasma thromboplastin component." If plasma lacks a third protein, namely "plasma thromboplastin antecedent," another disease (PTA deficiency) like hemophilia results.

Hemophilia is transmitted as a sex-linked recessive, that is, from the grandfather through the mother to the son. It can occur in the female only if both parents transmit the gene, which is extremely rare. PTC deficiency, a sixth as common as hemophilia, also is a disease of males in alternating generations, apparently transmitted as a sex-linked recessive. PTA deficiency occurs in either sex and in successive generations; it is probably carried by a dominant gene and is the least common of the three congenital diseases affecting the initial step of clotting.

Surprisingly small amounts of fresh normal blood or plasma will correct *in vitro* the error in hemophilia. Correction is progressively less efficient as blood is stored for a number of days. However, stored blood corrects the prolonged clotting time of blood deficient in PTC. Treatment of fresh normal plasma with chemicals, such as barium sulfate or calcium phosphate, destroys the effect of plasma on PTC-deficient blood without altering its effect on hemophilic blood. Both stored and chemically treated plasmas correct PTA deficiency (Table I).

TABLE I.

Patient's abnormality is:	Clot time of patient's whole blood or plasma is shortened (+) or not (−) by addition of:			
	Fresh normal plasma	Stored normal plasma	Normal plasma treated with $BaSO_4$	Normal serum
Hemophilia	+	−	+	−
PTC deficiency	+	+	−	+
PTA deficiency	+	+	+	+

It requires only a little ingenuity to prepare these plasmatic reagents and distinguish the types of hemophilioid abnormality by determination of the effect of such agents on the clotting times of whole blood or plasma. The importance of such distinction is that fresh blood or plasma is vital in the treatment of hemophilia, whereas stored blood is satisfactory for treatment of deficiencies of PTC and PTA.

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This is the thirtieth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

Second Step of Clotting

During the second step, active thromboplastin is developed as the result of interaction of the products of the first step with two additional proteins in plasma. In man, one of these proteins (labile factor) disintegrates rapidly with storage of blood. The second protein (stable factor) resists storage and, like PTC, is readily removed from plasma by barium sulfate.

Characterization of deficiencies of factors in the second step of clotting is simple. If either labile or stable factor is reduced, the clot time as determined by the Quick test is prolonged. Despite the similarity of labile factor to AHG and of stable factor to PTC, results of the Quick test usually are normal in the hemophilic group of diseases.

Congenitally, labile factor is reduced in parahemophilia. This disease can affect both sexes in consecutive generations, and its inheritance has certain of the genetic properties of a dominant gene. In certain diseases, stable factor also may be lacking on a genetic basis; just as in parahemophilia, this characteristic is an incomplete dominant and is not sex-linked.

The commonest error of coagulation is acquired deficiency of stable factor. This factor is reduced primarily, and prothrombin is reduced secondarily, by use of bishydroxycoumarin (dicumarol) and related drugs. Insufficiency of stable factor is the primary defect in deficiency of vitamin K and in hemorrhagic disease of the newborn. In advanced hepatic disease, both labile and stable factors, as well as prothrombin, tend to decrease, and eventually the concentration of fibrinogen also may be reduced. In all these conditions, the clot times as measured by the Quick test are lengthened.

Differentiation of abnormalities of labile and stable factors merely involves performance of the Quick test on the patient's plasma with added stored plasma or plasma treated with barium sulfate (Table II). If stable factor is reduced, a normal-appearing plasma leads one to suspect administration of a dicumarol type of drug, whereas if the plasma is icteric, obstructive jaundice or severe hepatic damage comes to mind, the differentiation being made by the patient's response to use of vitamin K; if the plasma is water clear, the presence of sprue or other enteric diseases that prevent absorption of fats and fat-soluble vitamins, such as vitamin K, might be considered.

Third and Fourth Steps of Clotting

The third step involves conversion of prothrombin to thrombin under the stimulus of the thromboplastin produced during the first two steps. Depletion of prothrombin alone is perhaps the rarest of clotting abnormalities. When it is lacking, an accompanying deficiency of stable factor almost invariably is present. The final step of clotting, and the only one visible, is the enzymatic (thrombin) conversion of soluble fibrinogen to insoluble fibrin.

TABLE II.

Patient's plasma lacks:	Clot time (Quick test) of patient's plasma is shortened (+) or not (−) by addition of:			
	Fresh normal plasma	Stored normal plasma	Normal plasma treated with $BaSO_4$	Normal serum
Labile factor	+	−	+	−
Stable factor	+	+	−	+

Congenital afibrinogenemia has been found in both sexes in successive generations, but it is probably transmitted by a recessive gene. Acquired deficiencies of fibrinogen recently have received much attention. In certain conditions, notably prostatic carcinoma and hypoxic shock, the blood appears to clot well but the clot tends to disintegrate or become lysed. In such cases, an active clot-destroying enzyme, namely, fibrinolysin, usually can be found in the blood.

Another condition is being reported clinically as "afibrinogenemia." This is a severe, even fatal, bleeding tendency seen at times in patients with abruptio placentae. The coagulationist is puzzled by the clinical diagnosis of "afibrinogenemia" because he may find a practically normal concentration of fibrinogen in the plasma associated with a potent fibrinolysin. In other instances, when fibrinogen is actually lacking, a deficiency of most of the other clotting factors, as well as of platelets, also exists. It appears much as if the patient's erythrocytes were suspended in serum rather than in plasma, probably because of absorption of clot-promoting substances from the placenta or uterus, with resultant intravascular coagulation.

Although the logical treatment in bleeding with abruptio placentae is use of whole blood or plasma, the diagnostic term "afibrinogenemia" has led

(Continued on Page 589)

HEXAMETHONIUM-INDUCED HYPOTENSION

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Induced or controlled hypotension has met with both great favor and disfavor among surgeons and anesthesiologists since its introduction by Gardner in 1946.[1] Later proponents have advocated the procedure for operations of such diversity as skin grafting, fenestration, sympathectomy and craniotomy. They have pointed especially to the advantages achieved in surgical exposure, reduction of the operative and anesthetic course, and diminution of blood loss. Certain surgical procedures which heretofore were impossible now have achieved actuality.[2]

Without doubt, deliberate hypotension does have these several virtues. However, the most casual survey of the current literature convinces the reader that the procedure may increase the morbidity and mortality rates beyond those which existed before its use. In response to a survey questionnaire in 1953,[3] British clinicians reported 21,125 cases of controlled hypotension. Of these *reported* cases, 553 major complications (3 per cent) and forty-six deaths (0.2 per cent) implicated the technique. Further, since there was approximately a 50 per cent response to the questionnaire, these statistics probably did not reflect the true number of complications attendant upon the use of this method.

The fact that surgical exposure is improved in certain operations associated with induced hypotension might serve as a strong indication for its use, but only if the added risk is fully appreciated. While the operative and anesthetic courses are appreciably shortened, little information is available concerning the role the *duration* of anesthesia and surgery *per se* plays in increasing the anesthetic mortality and the postanesthetic morbidity or mortality. The availability of whole blood for transfusions has not presented too great a problem in this locality, but it is conceivable that in some communities the lack of stored whole blood might limit the type and extent of necessary surgery.

Since purposeful hypotension carries with it certain risks and benefits peculiar to the technique, a series of studies were undertaken to examine more exactly the alterations which occur in various physiologic processes when anesthetized man is exposed to hexamethonium-induced hypotension. An analysis of such information should delineate some of the advantages, disadvantages, indications and contraindications more clearly. The following discussion is based largely upon the conclusions of the investigations completed at the University of Minnesota.[4]

Effects on the Cardiovascular System

Arterial Blood Pressure.—Within thirty seconds after a single intravenous injection of hexamethonium, the blood pressure began to fall, and it reached its maximum depth within five minutes. The duration of action of a 25 mg. intravenous dose varied from fifteen to fifty minutes. Twenty-five to 50 mg. of the ion produced an average systolic fall of 48.8 per cent and an average diastolic fall of 44.2 per cent below the pre-injection level. The degree of hypotension obtained appeared to be a function of several variables which are common to most operative procedures. Among those to be considered specifically were the dosage of hexamethonium, the depth of anesthesia, the operative position of the patient, the pre-anesthetic blood pressure level and the presence of shock.

The evidence indicated, for example, that there was a point beyond which the blood pressure could not be reduced by additional dosages of hexamethonium. While the maximum effective intravenous dosage appeared, on the average, to be in the vicinity of 50 mg. of the ion, greater quantities of the drug merely prolonged the duration without increasing the degree of the ensuing hypotension. Such findings were consistent with the accepted theory embodying chemical blockade of the effector apparatus in the autonomic nervous system. They demonstrated clinically the achievement of maximum pharmacologic effects without the appearance of toxicity symptoms.

The extent of the induced hypotension was proportionate to the depth of anesthesia. When a rise in blood pressure occurred in the lightly anesthetized patient who was refractory to additional doses of hexamethonium, the blood pressure diminished to the initial hypotensive level when the depth of anesthesia was increased. Correspondingly, this effect was reversed as the patient recovered from anesthesia. Such a reaction might be ascribed either to a reduction in the tone of the skeletal muscles surrounding the vascular bed, or to a depression of the central vasomotor apparatus by the anesthetic agents, or perhaps to a combination of these events.

Induced hypotension also could be augmented if the patient was placed in Fowler's position. The angle the torso assumed to the horizontal directly influenced the blood pressure response. Thus a change from the supine position to 10 degree Fowler's position further decreased the average systolic blood pressure 22.6 per cent and diastolic pressure 15.3 per cent. A change from the

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Inaugural thesis presented before the Minnesota Academy of Medicine, November 10, 1954.

supine position to 15 degree Fowler's position decreased the average systolic pressure 39.7 per cent. This was most probably the effect of gravity upon a relatively inert vascular bed.

Next, the percentage fall in blood pressure concomitantly appeared as a direct function of the pre-anesthetic blood pressure level. The normotensive patients had an average fall in systolic pressure 36.2 per cent, while among the hypertensives the average systolic fall was 46.4 per cent. In like manner, the average diastolic fall in the normotensive group was 37 per cent, while in the hypertensive group the average diastolic fall was 42.6 per cent. Individuals with symptoms and signs of increased intracranial pressure experienced systolic drops averaging 48.9 per cent and diastolic falls averaging 47.0 per cent following the administration of hexamethonium.

Venous Pressure.—The initial effect of hexamethonium upon the venous pressure was dependent primarily upon the position of the patient at the time the drug acted. Fowler's position favored a fall in venous pressure, while the supine or Trendelenberg positions predisposed to rises in venous pressure. Once hexamethonium attained its maximum effect, the subsequent venous pressure variations closely paralleled those of the arterial pressure. It was not surprising to find that factors which modified arterial pressure modified venous pressure in the same direction.

Circulation Time.—Alterations in circulation time assumed an inverse relationship to the blood pressure changes in these patients. The arm to ear circulation time was fourteen seconds in one individual with an initial systolic blood pressure of 174 mm. Hg. Following the administration of hexamethonium, his systolic pressure fell to 66 mm. Hg and the circulation time nearly doubled its original value. This result was consistent with the data on changes in cardiac output and peripheral resistance.

Cardiac Output.—Cardiac output studies were performed on twelve patients in the horizontal supine position in the following manner. General anesthesia was induced and maintained with the standard pentothal-curare-nitrous oxide technique. Studies were withheld for a period of twenty minutes in which time the investigator could monitor the vital signs and be reasonably certain that a steady state of cardiovascular function was present. Cardiac output was then estimated by the Hamilton dye method and simultaneous blood pressure and pulse measurements were recorded. After these determinations, hexamethonium was administered intravenously in incremental doses until a systolic blood pressure level of from 70 to 100 mm. Hg was attained. At this point, a second estimation of cardiac output was made together with blood pressure and pulse measurements. Because of the experimental nature of these investigations, care was taken that a severe drop in systolic pressure did not occur. Although the technique of controlled hypotension often involves blood pressure levels lower than those produced in this segment of the study, the additional value of the data obtainable by producing extreme hypotension did not justify the added risk involved.

The results of these studies are shown in Table I. Hexamethonium-induced hypotension produced an average decrease in cardiac output of 15.27 per cent in the anesthetized individual.

Electrocardiographic Changes. — Purposeful hypotension produced electrocardiographic signs typical of myocardial hypoxia, and the severity of the signs was a function of the degree of hypotension. Similar electrocardiographic signs of myocardial hypoxia were produced at blood pressure levels approaching normality, but were achieved by diminishing the amount of oxygen in the inspired anesthetic mixture. Further, the signs evoked by either hypotension and/or atmospheric hypoxia could be terminated readily by increasing the oxygen concentration in the inspired anesthetic mixture.

It was significant that the tracings obtained after four minutes of atmospheric hypoxia (10 per cent oxygen) with a blood pressure of 80 mm. systolic and 60 mm. diastolic were not as abnormal as the tracings obtained with an inspired oxygen concentration of 50 per cent and a blood pressure of 65 mm. systolic and 55 mm. diastolic.

Cardiopulmonary Phenomenon. — Positive atmospheric pressure-artificial respiration, with an inspiratory phase equal to one-third of the respiratory cycle, had very little effect upon the arterial pressure of patients under hexamethonium blockade. A 5 mm. Hg systolic excursion occurred when respiration was augmented. Diastolic excursions were altered even less. Constant positive intrapulmonic pressure of a moderate degree (6 mm. Hg) caused little change in the arterial pressure, while positive intrapulmonic pressure of a high degree (15 mm. Hg) produced a marked decline in arterial pressure with complete obtundation of the pulse pressure. Release of this sustained pressure evoked a gradual return of the blood pressure to the initial level without overshoot.

Atmospheric Hypoxia and Hypercapnia.—An inspired oxygen concentration of 10 per cent (80 mm. Hg) for a four-minute interval induced a further gradual fall in the blood pressure of individuals under the influence of hexamethonium from 85 to 74 mm. systolic and from 65 to 57 mm. diastolic. Administration of 100 per cent oxygen restored the blood pressure to its pre-hypoxic level.

A five-minute hypercapnic period produced no change in blood pressure, in spite of the fact that the alveolar carbon dioxide concentration attained a level of 18 to 20 per cent (as measured by the Nier mass gas spectrometer). These observations are the distinct converse of those observed in anesthetized individuals who are not given hexamethonium. In the latter, hypoxia and/or hypercapnia produced an initial rise in blood pressure.

Altered Response to Peripherally Acting Vasoconstrictors.—Individuals under hexamethonium blockade have a markedly increased response to adrenergic agents, paral-

TABLE I.

EFFECT OF C_6 ON CARDIAC OUTPUT

AGE	DOSE OF C_6	BLOOD PRESSURE		PULSE RATE		C.O.		CHANGE in %
		PRE.-C_6 B.P. in mm.Hg	POST.-C_6 B.P. in mm.Hg	PRE.-C_6 PULSE (beats/min)	POST.-C_6 PULSE (beats/min)	PRE.-C_6 C.O. L/min	POST.-C_6 C.O. L/min	
55	25	110/70	75/	82	76	2.4	2.0	-18%
48	75	125/85	80/50	76	72	4.8	4.0	-18%
54	175	130/80	100/	78	74	7.39	4.25	-42%
29	25	118/75	80/	106	92	2.91	2.85	-2%
22	125	120/80	105/	100	84	3.1	2.8	-10%
46	75	145/90	90/60	88	80	3.8	3.17	-16%
30	50	120/80	80/	84	82	3.0	3.0	0
27	75	120/75	100/70	78	74	5.0	5.8	+16%
47	50	130/80	90/	72	74	3.9	3.0	-23%
34	* 25	90/70	70/	84	88	2.0	1.58	-21%
55	50	130/75	85/	80	74	2.71	1.79	-33%
11	75	130/65	85/40	72	96	2.55	2.13	-16%
Average, 38.2	68.75	122.3/77.1	86.7/-	83.3	80.5	3.63	3.03	-15.27%
	* VERY RAPID INDUCTION CAUSED BLOOD PRESSURE CRASH BEFORE C_6 GIVEN.							

lelling the increased sensitivity to these agents seen in other sympathectomized individuals. Since hexamethonium virtually produces a chemical sympathectomy, any vasopressor administered while an individual is under hexamethonium blockade should be diluted and administered in increments about one-fourth the usual therapeutic dose until the desired blood pressure level is obtained.

The exaggerated vasopressor response was observed in one hexamethoniumized patient who was given 4 mg. of methoxamine hydrochloride (Vasoxyl®) intravenously. Within twenty seconds, a violent pressor response occurred, and the pressure rose from 110 to 340 mm. systolic and from 90 to 270 mm. diastolic.

Effects on the Central Nervous System

Anoxia Photometer Readings.—Changes in arterial pressure did not alter ear oxygen saturation significantly in patients under hexamethonium blockade *unless* the patients were tilted to Fowler's position. When this maneuver was executed, oximeter readings fell significantly and consistently. Similar falls in oximeter readings occurred when high continuous positive intrapulmonic pressure was applied for three minutes.

Cerebral Arteriovenous Oxygen Differences.—Cerebral arteriovenous oxygen differences [C(A-V)02] increased significantly and consistently after hexamethonium when compared to pre-administration controls. Such increases can be returned to pre-hexamethonium levels by correcting the hypotension with vasopressors.

The *average* pre-hexamethonium arterial blood pressure was 147.5 mm. systolic and 99.5 mm. diastolic, the pulse rate per minute 97 and the C(A-V)02 6.85 volumes per cent. Following the administration of hexamethonium, the average arterial pressure fell to 63.5 mm. systolic and 46.6 mm. diastolic, with a rise in pulse rate to 115.5, and a rise in the C(A-V)02 to 8.55 volumes per cent. Upon abolishing the hypotension with vasoxyl, the average pressure rose to 120 mm. systolic and 84.5 mm. diastolic, the pulse rate fell to 91 and the C(A-V)02 returned to 6.67 volumes per cent.

Electroencephalographic Tracings. — Electrocorticograms have been run in synchrony with arterial pressure tracings in an indirect attempt to determine the extent of cerebral hypoxia, if any, during induced hypotension. Cortical activity, as manifested by the electrocorticogram, progressively diminished as the blood pressor declined. In one case when the arterial pressure reached 55 mm. systolic and 45 mm. diastolic, all activity ceased. When a vasopressor was administered at this point, cortical activity re-appeared with the rise in blood pressure.

Discussion

The foregoing findings indicate that hexamethonium chemically denervates all structures under autonomic nervous control. Under such denervation, all reflexes having autonomic components as their efferent limbs are blocked, thus abolishing reflex-compensating mechanisms involved in normal circulatory homeostasis. The conversion of a patient's vascular bed from a high pressure system to a low pressure system is, no doubt, the result of dilatation of both the active vascular beds and the reservoirs. This increase in the capacity of the vascular bed will permit a pooling of blood in the more dependent portions of the body while the more elevated parts become ischemic. Of primary concern, then, is the alteration which occurs in the physiology of vital organs, namely the brain, the heart, the liver and the kidneys.

The Brain.—The ear oximeter tracings, C(A-V)02, electrocorticograms, and visual detection of cerebral cyanosis all indicate a marked decrease in the rate of cerebral circulation, particularly when the patients are placed in Fowler's position. Stagnant hypoxia must be the explanation for these unfavorable changes. The development of cerebral thrombosis could easily be accounted for by the reduction in the cerebral blood flow rate.

The Heart.—The reduction in blood pressure, heart rate and cardiac output should result in a decreased coronary blood flow. Simeone[5] showed that the coronary arterial blood flow as measured from the outflow at the coronary sinus was decreased by 33 per cent in the patients studied. In the presence of a diseased coronary artery system, a reduction in the flow rate through it could lead to coronary thrombosis. Doubtless, the work of the left ventricle is decreased during the hypotensive state and, as a result, the oxygen requirement of the heart should be less, yet electrocardiographic studies revealed changes consistent with myocardial hypoxia.

The Liver.—Mueller[6] has shown that the rate of hepatic blood flow is decreased significantly during purposeful hypotension. Associated with this was a decrease in the hepatic vein oxygen tension. Splanchnic oxygen consumption and vascular resistance did not change significantly.

The Kidneys.—Anesthetized patients subjected to purposeful hypotension develop marked oligemia or anuria. Glomerular filtration rates have been shown to be markedly reduced. Intravenous phenolsulfonphthalein administered at the onset of hypotension did not appear until five hours later. Certainly, the risk of renal failure must be considered another potential complication of hypotensive procedures.

The weight of evidence indicates that the induction of hypotension adds considerable risk to the anesthetic and surgical management of the patient. Although purposeful hypotension is a valuable addition to the anesthesiologists' armamentarium, the risk involved must be very carefully calculated in terms of the surgical procedure to be accomplished on a specific patient.

Conclusions

1. The action of hexamethonium on the arterial and venous pressures is presented. A single 25 mg. dose of hexamethonium produced an average fall of 48.8 per cent in the systolic pressure and 44.2 per cent in the diastolic pressure. Factors which influence the magnitude of this effect are enumerated and discussed. Preexisting hypertension, deep anesthesia, Fowler's position and shock exaggerated the pressure drop produced by hexamethonium. The venous pressure tended to parallel the arterial pressure except on induction of hypotension and during positive intrapulmonary pressure.

2. Circulation times, C(A-V)02 and anoxia photometer studies were correlated with the arterial pressure. Each of these tests provided evidence of circulatory impairment incompatible with good tissue blood flow.

3. Electrocardiographic and electroencephalographic studies were done for the purpose of detecting heart and brain hypoxia during the hypotensive state. In both instances, evidence of severe hypoxia was present.

4. Alterations in the pharmacologic responses to hypoxia and hypercapnia, as produced by hexamethonium, were studied. Neither oxygen want nor excessive carbon dioxide produced a pressor response in patients with hexamethonium ganglionic blockade.

Purposeful hypotension becomes dangerous when the capacity of the total blood reservoir exceeds the blood volume since it is possible to render the brain ischemic by placing the patient in a steep Fowler's position.

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President's Letter

AMERICAN MEDICAL EDUCATION FOUNDATION

Some four or five years ago, the American Medical Education Foundation was founded under the sponsorship of the AMA. Since that time, about $7,000,000 has been raised to assist the financing of medical schools.

The original contribution by the AMA was one-half million dollars, as a sort of pump-priming project, hoping that the members of the Association would take it upon themselves to carry on this laudable project. Since that time the AMA has kept up this contribution for four years, until this last year when, due to budget deficiencies, the contribution was decreased to $100,000. Dr. Louis W. Bauer, president of the American Medical Education Foundation, stated in Atlantic City this year that it was now up to the medical profession or members of the Association to take up the slack so that the contribution to our medical schools would be as much as in previous years.

Medical schools do need more income. Although they are better supported today than any time in history, their operating costs have increased greatly. Do we want the government to subsidize our medical schools and so control them? Income from tuition constitutes only 19.5 per cent of the total operating budget, and it is estimated that it costs from $12,000 to $15,000 to train a physician, and yet the average student's total four-year tuition is only $2,532. We, as physicians, *do* owe a debt to our medical schools, aside from the fact that we must keep them from government domination.

At the 1955 session of the House of Delegates of the Minnesota State Medical Association, it was voted to place solicitation for this project at county society level, with the hope that more effective results might be obtained in raising our state quota. County society committees will be supervised by a state-wide committee headed by Dr. H. E. Drill. It is the hope of the House of Delegates that this method of solicitation will bring Minnesota's contributions up to the desired standard of other states.

Our individual duties in respect to this fund are emphasized by the fact that our own state medical school has received much more from the Foundation than has been contributed in our state. It was a revelation to me to note that other states have made such magnificent contributions and we have done so little here in our own state.

Let's all get behind this project. Let us willingly serve on, or aid our local county committees. Let us all give annually to the American Medical Education Foundation.

Arnold O. Swenson

President, Minnesota State Medical Association

Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

HEALTH EXAMINATIONS

Recently an official of a corporation made arrangements for an annual physical examination for a number of key men in his organization. Above everything else, he stated that the company would insist upon a complete gastrointestinal x-ray study because one of the company's officials in Ohio had died of cancer of the bowel not many months after a study which did not embody a specific roentgenologic search for the possibility of colon malignancy.

This naturally brings up the question as to what should be done to constitute a "complete examination"—how far should one go?

The latitude allowed in such examinations depends on the amount set aside for such a purpose on the patient's behalf and may be restricted to a physical examination, often with an electrocardiogram and a chest x-ray, and in some more extensive examinations, to what might be called "the works."

Some clients assume that a thorough study can be made with only an analysis of the history of the patient and an examination of the body by the physician with a few laboratory tests. Other examinees are not content unless a survey includes, in addition to these fundamentals, an electrocardiogram, x-rays of the chest, abdomen, complete gastrointestinal study and probably one of the urinary tract as well. It may be enlarged by certain blood chemical studies, a metabolism test and consultations with various specialists such as the ophthalmologist and an otolaryngologist on one end of the body, and the urologist and proctologist on the other. A minimal examination of the type mentioned could be done for $10; the latter would represent an outlay of probably up to $150 per patient.

Unfortunately, the patient believes that a favorable report is carte blanche to health, but every physician knows from experience the fortuitous errors which may arise in any examination, and chagrin and haste to explain do not mollify the patient or his family when disaster follows up to a year after a "thorough" examination.

A good physician naturally wonders as to what he may do to give the patient confidence in the health survey which is so constantly urged in our periodicals, and how efficient the results may be.

Since the examination is based much upon the capacity to pay, whatever is done should be done as conscientiously as possible. I believe that prefatory to any examination, a thorough history is of exceeding importance. It not only offers leads to follow in conducting the physical examination, but also yields information relative to the personality and environmental factors which are also of utmost importance. Especially is the latter true because, to the patient and even to many physicians, the physical possibilities seem paramount.

Another most important element is the thoroughness of the physical examination by the physician, aided possibly by an examination in the more specific specialties. With these elements as the basis of an examination, laboratory data and the desired electrophysical methods may be added, the latter depending upon the apparent need in the judgment of the physician.

When the patient is dismissed and is informed concerning all abnormal findings and conclusions, and advice is given as to their correction and relief, the patient frequently says, "So I'm all right, doctor, everything else is O.K. with me." The examining physician who knows in his heart that no examination and no series of tests are infallible, should tell the patient in all honesty that so far as he can tell, with the information at his disposal and the examination, conditions may appear entirely normal and satisfactory, but that illness may always develop by chance and that no examination can be considered proof against inadvertent accidents of medicine. On the other hand, if a physician makes a good survey of a patient, he can assure him by pointing out that after an examination is completed and no gross abnormalities found, he may be considered quite free from the possibility of early disaster.

F. J. Hirschboeck, M.D.

THE SPECIALTY OF OTOLARYNGOLOGY

Probably no specialty has benefited more from the impact of the sulfanamides and antibiotics on its activities than has otolaryngology. Relieved of the burden of caring for a large amount of suppuration in the temporal bone, the nasal space, and the throat, the otolaryngologist is devoting greater activity toward the unsolved problems that are so numerous in his specialty, and the specialty is benefiting from this inquiry.

This inquiry, in turn, demands a broader base of preparation. That fact has led to a higher standard for certification. Today, preparation for the specialty requires a minimum of three years of graduate training in a residency. That has lessened the interest of those who would combine the practice of ophthalmology with otolaryngology. That combination was an American creation, and its existence probably delayed research in otolaryngology. However, that combination is disappearing as witnessed by the fact that there are no more combined residencies approved by the certifying boards. Some are concerned about a theoretical need for the combined practice of these specialties for small centers of population. This has been answered elsewhere. (Boies, L.R.: The future of otolaryngology. Laryngoscope, 62:709-721, 1952).

In recent years, there has been a noticeable dearth of applicants for residency training in otolaryngology. Probably one reason for this was the impression prevalent in some areas that the sulfonamides and antibiotics had so altered the opportunities in the specialty that there was very little work left.

Applicants are now more numerous. The number of approved residencies has been reduced by eliminating institutions where the faculty and teaching material are inadequate for the broad training in otology, rhinology, laryngology and broncho-esophagology that the specialty demands.

Today, there is a large demand for the services of one who is adequately trained.

Opportunities for continuation study beyond the residency stage are as numerous in otolaryngology as in any other specialty. There is easy access to the instructional courses and conferences of the American Academy of Ophthalmology and Otolaryngology offered annually on a week-long basis. The methods of graduate teaching by this organization have been widely copied in other specialties. Beyond the Academy, there are four additional special societies with years of background (American Laryngological, Rhinological and Otological Society, Inc., American Broncho-Esophagological Society, American Otological Society, American Laryngological Association) whose annual programs reporting research and clinical studies are open to the profession, and in recent years new groups have been established for the intensive study of specific problems such as allergy, otosclerosis, plastic procedures, etc.

Truly, there seem to be few specialties in which there is so much opportunity today.

Lawrence R. Boies, M.D.

LIFE INSURANCE IN A PHYSICIAN'S ESTATE PLANNING

The large insurance programs carried by many physicians indicate acceptance of insurance as the fundamental element of their estates. Life insurance possesses several features which make it the ideal foundation of a family protection program.

1. It eliminates the time element usually required for the accumulation of property. The insurance estate is created immediately upon the acceptance of an application by the insurance company and the payment of a premium.

2. The finest investment services in America safeguard the funds which underlie the life insurance contracts.

3. The distribution of the insurance estate, if payable to named beneficiaries, is accomplished without the delay or expense of probating. In addition, the insurance companies are willing to distribute the funds in regular installments, or hold them at interest for the beneficiaries, according to mutually agreeable modes of settlement. Furthermore, they guarantee the safety of all funds held in their custody, as well as a minimum rate of interest.

Insurance contracts are more wisely chosen if one will lay out a "blue-print" of the total insurance estate he wants ultimately to own, before he buys several uncorrelated policies. Then, in order to reduce the danger of uninsurability, it is well to acquire the entire program as soon as possible by the purchase of ordinary life or convertible term contracts. These can be changed later, as circumstances warrant, to forms with improved paid-up, endowment or retirement benefits.

Should the proceeds be distributed to heirs in lump sum or in restricted installments? There is no simple rule, the manner of settlement depending on personal and family conditions. Is the beneficiary a prudent person, or a spendthrift? Are the proceeds to be used to pay debts and taxes, or to provide regular income to dependents? The answers to such questions help determine the best mode of settlement.

Just as the lawyer should be employed in planning the descent and distribution of one's general estate, so should a competent life underwriter be consulted in arranging an insurance estate settlement plan. For the same reasons that one's will should be revised periodically, with the change of family conditions, likewise should the insurance program be enlarged or modified from time to time, in order that the distribution of the proceeds will be consistent with the insured's current family and financial situation.

Dean H. Field, C.L.U.
Chartered Life Underwriters

FUNCTION OF THE STATE INSURANCE COMMISSION

The Division of Insurance, headed by the Commissioner, is charged with the enforcement of all laws of this state relating to insurance. The department is divided into the following subdivisions: Rating, Agents' Licenses, Examining, Cashier and Accounting, Complaints—legal, Statistics. Each of these subdivisions or functions is headed by a supervisor.

The Commissioner of Insurance who also is the State Fire Marshal, has other duties to perform, such as being a member of the Compensation Insurance Board, a trustee of the Teachers Retirement Fund, a member of the Board of Public Employes Retirement Association, and a member of the Insurance Board. The Insurance Commissioner, along with the Banking and Securities Commissioners, acts upon all matters pertaining to the Department of Commerce. A portion of the Commissioner's time is also taken up with membership in the National Association of Insurance Commissioners.

The Insurance Department has thirty-three employes.

At the present time, there are licensed in the State of Minnesota 155 township mutuals and 690 other companies. Of the 690 companies, there are 84 domestic companies with assets totaling approximately three-quarters of a billion dollars.

There are 18,750 agents licensed to do business in this state, and the number of licenses issued to these agents totals 73,129.

All domestic companies licensed and the agents licensed in this state are under the direct supervision of the Commissioner of Insurance, who is charged with the responsibility of examining the insurance companies once every three years as to their financial standing and treatment of policyholders.

Companies other than domestic insurance companies licensed in this state are also indirectly under the supervision of the Insurance Commissioner.

Inasmuch as all companies are required to file an annual statement which must be sworn to by the principal officers of the company, examination and analyses of these statements are made by an examining staff each year.

The Insurance Department also participates in the examination of insurance companies operating in this state, even though they are domiciled and chartered by other states, thus protecting the policy holders of this state who purchase insurance in other than domestic carriers.

There are a number of insurance companies, not licensed in the State of Minnesota, which solicit the citizens of our state through the mail. The Insurance Commissioner makes every effort to advise the citizens of this state not to purchase any insurance in any unauthorized carriers.

The 2 per cent premium tax collected from the insurance companies licensed in this state amounts to in excess of 6 million dollars annually. The miscellaneous receipts, such as company fees, agents license fees, et cetera, total in excess of $207,000 annually, while the payroll and supplies and expenses to maintain the department each year total approximately $163,000.

Each year about 7,000 individuals visit the department for information regarding matters pertaining to insurance.

Cyril C. Sheehan
Commissioner, State of Minnesota
Insurance Department

THE PHYSICIAN AND THE DEATH CERTIFICATE

Completing and signing the certificate of death of a patient he has attended is about as unhappy a task as a physician can be called on to perform. It is, however, a legal duty he cannot avoid, and promptness in its performance not only facilitates the work of others but is a distinct service to the family of the deceased, as it is the purpose of this article to make clear.

When a family comes to a funeral director to make arrangements for a service, one of the first questions considered is the day of the funeral. Usually, the family prefers to have the service and interment within two or three days. Before the body may be interred, however, a permit must be obtained from the Registrar of Vital Statistics under the provisions of Sections 144.181, 144.183 and 144.164 of the Minnesota Vital Statistics Law, which require that a death certificate properly completed and signed by the attending physician (or the coroner where no physician was in attendance) be filed with the Registrar before the permit may be issued. This makes it necessary for the funeral director to obtain as soon as possible the signature of the attending physician to the death certificate.

Understanding co-operation between the physician and the funeral director will facilitate the work of both.

It is, of course, the responsibility of the funeral director to present to the physician a death certificate completed with the exception of the physician's diagnosis and signature. Many funeral directors feel that the doctors are uncooperative in this matter. Physicians are busy people, but if they will let the funeral directors in their community know the method they would prefer them to use in presenting death certificates for their signature, they will find the funeral directors only too glad to comply. If the usual practice of having a member of the funeral director's staff deliver the certificate to the doctor's office and wait until it is signed is felt objectionable, the certificate can be mailed to the doctor with a stamped, addressed return envelope. In such case, however, the doctor's assistant should be instructed to bring it to his attention promptly upon receipt and to see that it is promptly mailed after he has signed it.

It should be mentioned, in this connection, that when it is not possible for the doctor to state the cause of death definitely when the certificate is presented to him, he does not need to delay the signing of the certificate for that reason. Under the provisions of Section 144.164 of the Minnesota Vital Statistics Law, the attending physician may indicate on the certificate over his signature "diagnosis deferred," if the cause of death cannot be determined before interment or within five days, if interment is delayed.

In such case, the physician should enter in the medical certification portion of the death certificate—"diagnosis deferred," and sign and date it and return it to the funeral director. In due time, the State Registrar will provide the doctor with a form to be completed and returned to him so that the cause of death can be entered on the death certificate.

Besides their necessity for burial purposes, death certificates are important to the family of the deceased for other reasons. Among the purposes for which they are needed may be mentioned the collection of insurance, the cashing of jointly held bonds, the transfer of joint bank accounts, the conveyance of real estate held in joint tenancy, social security benefits, and veterans' benefits. Unnecessary delay in the completion and filing of the death certificate may obviously create hardship in the family. Correct, well-executed certificates are an oft overlooked source of good will for the years to come.

(Mrs. John) Dolores O'Halloran

BUSINESS PROSPECTS FOR 1955

The best phrase I have heard to describe 1955 is that it will be a year of competitive prosperity —in other words, a good year for those who recognize the need to compete for business with all the ability at their command. Indeed, it may turn out to be our No. 1 peacetime year.

As we look into the future, we see exciting potentialities for growth in the food processing industry. There is a strong trend toward the use of more prepared foods—enabling the housewife to have more time to spend on her home and with her family.

Nutritional research and flavor research offer us some of the most fascinating possibilities. The Nutrition Foundation, an organization for research financed by many of the food manufacturers, is today studying a wide range of subjects of nutritional importance. The same is true of a good many of our colleges and universities and government agencies. And, of course, companies like ours are continually studying nutritional needs and the effects of foods in meeting these needs.

We can assume that almost everyone enjoys food that is delicious, so it's a real consolation to know that *enjoyment of food is important nutritionally.* It is interesting that a good deal of attention is being given today to the basic nature of flavors and how to improve them.

As we learn more, we also learn that the scope for future development is broader than we imagined. Nowhere in the American economy is this observation more true than in agriculture and in farm yields. For example, in the 1940's we all thought that 10 tons of tomatoes per acre was a fine achievement. Today, 10 tons is only a fair crop. With what has been learned, and can be learned, about new plant varieties, new soil treatments, new sprays and new growing methods, it is not at all unrealistic to foresee an average of 20 tons per acre of redder more perfect tomatoes that taste better and have superior food values. The changes taking place in tomato growing parallel those for other agricultural products. What has happened in the past as to farm yields and what we believe is in store for us in the future tell something of the capability of the agricultural industry. I doubt that there is any cause to worry about feeding the growing population as far ahead as you and I can see—and a lot farther.

We are a rich and growing nation. Births last year reached a record high of 4 million. There are estimates that the population will hit 200 million by 1970. The potentialities for progress seem almost unlimited. Speaking for the company I know best, Campbell's business has been on an upward trend for a long time. If this trend continues—and there's every reason to think it will—the year 1955 should be the biggest and best in the company's history.

William B. Murphy
President, Campbell Soup Company

HISTOPLASMOSIS

(Continued from Page 537)

symptoms and signs due to infection with *H. capsulatum.* One of the children had fatal disseminated histoplasmosis. The results of a preliminary survey of skin sensitivity in the area of the site of the outbreak indicates that a point source of infectious spores exists there. In addition, the survey uncovered a higher incidence of reactors to histoplasmin than has previously been known to exist in this area. These data suggest that physicians in Minnesota should consider histoplasmosis in attempting to determine the etiology of certain pulmonary syndromes resembling atypical pneumonia. More information is needed concerning the rate of skin sensitivity to histoplasmin, as an index of the prevalence of the fungus in the state of Minnesota.

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Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

STATE SEVENTH HIGH IN OLD AGE ASSISTANCE

According to a recent bulletin from the Minnesota Taxpayers Association, the State of Minnesota is now seventh highest in the nation in payment for old age assistance. Only six states pay more for old age assistance than Minnesota.

The bulletin states that "the average payment to old age recipients for the country as a whole is $51.90 a month, while Minnesota's average payment is $65.12. Minnesota is $13.22 a month higher than the average for the country."

The Taxpayers Association also notes that if Minnesota's payments averaged the same as the country as a whole, the bill to taxpayers would be $8,261,013.36 a year less, based upon the figures for December, 1954.

Further figures show that one out of every six persons over 65 years of age in the state of Minnesota is receiving an old age pension. "The actual figure for Minnesota is 176 recipients out of each 1,000 persons over 65," the bulletin states.

The organization also points out that the proportion of old age pensions in the state is slightly less than the average for the country: The U. S. average is 183 per 1,000 persons aged 65 and over.

Among the midwest states that pay less than Minnesota are: Wisconsin, $62.44 per recipient; Iowa, $57.02; Michigan, $54.43; Ohio, $57.59; Indiana, $47.53; and Illinois, $59.69.

MORE FUNDS URGED FOR FDA

A special committee, which for five months has been investigating problems of the Food and Drug Administration, recommends that this agency have more money to work with, that it be authorized to hire more trained personnel, and that its administrative structure be improved. The above information is reported in a recent issue of the American Medical Association's "Washington Letter."

Staff Noted

The committee's staff, appointed by Secretary of Health, Education and Welfare, Oveta Culp Hobby, was under the chairmanship of G. Cullen Thomas, vice president of General Mills, Inc. Its membership included Dr. Harry Dowling, professor of medicine at the Illinois University; Dr. Leonard A. Maynard, director of the School of Nutrition at Cornell University; and Dr. Charles Franklin Poe, dean of the College of Pharmacy, Colorado University.

The committee's recommendations are:

"1. The FDA's personnel and facilities should be expanded three or fourfold, appropriations should be sufficient to finance the broader program, FDA should be given a modern building to house its Washington operations.

"2. An educational and informational program should be developed in the immediate office of the Commissioner. Its purpose should be a wider dissemination of more positive information to industry, the professions and the public.

"3. The FDA general counsel should have an 'adequate appropriation and staff' so legal actions can be 'prosecuted vigorously.' Actions should not be resorted to unless necessary.

"4. The headquarters staff should be strengthened to make possible better programming and internal management of FDA affairs."

VETS MEDICAL CARE TO GET NEW APPRAISAL

A recent issue of the "Washington Report on the Medical Sciences" notes that an eleven-state conference has been scheduled for September 27, in Indianapolis, in an attempt to "close the rift between the American Legion and organized medicine on medical care benefits for war veterans." The report states that Washington participation is assured.

The meeting will be sponsored by the Indiana State Dental and Hospital Associations, the Indiana Department of the American Legion and the state medical association's joint liaison committee. Invitations to attend have been extended to their counterparts in Illinois, Iowa, Kentucky, Ohio, Michigan, Minnesota, Missouri, Nebraska, Pennsylvania and Wisconsin. Also, to managers of 54 veterans hospitals in those states.

Guest Speakers

Prominent persons in all phases of this particular problem are slated to talk to the conference. From Washington, program speakers will include

Dr. William S. Middleton, chief medical director of Veterans Administration; T. O. Kraabel, director of rehabilitation, American Legion, and Dr. Dallas G. Sutton, representing the American Hospital Association. Also, Dr. Elmer Hess, Erie, Pennsylvania, president of the American Medical Association, will speak to the conference. Dr. Norman R. Booher is to be chairman.

STOP ABUSING HOSPITAL INSURANCE

A recent article in *Reader's Digest* shows how "free-loaders" are driving up the cost of insurance, and it also shows how this abuse of hospital insurance can be stopped. The article is written by Blake Clark.

The article, available in reprint form from the AMA, notes several examples of gross abuse of hospital insurance. Among them is the story of a couple who wanted to go on vacation, but could not find suitable baby sitters for their children while they were away. Their doctor said, "That's easy—just bring them to the hospital." So the children were admitted to the hospital, the insurance company payed the bill and professional baby sitting was had for a period of two weeks, at considerable cost to the insurance company.

The article states: "Let's not fool ourselves: if we do not look out, we are going to kill the goose that lays the golden eggs that pay our hospital bills. This kind of abuse drives up the price of hospital insurance for *everyone*. Some hospital-insurance rates have already been raised six times in eight years."

The Michigan State Medical Association has set up a committee to look into this abuse. The committee examined 12,102 cases in 25 typical hospitals and found "faulty use" of hospital facilities by 31.1 per cent of the insured patients; only 12.4 per cent of the uninsured were at fault. Four kinds of abuse were revealed: overstay, admission for diagnosis only, hospitalization merely for convenience, and unnecessary treatments.

Who's to Blame?

The article says that doctors cause most of the trouble "by sending patients to hospitals for diagnosis and treatment which could be handled in the physician's office; by permitting patients to remain in hospital longer than is necessary; and by carelessly continuing expensive treatment when the need is past." However, it also states that physicians are not alone in the blame. The article notes:

> "Hospitals carelessly run up expenditures for unneeded tests and medications. And the patient himself is too often at fault. Typical is the patient who says, 'As long as I'm here, how about an electrocardiogram and an x-ray picture of my stomach?' His attitude is, 'Shoot the works, Doc; I'm covered.'
>
> "Insurance agencies themselves are not blameless. They permit advertising that says, in effect, 'We pay everything,' and picture a happy family saved a disastrous $3500 in hospital charges."

How to Control?

A job of controlling such abuses is being done in Cincinnati, the article goes on. Though hospital costs have skyrocketed as fast in Ohio as elsewhere, Cincinnati's largest hospitalization program has increased fees only once in the past five years and has added hundreds of thousands of dollars to reserves. They did it by making all parties involved aware of the consequences of faulty use.

James E. Stuart, executive director of Cincinnati's Hospital Care Corporation, recently told his physician friends: "We are all in this boat together, plan officials, subscribers, hospitals and you—but the steering is in your hands. You decide who shall be hospitalized, when and where he is to be admitted, how long he will stay and what services will be provided."

Also, county medical societies and hospital officials are now working together to try to lick the problem. When one hospital administrator saw that his institution spent two dollars more per day per patient than his neighbor, he made his staff find ways to bring costs into line. The article reports that when a doctor forgot to cancel expensive medication and ran up an unnecessary bill of nearly $1000, the group put into effect a ruling that all prescriptions must be renewed every three days or the drugs are automatically canceled. "This and similar controls have resulted in substantial savings," the article notes.

Further, Stuart and his group have made it easier for the physician to resist patients' requests for undue "co-operation," the article points out. It says:

> "A plaque prominently displayed in the doctor's waiting room outlines the services which Hospital Care Corporation does *not* cover. This gives the doctor official support when he must tell a member that hospital fees

solely for diagnostic study or checkup are not underwritten.

"Stuart estimates that enlisting the co-operation of doctors, hospitals and patients has resulted in savings of $1,500,000 a year for Hospital Care Corporation."

The article concludes: "The Cincinnati record shows that an honestly used hospital plan can give the protection it promises, without rate increases that sooner or later will make it too costly for those who need it most."

FEE ADVICE FOR YOUNG PHYSICIANS

Advice on the What, How and When of setting up fees, has been outlined recently for young physicians just starting practice. This article is found in a new publication of the Mead Johnson Company, called *Mead Digest*. The booklet is designed especially for residents and interns, but is applicable to the established practitioner as well.

Actually, the article states, "the fee problem need not be complicated if sound basic principles are followed from the beginning. According to leaders in the field of medical economics, you can handle the whole subject smoothly if you plan your action in three progressive steps:

"1. Establish a fair fee schedule.

"2. Inform the patient of the fee in the proper manner.

"3. Pursue a systematic collection system."

Regarding the proper fees to charge, the publication states that there is no easy answer because fees differ in various communities and in different parts of the country. The best way is to check with the county medical society and other physicians, the article advises young doctors. "Many medical societies have worked up informal average fee schedules which can serve as a guide to local physicians in setting their charges," it goes on to say.

The article quotes some tips from the AMA which have been set up to guide doctors on their fee schedules:

"Charge enough to command respect.

"Set fees consistent with the average patient's ability to pay.

"Adjust fees on a cost basis only.

"Don't put bargain rates on medical care; this tends to lower quality.

"Give the best medical service possible per medical dollar."

One of the most important things for a young physician to consider is the prior discussion of fees with the patient, and also sending itemized bills rather than lumping the charge in one.

Why Patients Don't Pay

Along this same line, the same issue of *Mead Digest* summarizes a recent survey of patients in California who had not paid their medical bills. These figures represent delinquent accounts only, not the physicians' entire practices. Out of the 1,560 patients surveyed, 19 per cent had not paid because of financial hardships at the time; 20.23 per cent had not paid because of poor business methods in the doctor's office, including no billing, insufficient information to trace "skips," statements not itemized, and no collection follow-up in the doctor's office; 3.4 per cent were dissatisfied with the medical services; 2.04 disclaimed any responsibility for the bill; 2 per cent thought the fee was excessive; 21 per cent had not been informed by the doctor of some of the financial arrangements, such as x-ray charges and other costs; 9.61 per cent were negligent and slow to pay bills; .86 per cent were classified as deadbeats.

The survey, made by the Alameda County Medical Society and the California Medical Association, noted: "When steps were taken to correct the above difficulties, collections increased as much as 25 per cent. What is more important, patient satisfaction increased proportionately."

"IDEAL" SOCIALIZED MEDICINE

(Continued from Page 546)

in all these peoples. Now is the time for the physicians to forget their personal desires and look to saving our present philosophy of freedom by aiding in the establishment of a program that supplies enough sufficiently soon—for if *we* do not, others shall be glad to head the paternalism!

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Miscellaneous

PROPOSED PLAN OF INSURED ENDOWMENT FOR THE AMERICAN MEDICAL EDUCATION FOUNDATION

Working under the assumption that the financial needs of medical education in the United States will continue to exceed the moneys normally available from regular income sources, (i.e., tuition, interest on investments, annual gifts from alumni and general funds of the university allocated for the operation of the medical school) a constant search has been maintained to find a method whereby the Foundation will be assured of a more permanent income to meet the medical profession's quota in the national campaign to raise $10,000,000 annually for medical education.

To this end, many fields have been explored; legislation at state levels to increase dues to meet the giving load, volunteer committees, direct mail efforts and others, too numerous to mention at this point. The plan outlined in the following paragraphs has many advantages and, from this observer's point of view, the advantages far exceed the disadvantages that might arise.

In selecting an income plan for consideration, several basic factors had to be considered; they were: (1) a plan that would automatically include the bulk of America's practicing physicians, (2) a plan that would benefit the Foundation as well as the individual participant, (3) one that would identify the medical societies with the effort and relieve it of the financial strain that the current campaigns placed upon their reserves, (4) one that would conceivably give the State Society something tangible to add to services being rendered to its members and, at the same time, make it more attractive to other physicians who might consider membership, and (5) create a steady flow of funds into the Foundation's treasury in excess of $2,000,000 annually, thereby permitting the Foundation to acquire a degree of permanence and build a hard core of liquid assets that could be invested with the returns used to further assist medical education.

It is believed that the following plan will accomplish all these basic needs and still remain attractive to the membership of the state and local medical societies.

It is suggested that the state societies consider the following insurance endowment plan as a possible means of creating a stable and continuing source of income for the American Medical Education Foundation. If found acceptable, the plan should be presented to the House of Delegates for their further consideration and action. I am of the opinion that a $1,000, twenty-year endowment policy on each member of the association can be written on a group basis, with no evidence of insurability requirements. Participation would be limited to active dues-paying members up to and including age sixty-five. This will allow the full twenty-year span on all members before attaining age eighty-five.

Assuming that we are working with an even number block of 100,000 eligible members at the inception date of the plan, this would immediately provide a $100 Million Dollar endowment fund.

During the first few years, we could expect approximately 2,000 deaths per year. Proceeds from these death claims, which would be payable to the State Society or the Foundation direct, would produce upwards of $2,000,000 per year in income to the Foundation. Of course, as this original group grows older, the death rate will increase, and we can estimate that it will eventually reach 3,000 per year, which would result in $3,000,000 per year being paid in claims to the Foundation. If we now strike an average of 2,500 deaths per year during the 20-year endowment period, we see that approximately $50,000,000 would be paid in death claims, and at the end of the period, the balance of $1,000 per living member would become payable in a lump sum as an endowment. This amount would be the other $50,000,000.

Thus, we see that during the 20-year period $1,000 will be paid to the Foundation on every life insured, either as a death claim or as an endowment at the end of the period.

Assuming that the average annual premium will work out at about $50 per $1,000, you will note that whereas the first year's premium would be approximately $5,000,000, this total annual premium would reduce each year as deaths occur, leaving fewer to pay for the succeeding year. While I am not an actuary, I believe that the total premium that might be expected to be paid during the 20 years would approximate *$80,000,000* but *$100,000,000 would be returned* either in death claims or endowments. This gain of $20,000,000 would come from the investment income made on the funds received each year by the insuring company. This is life insurance at work.

The increasing value of the funds on deposit with the insurance company is reflected in the increasing cash or loan value of each certificate and would grow each year as renewal premiums are paid. These funds would be available, for loan purposes, to the owners of the certificates, (State Societies) and the funds could be made available to individuals such as interns, residents, and young doctors who have finished their training and need help in getting set up in practice. Thus, the Endowment Fund not only provides the ways but the *means* for helping the young doctors who are a product of these schools. This feature, made possible by a plan of this kind, is very important since it brings into being a new, very vital activity for the State Society *and should stimulate membership.*

So far, I have dealt insurance wise only with the original group which has been estimated at 100,000, who would be eligible at the inception date of the plan.

Now, assuming that there are 6,000 potential new members going into practice each year, this would mean adding $6,000,000 new insurance each year, the bulk of which would be at the younger ages. This would *reduce* the average rate of the entire group and eventually bring the average age down into the thirties, and the rate down below the estimated $50 per $1,000 mentioned above.

As in the case of the original group, death claims would be paid as they occur on these new members. Since this group in itself, is younger and the mortality rate is lower, death claims will be fewer, resulting in a lower return per year to the Fund from death claims. However, this will result in more certificates maturing as endowment, thus increasing the amount payable at the end of the endowment period. As in the case of the first group, each certificate issued in subsequent years on these new members will pay off in full either as a death claim or as a matured endowment so that $1,000 is paid off on each life eventually, live or die.

Since it is proposed that this insurance be paid for out of state dues,* the premium as part of these dues would be tax deductible for the individual doctor. Now, if we can assume that the average doctor reached the 50 per cent Income Tax bracket, Uncle Sam will actually be paying one-half of the cost involved and instead of a $50 increase in cost of membership, there would actually be a $25 net increase in annual outlay on the part of the member doctor—which would appear to be a very insignificant amount to produce such terrific results.

Any further information you may need or want, I will be glad to develop for you. In order to establish a definite rate for the original group, we would need the following data:

(1) Distribution of present membership by attained age.

(2) Deaths in 1953 by attained age at death (can be lumped for ages over sixty-five).

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*Dues, for doctors who survive the 20-year endowment period of their individual certificate, could be reduced at the end of the endowment period. Twenty-year participation on the part of any one doctor is all that has been contemplated in this proposal.

PAINFUL SHOULDER

(Continued from Page 558)

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THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

SAINT PAUL WOMAN SENTENCED FOR ABORTION

Re: State of Minnesota vs. Assunda Willner, also known as Sue Willner

On July 11, 1955, Assunda (Sue) Willner, forty-five, 343 West Central Avenue, Saint Paul, Minnesota, entered pleas of guilty before the Hon. Ronald E. Hachey, Judge of the District Court of Ramsey County, to two informations, one of them charging her with the crime of abortion and the other charging her with having a prior conviction for a felony, and the Court continued the matter to July 18 for sentencing the defendant. Mrs. Willner had previously been convicted of the crime of abortion in Ramsey County District Court on October 9, 1947. When Mrs. Willner again appeared before Judge Hachey on July 18, she was sentenced to a term of not to exceed eight years in the State Reformatory for Women at Shakopee, Minnesota, the sentence being suspended and the defendant placed on probation on condition that she would depart from the State of Minnesota by midnight and remain outside of the state forever. The court also provided that the defendant must report to the Probation Officer in the place where she intends to reside and also that she must keep the Ramsey County Probation Officer informed in reference to her place of residence.

In sentencing the defendant, Judge Hachey stated that he had given a great deal of thought to the case and he had no difficulty whatsoever in reaching the conclusion that the defendant is a bad risk for ordinary probation since she had taken advantage of everyone who had ever given her a "break" in her entire lifetime. Judge Hachey added that when he was a prosecutor in the office of the United States District Attorney, he had experience with narcotic peddlers and "white slavers," but he had never had a case involving an abortionist, although he puts them all in the same class. After telling Mrs. Willner that she had reached in life what he considers the "low of lows", Judge Hachey said that he was only sorry that he could not sentence her to a term of ten or fifteen years, but he realized that if she was sent to the reformatory for the maximum term of eight years she would perhaps be paroled after a few years and that was too good for her. The Court warned Mrs. Willner that if she returned to the State of Minnesota after midnight, she would very likely have to serve out the full eight years in that event.

The abortion which was performed by Mrs. Willner on May 8, 1955, upon a thirty-two-year-old Minneapolis woman, who was married and the mother of three children but separated from her husband, came to the attention of the authorities when the patient began to hemorrhage as a result of the abortion and was taken to Minneapolis General Hospital. The defendant was paid the sum of $300.00 for the abortion, which she performed by means of a catheter. In addition to the prior conviction for abortion, Mrs. Willner has two convictions for practicing healing without a basic science certificate, one in 1942 and another in 1951. In the first case, the defendant was placed on probation, whereas in the 1951 case she was sentenced to serve sixty days in the Ramsey County Jail. Mrs. Willner holds no license to practice any form of healing in the State of Minnesota.

In Memoriam

WILLIAM PITT ABBOTT

Dr. William P. Abbott, former Duluth physician, was found dead in his home on Thursday, May 19, 1955. He had died on Tuesday or Wednesday. Dr. Abbott was seventy-four years old.

A resident of Faribault in his youth, Dr. Abbott attended Shattuck School. He graduated from the University of Minnesota Medical School in 1906 and entered general practice in Duluth in 1907.

Ill health forced him to retire from active practice in 1933, and he moved to a location near Grand Marais, Minnesota. At the time of his death, he was living alone with his dog. On Tuesday before his death, the dog was struck by a car and killed. Two days later, Dr. Abbott was found by a neighbor after the mailman reported that he had not picked up his mail.

Dr. Abbott had been a member of the St. Louis County Medical Society and the Minnesota State Medical Association.

A son, Agatin T. Abbott, a university professor of Moscow, Idaho, and a sister, Mrs. C. W. Newhall of Morristown, survive.

HERBERT BAKER AITKENS

On June 6, 1955, Dr. Herbert B. Aitkens, one of the early physicians in Le Center, died at his home. He had been a bed patient for two years. He was eighty-eight years old.

Born in Highcliff, England, Dr. Aitkens came to Canada when he was nineteen. After attending the University of Manitoba in Winnipeg, he attended medical school at the University of Minnesota, graduating in 1893.

In 1894, Dr. Aitkens located in Le Center. He was a board member of St. Paul's Episcopal Church and served on the school board for many years. In 1944, a banquet was held honoring him for his fifty years of service in the community. Four years later, he retired because of failing health.

He was past president of the Nicollet-Le Sueur County Medical Society and a life member of the Minnesota State Medical Association. In 1943, he became a member of the Association's "Fifty Club."

Mrs. Aitkens, the former Anna E. Barker, survives her husband. Dr. Aitkens also leaves a daughter, Mrs. A. G. Tuberg of Glendale, California, and two step-sons, Frank Aitkens of Minneapolis and Leonard Aitkens of Mamaka, Alberta, Canada.

WILLIAM HENRY AURAND

Dr. W. H. Aurand, eighty, a Minneapolis physician since 1901, died July 17, 1955. He was born at Green Bay, Wisconsin. He attended Minneapolis Central High School and the University of Minnesota, graduating from the medical school in 1901.

Dr. Aurand interned at St. Barnabas Hospital, where in 1938 he became chief of staff.

He was an active member of the Hennepin County Medical Society, serving on the Executive Committee and as a delegate to the Minnesota State Medical Association from that county society. He was a life member of the State Association and had been a member of the "Fifty Club" since 1951.

Dr. Aurand is survived by his wife, Jane; a son, Calvin, Des Moines, Iowa; and a sister, Mrs. Harry G. Slyder, Sioux Falls, South Dakota.

PAUL ARTHUR O'LEARY

Dr. Paul A. O'Leary, internationally recognized authority on diseases of the skin and syphilis, died in St. Mary's Hospital in Rochester, Minnesota, on July 20, 1955. Dr. O'Leary was born in Brooklyn, New York, on November 11, 1891, the son of Anna Belle Coy O'Leary and Jeremiah J. O'Leary. He was a student at Dartmouth College in 1910 and 1911, and at the Long Island College of Medicine, from which he received the degree of doctor of medicine in 1915. In 1915 and 1916, he was an intern in the Long Island College Hospital in Brooklyn.

Dr. O'Leary came to Rochester, Minnesota, on January 1, 1917, as an assistant in dermatology and syphilology in the Mayo Clinic. In August of that year he was appointed to the staff of the Mayo Clinic. From 1917 to 1919, he was a first lieutenant in the Medical Corps of the National Army. In May, 1924, when Dr. John H. Stokes left the Mayo Clinic, Dr. O'Leary was named to replace him as head of the Section of Dermatology and Syphilology. He retained that post until July, 1953, when he became a senior consultant. His tenure as a member of the staff of the Mayo Clinic was longer than that of any other active member now living.

Dr. O'Leary lived through an era which witnessed all but the absolute eradication of a devastating disease that had afflicted man for ages past. When he was a young man the treatment of syphilis was far from satisfactory, and it remained so for many years. Treatment was cumbersome to administer and tiresome to the patient because of the need for repetition. Dr. O'Leary applied himself to the study of this stubborn disease at the very outset of his career, and his first published paper was devoted to that subject. He did important work in the investigation of syphilis as it affects various internal organs, particularly in the abdominal cavity and the nervous system; and he was an early worker in the application of high fevers and inoculation with malarial organisms in the treatment of syphilis of the central nervous system. Finally, as he approached the end of his active career in medicine, the antibiotic drugs were introduced, and the importance of new infections with syphilis was thereby rendered almost negligible.

Many honors came to Dr. O'Leary in recognition of his long devotion to the study of syphilis and such diseases of the skin as scleroderma, a condition in which the skin becomes thickened, hard, rigid and hidebound; and dermatomyositis, in which the skin, underlying tissues and muscles become involved by inflammation. In 1936 and 1937 he was chairman of the section on dermatology of the American Medical Association, and in 1946 he was elected president of the American Dermatological Association. He was also elected to the presidency of the American Academy of Dermatology and Syphilology, of which he was one of the founders, and the Minnesota Dermatological Society and the Chicago Dermatological Society. In 1926 he had published, with Dr. John H. Stokes, and others, the volume, *Modern Clinical Syphilology;* in 1947 he was appointed editor-in-chief of the *Archives of Dermatology and Syphilology.* In 1939 he was appointed a member of the National Advisory Health Council by Secretary of the Treasury, Henry Morgenthau, Jr. He served also as chairman of the dermatoses investigating committee of the United States Public Health Service. With the aid of a private grant, he organized a nation-wide co-operative system for the collection and exchange of information about diseases of the skin and syphilis which has since received international recognition and tribute. In 1940 he was secretary-general of the Tenth International Congress on Dermatology and Syphilology, and in 1951 he was president of the staff of the Mayo Clinic.

An entire issue of *The Journal of Investigative Dermatology* was dedicated to Dr. O'Leary in 1954. At that time the journal called attention to his international reputation in research and treatment, and the graduate training of young physicians in the specialty of diseases of the skin. More than 100 received such graduate training under Dr. O'Leary's direction.

Dr. O'Leary was certified as a specialist in dermatology and syphilology in 1933 by the American Board of Dermatology, Inc. He served as a member of that board from 1938 to 1948. He was a fellow of the American College of Physicians and a member of the New York Academy of Science, the Pan-American Medical Association, the American Association for the Advancement of Science, the Society for Investigative Dermatology, the American Association of University Professors, the Minnesota Academy of Science, Minnesota State Medical Association, Minnesota Academy of Medicine, the American Medical Association, Society of the Sigma Xi, Alpha Kappa Kappa, professional medical fraternity and Beta Theta Pi, academic fraternity. He was also an honorary member of the Royal Society of Medicine of England, the British Association of Dermatologists, the German Dermatological Society, the Berlin Dermatological Society, the Dutch Society of Dermatology and Syphilology, the Hellenic Antivenereal Society of Greece, the Manhattan Dermatological Society and the Dallas Dermatological Society. In 1950 he was made an honorary member of Alpha Omega Alpha, national honor medical society. He was also a corresponding member of dermatological societies in France, Sweden, Greece, Denmark, Argentina and Hungary.

At one time he was keenly interested in fine horses, and often was called upon to act as judge at horse shows. He was also an early aviation enthusiast, an interest that was deevloped particularly as a result of his friendship with the automobile and aviation industrialist, E. L. Cord. He was especially skilled in woodworking, an avocation which occupied much of his leisure moments.

Dr. O'Leary was married to Miss Ruth Youmans, of Winona, Minnesota, on June 18, 1921. Dr. and Mrs. O'Leary had two children: Paul Arthur O'Leary, Jr., of Los Angeles, California, and Patricia O'Leary, of Phoenix, Arizona.

BLOOD COAGULATION

(Continued from Page 572)

to stress on use of fibrinogen. Fortunately, commercial "fibrinogen" is a mixture of proteins and contains other clotting factors of some use to the patient; commercial "antihemophilic globulin," which contains even more of such factors, including fibrinogen, might be more useful. If intravascular clotting continues unabated in the pregnant patient, it is hard to conceive of plasma or any of its derivatives being satisfactory for therapy.

Much remains to be learned about the clotting of blood, particularly concerning biochemical characterization of the individual components and their interactions. The burden of transferring each new advance in research to the practice of medicine should be assumed by the clinical pathologist.

RADICAL TREATMENT OF BRAIN ABSCESS

(Continued from Page 550)

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7. Groff, R. A., and Grant, F. C.: The surgical treatment of brain abscess by exposure and enucleation. Ann. Surg., 111:925-941, 1944.
8. Kahn, E. A.: The treatment of encapsulated brain abscess. J.A.M.A., 108:87-91, 1937.
9. King, J. E. J.: The treatment of brain abscess by unroofing and temporary herniation of abscess cavity with avoidance of usual drainage methods. Surg., Gynec. & Obst., 39:554-658, 1924.
10. LaBeau, J.: Radical surgery and penicillin in brain abscess. J. Neurosurg., 3:359-374, 1946.
11. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord. Glasgow: J. Maclehose and Sons, 1893.
12. Vincent, C.: Sur une méthode de traitment des abscès subaigus des hémisphères cérébraux; large décompression, puis ablation en masse sans drainage. Gaz. méd de France, 43:93-96, 1936.

Reports and Announcements

MEDICAL MEETINGS

National

Academy of Psychosomatic Medicine, 2nd annual meeting, Plaza Hotel, New York City, October 6-8, 1955.

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, the Homestead, Hot Springs, Virginia, September 8-10, 1955.

American Congress of Physical Medicine and Rehabilitation, Hotel Statler, Detroit, Michigan, August 28-September 2, 1955.

American Roentgen Ray Society, Palmer House, Chicago, Illinois, September 20-23, 1955.

American Medical Writers' Association, 12th annual meeting, Hotel Jefferson, St. Louis, Missouri, September 30, 1955, followed by Workshop on Medical Writing, October 1.

Association of Military Surgeons of the United States, 62nd annual convention, Statler Hotel, Washington, D. C., November 7-9, 1955.

Endocrinology and Metabolism, 7th annual Postgraduate Assembly of the Endocrine Society, Indiana University School of Medicine, Indianapolis, Indiana, September 26-October 1, 1955.

Mississippi Valley Medical Society, 20th annual meeting, Hotel Jefferson, St. Louis, Missouri, September 28-30, 1955.

National Society for Crippled Children and Adults, Palmer House, Chicago, Illinois, November 28-30, 1955.

Occupational Skin Problems, course sponsored by Institute of Industrial Health, University of Cincinnati, Kettering Laboratory, Cincinnati, Ohio, October 10-14, 1955.

Veneral Disease Postgraduate Course, 24th session sponsored by University of Chicago and Public Health Service, University of Chicago, September 26-30, 1955.

Winston-Salem Heart Symposium, Robert E. Lee Hotel, Winston-Salem, North Carolina, September 30, 1955.

State

Michigan State Medical Society, Pantlind Hotel, Grand Rapids, Michigan, September 26-30, 1955.

Montana Medical Association, Baxter Hotel, Bozeman, Montana, September 15-18, 1955.

International

International College of Surgeons, United States and Canadian sections, 20th annual congress, Convention Hall, Philadelphia, Pennsylvania, September 12-15, 1955.

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

AMERICAN COLLEGE OF CHEST PHYSICIANS POSTGRADUATE COURSES

The Council on Postgraduate Medical Education of the American College of Chest Physicians announces two postgraduate courses on diseases of the chest.

The 10th annual postgraduate course, Hotel Knickerbocker, Chicago, Illinois, October 3-7, 1955.

The 8th annual postgraduate course, Park-Sheraton Hotel, New York City, November 14-18, 1955.

Tuition is $75 for each course, which includes roundtable luncheons. Further information may be obtained from the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

AMERICAN COLLEGE OF GASTROENTEROLOGY

The American College of Gastroenterology announces that its annual course in postgraduate gastroenterology will be given at the Shoreland in Chicago on October 27, 28 and 29, 1955. The course will again be under the direction of Dr. Owen H. Wangensteen, professor of surgery, University of Minnesota Medical School, who will serve as surgical co-ordinator, and Dr. I. Snapper, director of medical education, Beth-el Hospital, Brooklyn, N. Y., who will serve as medical co-ordinator.

The subject matter to be covered, from a medical as well as surgical viewpoint, will include the advances in diagnosis and treatment of gastrointestinal diseases and a comprehensive discussion of diseases of the mouth, esophagus, stomach, pancreas, spleen, liver and gallbladder, colon and rectum, with special studies of radiology and gastroscopy.

For further information, write to the American College of Gastroenterology, Department P.G., 33 West 60th Street, New York 23, N. Y.

Minnesota physicians are invited to submit items **"of general interest"** concerning themselves or their colleagues for inclusion in this section. Items need not be written in finished form. All that is needed are the facts—answers to the old questions: who, what, where, when and why? Send information as desired to MINNESOTA MEDICINE, 2642 University Avenue, St. Paul 14, Minnesota.

AMERICAN GOITER ASSOCIATION AWARD

The American Goiter Association is offering the Van Meter Prize Award of $300 and two honorable mentions for the best essays on original work on thyroid gland problems. The award will be made at the annual meeting of the association at Chicago, Illinois, May 3-5, 1956.

Essays may cover either clinical or research investigations, should not exceed 3,000 words in length and must be in English. Duplicate typewritten copies, double spaced, should be sent to the secretary, Dr. John C. McClintock, 149½ Washington Avenue, Albany, New York, not later than January 1, 1956.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

The Northern Minnesota Medical Association will hold its annual meeting at Bemidji on September 2 and 3, 1955. The scientific program is as follows:

Friday, September 2

A.M.

10:00 "The Treatment of Varicose Veins." Dr. W. J. Deweese, Bemidji.

10:30 "Surgical Repair of Diaphragmatic Hernia." Dr. R. H. LaBree, Duluth.

11:00 "Post-partum Bleeding." Dr. W. F. Mercil, Crookston.

11:30 "Urinary Tract Infections." Dr. C. D. Creevy, Minneapolis.

12:30 Business Meeting.

P.M.

2:00 "Differential Diagnosis and Treatment of Pleural Effusions." Dr. W. R. Schmidt, Minneapolis.

2:50 "The Treatment of Diabetes." Dr. L. O. Underdahl, Rochester.

3:40 Panel Discussion: "Rheumatic Fever." Epidemiology—Dr. J. J. Coll, Duluth. Diagnosis—Dr. Emmerson Ward, Rochester. Treatment—Dr. Harry Orme, Fargo, N. D.

Friday Evening

7:00 Annual Banquet. Principal speaker: Dr. Theodore H. Fenske, Assistant Dean, Institute of Agriculture, St. Paul.

Saturday, September 3

9.00-12:00 noon. Clinico-Roentgen-Pathological Conference. Led by Dr. Henry G. Moehring, Duluth.

ARTHRITIS AND RHEUMATISM FOUNDATION RESEARCH FELLOWSHIPS

The Arthritis and Rheumatism Foundation is offering three types of research fellowships in the basic sciences related to arthritis. Predoctoral fellowships range from $1,500 to $3,000 per year, depending on family responsibilities, and are tenable for one year with prospect of renewal. Postdoctoral fellowships range from $4,000 to $6,000 per year, depending on family responsibilities, and are tenable for one year with prospect of renewal.

Senior fellowships for more experienced investigators carry an award of $6,000 to $7,500 per year and are tenable for five years.

Deadline for applications is October 15, and awards will be made in January, 1956. For information and application forms, write the Medical Director, Arthritis and Rheumatism Foundation, 23 West 45th Street, New York 36, New York.

MINNESOTA CHAPTER OF ARTHRITIS AND RHEUMATISM FOUNDATION

The attention of physicians of the state is called to the newly organized Minnesota Chapter of the Arthritis and Rheumatism Foundation. The state chapter will devote its efforts to aiding research, education and treatment in the field of rheumatic diseases.

Dr. Charles Slocumb, Rochester, is president, and Judge Paul S. Carroll, Minneapolis, is chairman of the Minnesota chapter.

A national fund-raising campaign will be held this coming November. Any inquiries can be directed to the Executive Secretary, R. V. Stevenson, 89 South Tenth Street, Minneapolis, Minnesota.

MINNESOTA ACADEMY OF GENERAL PRACTICE

The Minnesota Academy of General Practice will hold its fifth annual Fall Refresher at the Radisson Hotel, Minneapolis, on October 19, starting at 8:30 a.m. Eight hours of concentrated instruction will be given by fourteen authorities, highlighted by a talk by Dr. H. E. Rynearson, Rochester, at the noon luncheon. Also appearing will be Dr. Bert Seligman, Toledo; Dr. Gene Stollerman, University of Chicago, and Dr. George Logan, Rochester.

There is no registration fee, but a five-dollar luncheon ticket is required. Registrations are advisable and should be made through Dr. James A. Blake, 15 Ninth Avenue South, Hopkins, Minnesota.

MINNESOTA HEART ASSOCIATION

Dr. J. A. Cosgriff of Olivia was elected president of the Minnesota Heart association for 1955-1956 at the annual meeting July 12 in St. Paul. Dr. Cosgriff, a past president of the Minnesota chapter of the American Academy of General Practice, succeeds Dr. Charles N. Hensel of St. Paul.

Other officers elected were Dr. Robert Parker, Mayo Clinic, first vice-president; E. B. Eliason, Minneapolis, second vice-president; Eugene L. Reichel, Minneapolis, third vice-president; Dr. Milton Hurwitz, St. Paul, secretary, and Bernard B. Knopp, St. Paul, treasurer.

A total of $154,464 was allocated for heart research in the state during the next year.

WANGENSTEEN ANNIVERSARY PROGRAM

The Owen H. Wangensteen Surgical Education Foundation, a group made up of former residents of Dr. Wangensteen, is presenting a special program on September 22, 23 and 24, honoring the twenty-fifth anniversary of the appointment of Dr. Wangensteen to the chair of professor of surgery at the University of Minnesota.

The program will consist of a series of papers on surgical subjects given by present and former associates of Dr. Wangensteen. Included among the speakers will be Dr. John R. Payne, University of Buffalo; Dr. Clarence Dennis, New York University Medical School; Dr. Arnold Kremen, Columbia University Medical School; Dr. K. Alvin Merendino, University of Washington Medical School; Dr. David State, Cedars of Lebanon Hospital, Los Angeles; Dr. Irving Ariel, Pack

Medical Group, New York; Dr. George E. Moore, Roswell Park Cancer Institute, Buffalo, N. Y.; Dr. Edward E. Mason, University of Iowa Medical School; Dr. H. William Clatworthy, Children's Hospital, Columbus, Ohio, and members of the surgical department of the University of Minnesota.

On September 22 there will be an operative clinic in cardiac surgery, presented by Dr. Richard Varco, Dr. C. Walton Lillehei and Dr. F. John Lewis. There will be a public banquet on the evening of September 23, at which time the major address will be made by John Russell, executive director of the John and Mary Markle Foundation. A meeting of the Owen H. Wangensteen Surgical Education Foundation will be held on the evening of September 24. The surgical program will be presented in the Mayo Memorial Auditorium of the University of Minnesota Hospitals on September 23 and the morning of September 24.

A cordial invitation is extended to all physicians to attend this program.

CONTINUATION COURSES

The University of Minnesota will present its annual continuation course in *Radiology for Radiologists* at the Center for Continuation Study November 7 to 12. Subject of this year's program will be neuroradiology.

Guest faculty will include Dr. Donald L. McRae, chief of radiology, Montreal Neurological Institute, McGill University Faculty of Medicine, Montreal; Dr. Ernest H. Wood, Jr., professor of radiology, University of North Carolina School of Medicine, Chapel Hill; and Professor Erik Lindgren, professor of radiology, Serafimerlasarettet, Stockholm, Sweden.

The program will be presented under the direction of Dr. Leo G. Rigler, professor and head, Department of Radiology, and Dr. H. O. Peterson, clinical professor, Department of Radiology, University of Minnesota Medical School.

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The University of Minnesota announces a continuation course in *Bacteriology and Blood Bank Techniques for Medical Technologists,* which will be held at the Center for Continuation Study from November 14 to 16. The course, which is open to all qualified medical technologists, will be presented under the direction of Dr. Ellis S. Benson, acting director, Hospital Laboratories.

Minnesota Academy of Medicine

Meeting of November 10, 1954

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, November 10, 1954. Dinner was served at 7 o'clock, and the meeting was called to order at 8:10 p.m. by the President, Dr. C. N. Hensel.

There were forty-eight members and two guests present.

The Secretary read a letter from Dr. S. E. Sweitzer asking that his name be transferred to the Senior List and stated that this had the approval of the Executive Committee. Upon motion, which was seconded, this was carried.

The Secretary read the memorial to Dr. Lee Barry, who died December 3, 1953.

A vote on the amendment presented at the October meeting was taken and this was carried.

Dr. Callahan introduced his guest, Dr. Dale Cameron; and Dr. Grant introduced his guest, Dr. McCloud, son of a former president of the Academy, Dr. C. N. McCloud.

Dr. Hensel appointed Drs. Hengstler (chairman), Spink and Regnier a committee to investigate the matter of a scholarship by the Academy, and make recommendations at a later meeting.

The scientific meeting followed.

Dr. Wallace D. Armstrong, of the University of Minnesota Medical School, read his Inaugural Thesis on "Radioisotope Studies of the Physiology of Calcified Tissues." Lantern slides were shown. (to be published later)

Dr. Frederick H. Van Bergen, of the University of Minnesota Medical School, read his Inaugural Thesis on "Hexamethonium-Induced Hypotension." (See page 573).

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

Woman's Auxiliary

RAMSEY WIVES PLAN FALL FASHION FESTIVAL
Mrs. L. G. Culver

On June 27, Mrs. H. O. Peterson, president of the Woman's Auxiliary to the Ramsey County Medical Society, called to order a special meeting held at the home of Dr. and Mrs. Donald R. Lannin, 1527 Edgcumbe Road, Saint Paul. The purpose of the meeting was to discuss changing the regular meeting day. Henceforth, the meetings of the Ramsey county auxiliary will be held on the third Monday of each month. It was a gorgeous day, the attendance was very good, and Lannin's beautiful lawn was enjoyed to the utmost.

There was considerable discussion about the Benefit Fall Fashion Festival, which will be held on Thursday, September 15, at the Hotel Saint Paul. Mrs. Rodney F. Sturley, general chairman of the event, reported plans are well under way. Proceeds will go to the American Medical Education Foundation and other philanthropic activities. Last year's show was very successful, and there is every reason to believe this one will be also. It isn't only in the spring that a lady's fancy turns to—a new hat, suit, shoes and gloves! Tickets are available to everyone. Drop a card to Mrs. Charles Eginton, 1808 Beechwood, Saint Paul 5. Luncheon is $3.00 and you may win a door prize!

Of General Interest

Dr. Sol Austrian, after four years of practice at Cosmos, began postgraduate study in pediatrics at the University of Minnesota in August. His practice in Cosmos has been taken over by **Dr. Russell A. Thompson,** a graduate of the University of Minnesota Medical School who recently completed his internship at St. Mary's Hospital, Duluth.

* * *

Principal speaker at a meeting of the Luverne Rotary Club on July 18 was **Dr. S. A. Slater,** head of the Southwestern Minnesota Sanatorium, Worthington. He reviewed the history and accomplishments of the sanatorium from its opening in 1919 but warned that "the battle against tuberculosis is not yet won."

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A service recognition certificate was presented to **Dr. Frank J. Hirschboeck,** Duluth, at the annual summer meeting of the Minnesota Safety Council in Duluth on August 19.

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Dr. and Mrs. Philip S. Hench, Rochester, left on July 30 for a three-week trip to Panama and South America. While in Rio de Janeiro, Dr. Hench presented two papers at the First Pan-American Congress of Rheumatology.

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Dr. G. H. Mesker and **Dr. F. W. Penhall** were honored at the fifty-fourth annual Old Settlers Reunion at Hector late in July. Dr. Mesker practiced at Olivia for more than fifty years. Dr. Penhall, who was formerly on the staff of the Willmar State Hospital, now lives at Morton. Both physicians are retired.

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Word has been received of the death of **Dr. Auguste Rollier,** who died on October 30, 1954, a month after his eightieth birthday in Lerpin, Switzerland, where he had lived most of his life. He was assistant to Professor Kocher of Berne after securing his license to practice there. He made many researches in heliotherapy and opened the first clinic for the treatment of surgical tuberculosis by sunlight with considerable success.

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The practice formerly conducted under the name of Drs. Rutledge and Moberg in Detroit Lakes is now known as the Detroit Lakes Clinic. Associated with the clinic are **Dr. L. H. Rutledge, Dr. C. W. Moberg, Dr. M. E. Odland** and **Dr. Ernest S. Lorentzen.**

* * *

Seven physicians have been named as new members of the staff of the Mayo Clinic, Rochester. The new consultants are **Dr. Philip E. Bernatz, Dr. Norman P. Goldstein, Dr. Paul A. Green, Dr. Norman G. Hepper, Dr. John G. Mayne, Dr. Harold J. Swan** and **Dr. Richard A. Theye.**

Dr. Leo G. Rigler, chief of the department of radiology at the University of Minnesota, who is on a leave of absence from the university, has joined the staff of the City of Hope Medical Center in Los Angeles, California. While there, he will be director of postgraduate medical education and chief of x-ray diagnosis.

* * *

Dr. Arthur J. Henderson has been appointed coroner of Ramsey County to fill the unexpired term of **Dr. Carl Ingerson,** who resigned August 1. Dr. Henderson has been deputy coroner for ten years. Dr. Ingerson, after holding the office of coroner for thirty-six years, resigned because of ill health.

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A new clinic building is being constructed in Glencoe by **Dr. Arthur Neumaier.** The building, which is expected to be completed by November 1, will also have offices for a dentist.

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Dr. Carl R. Wall, Minneapolis, was the subject of a biographical sketch in the Town Toppers column of the *Minneapolis Star* on August 2.

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Dr. William R. Bagley, Duluth, has been reappointed a member of the St. Louis County Sanatorium Commission for three years.

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Dr. and Mrs. C. A. Boline, Battle Lake, left in mid-July for a two-month tour of Europe. While they are away, Dr. Boline's practice is being conducted by **Dr. George DeBelly,** formerly of Nebraska.

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Dr. Joseph P. Connolly of the Malerich Clinic, W. St. Paul, is taking a one-year residency in internal medicine at Miller Hospital, St. Paul.

* * *

Three retired members of the Mayo Clinic staff, Rochester, have been awarded certificates of merit by the University of Minnesota in honor of their long service as members of the faculty of the graduate school. Receiving the certificates were **Dr. Henry P. Wagener,** who retired July 1, and **Dr. Fred Z. Havens** and **Dr. Luther Thompson,** both of whom retired April 1.

* * *

A new federal office has been set up in Bemidji to administer Indian medical and public health activities in Minnesota, Wisconsin, Michigan and Iowa. Acting medical officer in charge of the office is **Dr. Sidney Finkelstein,** Cass Lake.

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Dr. M. D. Starekow, Thief River Falls, was elected president of the Thief River Falls board of education at its organization meeting in mid-July.

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Selected as the first member of the Sandstone Hall of Fame, **Dr. H. P. Dredge** was honored at a banquet in the Sandstone State Hospital on July 16. Dr. Dredge, now eighty-four, has practiced medicine at Sandstone for fifty-two years. Always active in civic and community affairs, Dr. Dredge was also one of the promoters of the new Pine County Memorial Hospital being erected at Sandstone. The waiting room of the hospital will be furnished in his honor.

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Dr. Louis A. Buie, Rochester, has been appointed a national consultant to the Surgeon General of the U. S. Air Force. The medical department of the Air Force does not have a formally organized group of civilian consultants but calls on qualified men when necessary.

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Dr. John Eustermann of the Mankato Clinic, Mankato, has been appointed to the board of directors of the Minnesota Heart Association.

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Dr. Paul M. Smith, Lake Crystal, was elected president of the Southern Minnesota Chapter of the Minnesota Academy of General Practitioners at Mankato in June. Other officers elected were **Dr. Daniel Halvorsen,** Owatonna, vice president, and **Dr. Orville Jones,** Mankato, secretary-treasurer. Principal speaker at the meeting was **Dr. Asher White,** Minneapolis, who spoke on "Diseases of the Chest Resembling Heart Diseases."

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Construction of a new Alexandria Clinic has begun at Alexandria. The building, which will measure 60 by 106 feet, is expected to be completed by January 1. The clinic staff is composed of **Dr. G. W. Clifford, Dr. H. L. Stemsrud, Dr. C. E. Carlson, Dr. L. F. Wasson, Dr. P. M. Geiser, Dr. D. E. Perkins,** and **Dr. W. G. Heegaard.** The clinic has been in its present location for ten years.

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Dr. William V. Knoll, formerly of Duluth, has become pathologist at the new St. Joseph's Hospital, Brainerd. Dr. Knoll, who formerly was pathologist at St. Mary's Hospital, Duluth, was honored before he left for his new post at a testimonial dinner in Duluth in June. He has been succeeded at St. Mary's by **Dr. Arthur C. Aufderheide,** formerly associate pathologist at the hospital.

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Miss Jeanne Mary Reince and **Dr. Robert Louis Koller,** Minneapolis, were married at St. Matthew's Catholic Church, Green Bay, Wisconsin, on June 25.

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Dr. Allan J. Blake and **Dr. James A. Blake,** Hopkins, were honored at a dinner in late June by the police and fire departments of Hopkins. They were presented with engraved plaques in appreciation of their years of "good-natured service in time of emergencies."

* * *

Dr. C. L. Sherman, Luverne, has been elected president of the Southwestern Minnesota Sanatorium district for the forty-first consecutive time.

Dr. W. W. Higgs, Park Rapids, was recently honored at ceremonies at the University of Wisconsin marking his fifty years as a physician. Dr. Higgs has practiced in the Park Rapids area for forty-three years.

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Dr. John S. Lundy, Rochester, has been appointed a member of the medical advisory committee of the American National Red Cross. The committee is limited to nine members.

NEW LOCATIONS

Dr. John B. Rutledge, recently discharged from the Air Force, has opened offices for the practice of medicine in Frazee.

Dr. John K. Meinert has become associated with the Lakeland Medical Center, Willmar.

Dr. Edward Kiolbasa has become associated in practice with **Dr. F. M. McCarten** and **Dr. R. E. Carlson** in Stillwater.

Dr. F. D. Bucher, recently in service in the Navy, has reopened his offices in the Starbuck Clinic, Starbuck.

Dr. William P. Miller, a former Ortonville resident, has become associated in practice with **Dr. John Allen** in Montevideo.

Dr. Robert Fedor, a native of Worthington, has become affiliated with the Litchfield Clinic, Litchfield.

Dr. W. B. Halme, after two years in military service, has resumed his practice in Wadena.

Dr. Howard R. Snider, formerly of Minneapolis, has become associated with the Mankato Clinic, Mankato.

Dr. Marvin W. Johnson, formerly of Milaca, has become associated in practice with **Dr. Joseph C. Houts** in Dassel.

Dr. Raymond Chittum has become affiliated with the East Range Clinic in Aurora.

Dr. Virgil A. Watson, formerly of Kalamazoo, Michigan, has become associated in practice with **Dr. William C. Dodds** in Detroit Lakes.

Dr. Karl Helwig has opened offices for the practice of medicine in Grove City.

Dr. Arnold L. Hamel has become associated in practice with **Dr. M. H. Seifert** and **Dr. J. A. Dupont** in Excelsior.

Dr. Jack H. Gardner has opened offices for the practice of medicine in Ceylon.

Dr. Abraham Alpert has become affiliated with the East Range Clinic at Virginia.

Dr. Fredric L. Mast, formerly of Minneapolis, has become associated with the Adams Clinic in Chisholm.

Dr. A. J. Lenarz, after several months of postgraduate surgical training in St. Paul, has resumed his practice at Browerville. Now associated with him in practice is **Dr. William J. Byrne,** formerly of St. Paul.

Dr. John B. O'Leary, formerly of Minneapolis, has become associated in practice with **Dr. B. J. Hughes** in Brainerd.

Dr. Vernon Vix, formerly of Minot, N. D., has become affiliated with the Worthington Clinic, Worthington.

Dr. Carol L. Plott of Lancaster, Penn., will become associated in practice with **Dr. R. W. Nicholson** at Heron Lake on September 1.

Dr. Alvin C. Peterson, son of the late Dr. A. A. Peterson, has returned to Mora to open offices for the practice of medicine.

Dr. Joseph E. Henry, after a year of military service in Germany, has resumed the practice of medicine at Milaca.

Dr. Donald G. Alton has become associated in practice with **Dr. H. P. Hinderaker** in Bird Island.

Dr. W. G. Larson has become associated in practice with **Dr. R. F. Mears** in Northfield.

Dr. Arthur Sethre has become affiliated with the Fergus Falls Clinic at Fergus Falls.

Dr. Richard Runquist has become associated in practice with **Dr. J. V. Carlson** in Westbrook.

Dr. Paul H. Potter, after a term of service in the Army, has begun a four-year residency in pathology at St. Luke's Hospital, Duluth.

Dr. Troy Rollins, formerly of Elmore, has finished a two-year period in the Navy and has joined the staff of the Rochester State Hospital. On January 1, 1956, he will start a three-year residency in dermatology at the Mayo Clinic.

Dr. Myles Eftelund, formerly of Erskine, has joined the staff of the Karlstad Hospital, Karlstad.

Dr. Dennis E. Lofstrom and **Dr. Robert E. Wilson** have taken over the operation of the Pine River Clinic, Pine River.

MINNESOTA BLUE SHIELD-BLUE CROSS

Statistics just released from Minnesota Hospital Service Association reveal that during the first five months of 1955 a total of 69,198 hospital cases resulted in payment of benefits in excess of $8,000,000.

During the same period in 1954, a total of 70,422 hospital cases resulted in benefit payments of approximately $7,400,000. Though cases in the first five months of 1955 showed a slight decrease, the number of days of hospital care increased from 422,219.1 in 1954 to 429,982.8 in 1955.

The four most predominant diagnoses are still respiratory, digestive, pregnancy and accidents. Pregnancies during the first five months of 1955 ranked first, with a total of 11,477 cases paid by Blue Cross.

Since 1933, 1,799,192 Blue Cross subscribers have used approximately 11,000,000 days of hospital care and $119,000,000 in benefits.

As of May 31, 1955 approximately 1,006,423 Minnesotans were covered by Blue Cross.

At the Annual Meeting of the Minnesota State Medical Association in Minneapolis in May of 1955, increased interest in Blue Shield was clearly shown by the more than 200 doctors who visited the Blue Shield booth.

Among the subjects that were most important to the doctors was the physician-patient relationship. Many were of the opinion that prompt and correct filing of Blue Shield claims and rapid processing of the claims by Blue Shield lead to an improved relationship between the doctor, his patient, and Blue Shield.

Doctors' comments and questions were varied, but pertained primarily to the methods involved in the reporting of services rendered and the payment of claims. From the information exchanged, it is believed that many of these problems may be solved by a more complete report of the services rendered. Every effort has been made by Blue Shield to provide a Medical Service Report or claim blank which is worded in such a manner as to reduce to a minimum the time and effort required for its proper completion. Items contained in the Blue Shield Medical Service Report which are frequently incomplete and result in delayed payments are: the date the doctor's service was rendered, the description of the service rendered, the hospital admission and discharge date, the signature and serial number of the attending physician, Workmen's Compensation status, and the relationship of the patient to the subscriber. Omission of these items requires Blue Shield to obtain additional information from the doctor's office and results in the delayed payment of the claims involved. The delay incurred by incomplete data may result in a disturbed patient relationship and may lead to dissatisfaction between the patient, the physician, and Blue Shield.

FRACTURES OF THE PELVIS

(Continued from Page 564)

results of the treatment of the fractured pelvis, but rather upon the damage to the upper end of the femur with the possibility of aseptic necrosis.

Separation of the symphysis is accompanied by separation of the sacroiliac joints posteriorly. The degree of pain and disability varies directly with the extent of the separation. These patients were managed initially by suspending the pelvis in a sling and compressing the wings of the ilium together. Stabilization of the symphysis does not depend upon bony healing. Soft tissue healing should be sufficiently strong in about three weeks, after which activity was encouraged. These patients had considerable persistent pain over the sacroiliac joints posteriorly, which was treated by the use of a trochanteric belt.

Conclusion

In this group of fractures of the pelvis, associated additional injuries occurred in two-thirds of the injured patients. These coincidental injuries were frequently of much greater importance than the pelvic fracture. Three-fourths of the fractures of the pelvis could be treated simply, without recourse to special methods of management.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicans.

INTESTINAL OBSTRUCTIONS. Third Edition. Owen H. Wangensteen, B.A., M.D., Ph.D. Professor of Surgery, University of Minnesota, Minneapolis. 838 pages. Illus. Price $15.50, cloth. Springfield, Illinois: Charles C Thomas, 1955.

HANDBOOK OF PEDIATRICS. Henry K. Silver, M.D., Associate Professor of Pediatrics, Yale University School of Medicine; C. Henry Kempe, M.D., Assistant Professor of Pediatrics, University of California School of Medicine, San Francisco; and Henry B. Bruyn, M.D., Assistant Professor of Pediatrics and Medicine, University of California School of Medicine and Assistant Clinical Professor of Pediatrics, Stanford University Medical School, San Francisco. 548 pages. Illus. Price $3.00, paper cover. Los Altos, California: Lange Medical Publications, 1955.

THE MAYO CLINIC. Lucy Wilder. 2nd edition. Illus. $3.75. Springfield: Charles C Thomas, 1955.

This thin volume is a judicious condensation of a great deal of material that could have been written about the Mayo Clinic. One can only imagine the fortitude required to keep it brief and the anguish over material necessarily omitted. The book is a genuine triumph of selection. For the most part, it has been prepared in response to a demand on the part of the visitors to Rochester for authentic information about the origins of the Mayo Clinic, its present organization and an explanation of the many buildings that are part of the medical work there. This purpose has been beautifully fulfilled by Mrs. Wilder's book.

The charitable work of the founders of the clinic is suitably portrayed as traditionally a part of the physician's life then and now and in all latitudes and climes.

It is not likely that much of the content of this book will be new information to the readers of this journal. As a simple and really charming explanation of the Mayo Clinic, the book can be sincerely recommended.

William B. Martin, M.D.

CAN WE FURTHER REDUCE INFANT MORTALITY?

(Continued from Page 562)

interest of the fetus. Early detection and treatment of toxemia factors should be emphasized. The avoidance of podalic version and difficult mid-forceps with studied management of mid-pelvic contractions is advised. Early diagnosis of multiple gestation with restriction of activities from the seventh month on is to be recommended. Perhaps the common practice of performing a repeat cesarean section two weeks before the expected date of confinement should be altered to one week or less. There are frequently miscalculations as to the length of gestation.

The most critical period in the lives of the mother and her baby is during labor and delivery. Special care and judgment must be used in prolonged labor. More attention should be given to the immediate postpartum care of the newborn infant, especially during the first eight minutes after birth, when conditions occur which often lead to death in the early hours or days of fetal life. This period is definitely the obstetricians' responsibility. He should know how to resuscitate the baby and administer the necessary care. Most of the deaths occur in the first twenty-four hours of life or so-called "critical period."

Further reduction in neonatal mortality requires improvement of pre-natal care, starting early in pregnancy; the prevention of preventable deaths in hospitals; and the reduction of mortality, particularly in early age and in low weight groups. A continuous and more extensive neonatal mortality study as well as an intensive study of fetal deaths will help reach this desirable objective.

BILATERAL GLENOID HYPOPLASIA

(Continued from Page 569)

cases also showed webbing of the axillae and marked limitation of abduction.

Although several cases of bilateral glenoid hypoplasia have been described in the European literature, we are unable to find a description of this entity in a patient from this country. Because of the non-incapacitating nature of the deformity it seems likely that the majority of the patients do not seek medical advice.

Conclusions

1. A single case of bilateral glenoid hypoplasia is described.

2. Bilateral failure of development of the secondary ossification centers for the inferior rims of the glendoids is associated with flattening of the humeral heads, limitation of abduction, and external rotation and webbing of the axillae.

3. The malformation is apparently not associated with familial or hereditary factors. There seems to be no sex predominance.

4. Reviews of several cases in European literature have been presented. No case reported from this country has been encountered.

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3. Scaglietti, O.: The obstetrical shoulder trauma. Surg., Gynec. & Obst., 66:868, 1938.
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Symposium on the Lung

Observations on the History of the Bronchopulmonary Segments

EDWARD A. BOYDEN, Ph.D. (Med. Sc.)
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IN introducing this series of papers concerning the lung, it is instructive to look back over the seventy-five years in which current knowledge of the segmental anatomy of the lungs has developed, and to see what manner of men were responsible for it.

Prior to the year 1880, little attention had been paid to the mode of branching of the bronchial tree except in its peripheral portions. Generally, the mode was considered to be dichotomous and symmetrical. This concept was changed by a Swiss anatomist, Christoph Theodor Aeby, who presided over the anatomical institute at Bern for twenty-one years.

His interest in the lungs began in 1878, when he injected the trachea of an intact cadaver with Wood's metal. This taught him that the air passage which was most directly continuous with the trachea was the right bronchus and not the left, as had been supposed hitherto. Also, he was struck with the asymmetry of the tracheobronchial tree, noting for the first time that the upper lobe bronchi were differently related to the corresponding pulmonary arteries: the right upper was "eparterial," the left was "hyparterial." Thereupon, he set out to analyze the mode of division of the bronchi in fourteen mammalian families and to homologize the human pattern with that of the lower orders. Especial prominence was given to the interpretation that each lung consisted of an axial stem with alternate dorsal and ventral branches. Thus arose the "doctrine of the stem bronchus" which was to persist on the continent of Europe until the end of the second world war.

Although Aeby moved to Prague toward the end of his career, to succeed Toldt, he belonged to Switzerland. At Basel, he had been a fellow student of the great embryologist-to-be, Wilhelm His, and had absorbed from the less well-known Professor Rütimeyer an undying interest in comparative anatomy. In his fourth year of medicine, he had studied under the great Henle—that genius who had personally redrawn all parts of the human body and created a new gross anatomy—the science we know today. Aeby, too, was endowed with great energy, and with wit and humor. Being a student of Nature, he participated in mountain climbing—an interest just newly developed by civilized man. In the Alps, he climbed at least six of the high peaks—the highest of these in August, 1871—and lived to write an article for Sonntag's *Post* entitled, "A Storm on the Jungfrau." Perhaps it was this stored-up energy which enabled him without the help of even a prosector to teach anatomy, histology and comparative anatomy, to lecture four to six hours a day, and to produce fifty scientific articles and one book before he died from lung disease at the age of fifty.

The second great figure in the history of this subject was William Ewart, pathologist and consulting physician at Brompton and at St. George's Hospital in London. Ewart was obviously the scholarly type of physician, although as a medical student he had voluntarily participated in the Franco-Prussian war—perhaps because of devotion to his mother's country or because he had taken his arts degree at the University of Paris. It is significant that after graduating from Cambridge (M.B. in the first class of the Natural Science Tripos) he became a lecturer in physiology and a demonstrator in physiological

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From the Department of Anatomy, University of Washington.
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chemistry, and that after postgraduate work in Berlin, he pressed on to obtain the degree of M.D. at Cambridge. Thus Ewart laid the foundations of knowledge in a field and its literature that was said to be "remarkably intimate and well documented."

However, his was essentially an original mind. His supervision of out-patient practice was said to be "an informative and whimsical demonstration of the various clinical material which came under his eye," and his clinical teaching wholly unlike the stereotyped teaching of the day. Often, patients were disconcerted by his methods of cross examination, and family practitioners felt that he failed to render the prompt and straightforward assistance that was expected of him, because of his preoccupation with the scientific aspects of the disease. "And thus it came about that William Ewart, with great scientific qualities and personal characteristics that endeared him to many, made no headway at all as a consultant physician."

The above sentence—penned after Ewart's death at the age of eighty-one, and concluding the obituary account in *Lancet*—bears the date of 1929. Even then, his "most important literary work" was erroneously considered to be the Gulstonian lectures on "pulmonary cavities." Had he lived only a few years longer, he could have known that his great monograph of 1889—"The bronchi and pulmonary blood vessels: Their anatomy and nomenclature; with a criticism of Professor Aeby's views of the bronchial tree of mammalia and of man"—was to become the cornerstone of an enduring edifice in the history of surgery and medicine.

At the time that Ewart wrote this monograph, the pressing mission of embryology and anatomy was to relate human structure and that of the lower orders into a phylogenetic whole. So Ewart was breasting the current when he took issue with Aeby and asserted that a mammalian pattern should not be read into the structure of human lungs. Many years later, the American anatomist Huntington was to support this view. Meanwhile, Ewart maintained that the shape of the human thorax was such that the bronchi were more radially disposed than in quadrupeds and were not, therefore, dorsal and ventral branches of an axial structure. On this basis, he identified nine major bronchi that supplied respective "districts" of the lungs. If the middle lobe district be divided, these are essentially the bronchopulmonary segments of our day. But the time had not yet come for another step forward. Surgery of the lung and better techniques in anesthesia were virtually unknown; and the comparative anatomists were in the saddle. Hence, Ewart's fruitful ideas had to await a new "millenium"—of four decades; and Aeby's mammalian patterns became the clinical map of the lungs.

In 1934, a brilliant young surgeon of Brompton Hospital, H. P. Nelson, revived Ewart's "districts" and spread the knowledge of them throughout the British Commonwealth. Two years earlier, essentially the same plan had been "discovered" by a young American surgeon. While it has been the intention of the writer not to discuss living contributors at this time, it is necessary at this point to speak of Amiel Glass. Dr. Glass was a surgical interne in Mt. Sinai Hospital in New York when pioneers like Lilienthal were trying to find new ways of treating pulmonary disease. These surgeons impressed upon him the need for locating lesions more precisely. With the aid of the experienced bronchoscopist, Rudolph Kramer, Glass injected the lungs, found eleven "bronchopulmonary segments" and mapped their distribution on the surface of the lungs. While his work in no way compares in breadth and depth with Ewart's, nevertheless it gave new impetus to the study of pulmonary disease in America. Therefore, it is fitting that Glass's work should be an inspiration to young doctors—an example of what may be accomplished when youth concentrates upon specific problems in a chosen field.

For later steps in the development of the concepts of bronchopulmonary segments, the reader is referred to the writer's chapter on the history of this subject.* The purpose of the present article is to call attention to human values in one small field of medicine—to the lives of men who have become dedicated to the advancement of knowledge for its own sake. For much as we may hope that our work will be of use in the world, the driving force to the discovery of new truth is the inquisitiveness of the human mind.

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*Segmental Anatomy of the Lungs, Blakiston Division, McGraw-Hill Book Co., 1955.

Pulmonary-Function Tests

WARD S. FOWLER, M.D.
Rochester, Minnesota

THE principal function of the lungs is to provide efficient and adequate exchange of oxygen and carbon dioxide between the atmosphere and the blood. However, this exchange is accomplished not only by the action of the lungs but by the whole complex process of breathing, which involves the medullary respiratory center, the peripheral nerves and muscles, the thoracic wall, the upper airway, the tracheobronchial airway, the pulmonary alveoli and the pulmonary capillary circulation. Emphasis is placed on this fact because disturbances of respiration may arise in any one or several of these sites. Also, the whole respiratory apparatus is involved not only in many so-called pulmonary-function tests but also in the effects of a particular primary abnormality.

In the evaluation of pulmonary function, careful analysis of the history and the results of physical examination is essential. The presence, type, degree and progression of dyspnea merit particular attention. Observation of the patient during and after exercise is often useful. The principal value of pulmonary-function tests is to provide objective measurements. These may be helpful in differential diagnosis and in evaluating pulmonary performance and reserve. Assessment of therapy is often aided by their employment.

Pulmonary-function tests share the limitations of certain other functional tests, such as some used in renal and hepatic disease. A disturbance of the function measured will be detected only if the disease process is sufficiently great. Also, the anatomic or pathologic basis of an abnormality usually must be defined by or in conjunction with other methods of diagnosis. The interpretation of results of pulmonary-function tests, as with most testing procedures, requires their integration with the other aspects of the particular clinical problem.

The most practical tests are those for measuring ventilatory capacity, which is not only affected in various ways by many diseases but is also assessable by tests that can be done easily in a period of minutes with a small recording spirometer of the Benedict-Roth type.* Three procedures are of particular value:

1. The first is the determination of vital capacity, namely, the maximal volume of gas that can be expired following a maximally deep inspiration. Reductions of vital capacity occur with a variety of diseases which affect the ventilatory apparatus, such as poliomyelitis, kyphoscoliosis, and pulmonary alterations associated with fibrosis, emphysema, bronchospasm and vascular congestion.

2. The second procedure is to have the subject perform maximal expiration as *rapidly* as possible. This test gives additional information about the expiratory flow rates that can be achieved with maximal effort. Respiratory obstruction, such as occurs in asthmatic bronchitis and in emphysema, is shown by the prolonged duration and reduced rate of flow of expiration. These abnormalities may be present even though impairment is not sufficiently advanced to produce a definite reduction of the total volume expired (vital

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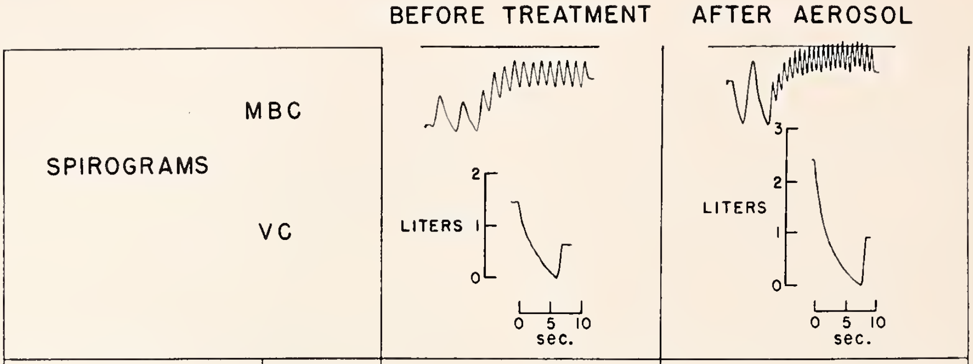
Read at the Continuation Course in Recent Advances in Internal Medicine at the Center for Continuation Study, University of Minnesota, February 15, 1955.
From the Section of Physiology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

*A conventional cylindrical spirometer of the type used for measurement of basal metabolic rate can be employed, provided the valves and carbon dioxide absorber are removed to reduce resistance to rapid flow of gases. However, spirometers are available commercially (Warren E. Collins, Inc., 555 Huntington Avenue, Boston 15, Massachusetts) with several modifications which make them much more satisfactory for pulmonary-function tests and which can also be used for measurement of metabolic rate. One of the speeds of the recording drum made by this firm is faster than that used for measurement of metabolic rate, and this feature is essential for proper measurement in the tests that involve rapid breathing.

capacity). Figure 1 illustrates the typical expiratory spirograms of a patient with far-advanced asthmatic bronchitis and emphysema. The slow, prolonged expiration is evident both on inspection of the spirograms and on physical examination of the patient. However, in cases of less-advanced emphysema, when physical and roentgenologic examination may give equivocal results, measurement of the forced expiratory spirogram usually reveals definite slowing. The most useful measurements are the percentage of the total volume expired in the first second, which should normally exceed 65 to 70 per cent, and the mean flow rate of the middle half of volume expired (maximal midexpiratory flow), which should normally exceed 2 liters per second. Figure 2 illustrates a different type of spirogram found in other types of pulmonary impairment. The vital capacity is reduced in this case because of pulmonary fibrosis, but air can be expelled with normal rapidity. This has been called "restrictive impairment," in contrast to the obstructive impairment illustrated by the spirograms of the patient with emphysema (Fig. 1).



TEST	NORMAL	BEFORE TREATMENT	AFTER AEROSOL
Vital Capacity (L.)	4.2	1.7	2.4
Max. Breathing Cap. (L./min.)	100	22	38
Max. Midexp. Flow (L./sec.)	>2.0	0.2	0.4
RV/TC (%)	<40	69	54
7 min. N_2 Index (%)	<2.5	10	17
Art. O_2 Saturation (%)	95–98	89	89

Fig. 1. Immediate response to a bronchodilator aerosol in a patient with asthmatic bronchitis and pulmonary emphysema.

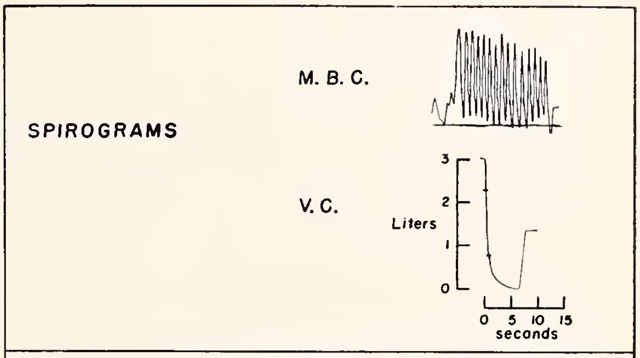
3. The third procedure is the determination of maximal breathing capacity by having the subject breathe as deeply and rapidly as possible for about 15 seconds, as illustrated in Figures 1 and 2, and measuring the volume of gas expired in liters per minute. Maximal breathing capacity is a test of the over-all "bellows" apparatus and may be impaired by skeletal and neuromuscular abnormalities as well as by pulmonary disease. However, only a part of the vital capacity is normally used in this type of rapid and deep breathing, so that relatively large or normal values may be achieved if there is minimal obstruction of the airway, even though the vital capacity is reduced, as for example, in certain types of pulmonary fibrosis and in pulmonary congestion. This is illustrated in Figure 2.

The preceding tests may be used before and after the administration of a bronchodilator drug to assess the extent of readily reversible changes in chronic bronchitis and emphysema, illustrated in Figure 1. It will be noted in this particular case that considerable ventilatory improvement was demonstrated and that this was accompanied by subjective reduction of dyspnea. However, the ventilatory tests still gave grossly abnormal results after treatment, and other measurements, which will be discussed below, failed to show improvement.

Measurement of the residual volume (the volume of gas remaining in the lungs after maximal expiration) must be done by more complex techniques. One standard method is to have the subject breathe 100 per cent oxygen for seven minutes, during which time the nitrogen normally present in the lungs (at a concentration of about 80 per cent) is progressively washed from them into an external bag or spirometer, where its volume may be measured. Normally, a seven-minute period of breathing oxygen will reduce the alveolar concentration of nitrogen to less than 2.5 per cent, but if large portions of the lung are poorly ventilated, their concentration of nitrogen will be reduced less rapidly, so that abnormally high concentrations of nitrogen can be detected by analysis of the alveolar gas delivered by a forced expiration after the seven-minute period. The measurement of residual volume and the detection of retarded alveolar ventilation are facilitated by the use of instruments that have been developed recently for continuous automatic analysis of gases, especially if considerable numbers of patients are to be studied. In emphysema the residual volume is increased both in absolute volume and in its percentage (RV/TC) of the total pulmonary capacity (residual volume plus vital capacity), and the seven-minute nitrogen index is usually elevated (Fig. 1). The case of fibrosis illustrated in Figure 2, in contrast, shows a reduced total capacity and a small nitrogen index.

The preceding tests indicate the performance of both lungs together. However, it may be of assistance to know the relative performance of each lung in situations in which total pulmonary reserve is reduced by bilateral disease and resection is contemplated. One healthy lung can meet the cardiorespiratory requirements, whereas one diseased lung may not. A diseased lung, although appearing aerated on roentgenograms of the thorax, may be contributing either relatively little or much to the over-all exchange of gases, and its removal may produce, respectively, a small or a large impairment. The relative performance of the two lungs can be assessed by bronchospirometry, a method of intubating the trachea and left main bronchus and measuring the vital capacity, ventilation and oxygen consumption of each lung separately.

It is more difficult to evaluate the efficiency with which the lungs maintain the levels of oxygen and carbon dioxide in the arterial blood. Unless arterial hypoxemia is of considerable magnitude, cyanosis (which also depends on other local factors) is detected with difficulty. Ear oximeters, which measure arterial oxygen saturation by the optical characteristics of the blood in the transilluminated pinna of the ear, are useful in the detection of arterial hypoxemia. They can eliminate the necessity for direct chemical analysis of blood obtained by arterial puncture in many cases of suspected hypoxemia, especially if maximal accuracy is achieved by the use of suitable recording apparatus.



TEST	NORMAL	——
Vital Capacity (L.)	4.9	3.1
Max. Breathing Capacity (L./minute)	116	100
Max. Midexpiratory Flow (L./second)	> 2.0	2.5
Total Capacity (L.)	6.6	3.9
7 minute N_2 Index (%)	< 2.5	0.3
Arterial O_2 Saturation (%)	95-98	
Rest		98
Exercise		90

Fig. 2. Results of pulmonary-function tests in a patient with pulmonary fibrosis.

Hypoxemia may be produced by one or several of these factors: (1) venous blood passing through poorly ventilated parts of the lung, (2) reduction of pulmonary diffusing capacity, (3) anatomic venous-arterial shunts and (4) depression of total minute volume of alveolar ventilation. The arterial oxygen saturation during or immediately after exercise may be decreased even though normal levels are maintained at rest. This is frequent in certain kinds of pulmonary fibrosis which reduce the diffusing capacity of the lungs without major impairment of the ventilatory capacity, as illustrated in Figure 2. The measurement of arterial oxygen saturation is also useful

(Continued on Page 658)

Clinical Importance of Hypercapnia

E. B. BROWN, JR., and FLETCHER MILLER
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UNTIL recently, little clinical importance was attached to the effects of alveolar carbon dioxide concentrations in excess of 10 per cent. This was true because it was assumed that a reduction in respiratory ventilation sufficient to produce a significant increase in alveolar carbon dioxide tension would produce a much more critical decrease in oxygen tension. In 1926, Van Slyke wrote, "Medical and lay literature contain numerous cases of death from oxygen lack; in respiratory disease, in high altitude, in carbon monoxide poisoning, but it is doubtful that it contains any case of death due primarily to impaired respiratory ability to excrete carbonic acid." Certainly, it is still true that anoxia is of paramount importance in cases of hypoventilation with the patient breathing air; however, there are conditions under which carbon dioxide can accumulate to very high concentrations without concomitant anoxia.

It will be the purpose of this paper to discuss the conditions which are favorable for the development of this situation, to point out certain clinical situations in which these conditions are likely to be encountered, and to mention some of the possible sequelae of severe hypercapnia when it does occur.

Conditions Favoring Carbon Dioxide Retention Without Anoxia

Draper, et al[1,2] have demonstrated that dogs will maintain adequate arterial blood oxygenation for thirty to forty-five minutes in the absence of any respiratory movement if the dogs have breathed 100 per cent oxygen for forty-five minutes prior to the respiratory arrest, and if a patent airway is maintained during the arrest with oxygen as the ambient gas. During this time, alveolar carbon dioxide concentration regularly rises to 40 per cent or higher. They have called this phenomenon "Diffusion Respiration." Under these conditions, oxygen continues to diffuse into the mixed venous blood presented to the pulmonary capillaries since the alveolar oxygen tension is high. However, within the first few seconds of respiratory arrest, the alveolar carbon dioxide tension reaches the level of that in the mixed venous blood. As metabolism continues, the carbon dioxide concentration increases in the tissues, body fluids, and alveolar air practically simultaneously with the major part remaining dissolved in the tissues and blood. Under these conditions, very little of the CO_2 produced is delivered into the lungs because of the low gradient between blood and alveolar air. Since oxygen is being removed from the lungs at its normal rate, the volume of air in the lungs diminishes and more of the ambient gas mixture is drawn into the lungs through the patent airway, replacing in large part the oxygen being absorbed by the blood.

These studies indicate that the conditions favorable for retention of carbon dioxide without anoxia are the following:[3]

1. Inadequate alveolar ventilation.
2. Replacement of the nitrogen of the body with oxygen.
3. High concentration of oxygen in the air available for movement into the lungs.
4. Patent airway.

These conditions are satisfied, partially at least, when a patient has been breathing oxygen, or a mixture containing a high percentage of oxygen, for thirty minutes to an hour and then is subjected to a reduction in respiratory ventilation.

Hypercapnia Under Clinical Conditions

In 1950, Beecher[4] reported studies in which he had made measurements of arterial blood carbon dioxide tension during several types of thoracic surgery. In sixteen patients upon whom pneumonectomies were performed, he found an

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Much of the work reported in this paper was supported by grants from the Minnesota Heart Association and the Graduate School of the University of Minnesota.

TABLE I. SUMMARY OF HIGHEST ALVEOLAR CO_2 VALUES WITH pH CORRELATION DURING FIFTY-THREE MAJOR SURGICAL PROCEDURES

Alveolar CO_2 Level	No. of Cases	Percentage of Total Series	Estimated pH of Arterial Blood
10-15% CO_2	14	26.4	7.16-6.95
15% CO_2 or greater	4	7.5	6.95-or less

average alveolar carbon dioxide concentration of 13.7 per cent with an arterial blood pH of 7.09 at some time during the surgery. He stressed position of the patient and the type of surgical procedure being performed as important factors favoring the development of hypercapnia under these conditions. Miller,[5] using a mass spectrometer to record alveolar carbon dioxide concentrations, found elevated tensions in patients undergoing other types of surgery as well as thoracotomies. Table I presents a summary of these data. The pH values in this table are not determined values but are estimated from the CO_2 tensions. It can be seen that 26 per cent of the patients undergoing general anesthetization and major surgery experienced alveolar carbon dioxide concentrations of 10 to 15 per cent during some fifteen-minute period of the operation. We would expect this hypercapnia to be associated with a pH depression to values of 7.16 to 6.95. In 7.5 per cent of the cases, a carbon dioxide level compatible with an arterial pH of 6.95 or lower was observed. These cases were representative of cases from a general surgical service and included laparotomies, thoracotomies, and craniotomies. In most instances, the anesthesiologist was unaware of the state of the carbon dioxide level during the surgery.

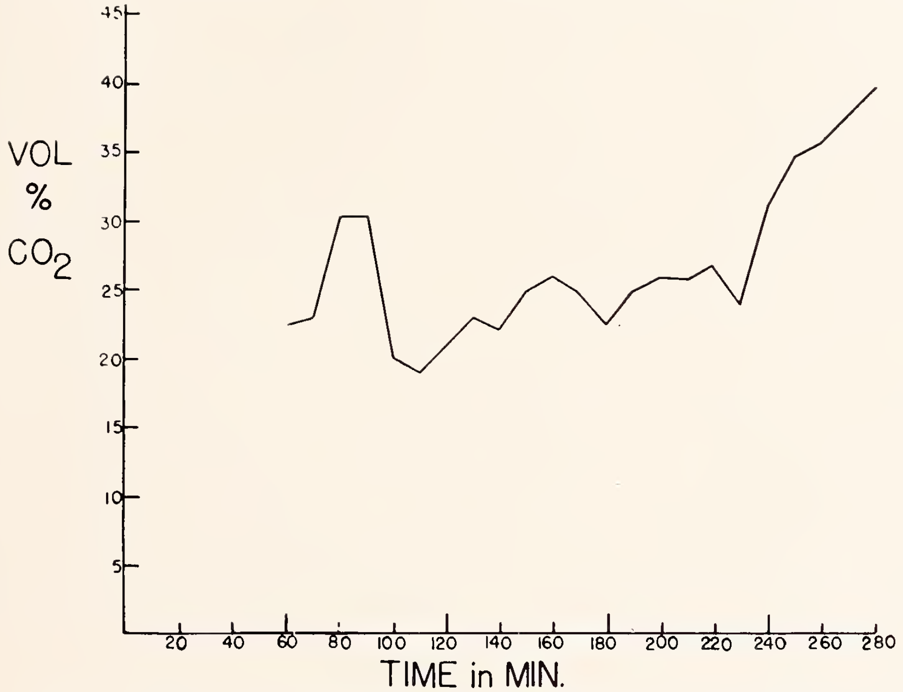

Fig. 1. Alveolar CO_2 levels during pneumonectomy. No recordings were obtained during the first 50 minutes. Despite his knowledge of the mounting CO_2 level, the anesthesiologist was unable to correct the situation with vigorous positive pressure respiratory supplementation. (Reproduced from "Surgical Forum" 1950. By permission of W. B. Saunders Co., Philadelphia).

In a series of seventeen minor operations performed under cyclopropane anesthesia, Buckley et al[6] found alveolar carbon dioxide concentrations as high as 20 per cent in two patients. The average of the highest levels reached during the operation in this series of patients was 12.3 per cent. None of the patients maintained a normal alveolar carbon dioxide concentration throughout the procedure.

Van Bergen et al[7] have also established that a significant respiratory acidosis is often present in poliomyelitis patients being ventilated artificially while breathing high oxygen mixtures.

Sequelae of Severe Hypercapnia

What are the results of such degrees of hypercapnia? The only clinical evidence of the presence of this severe respiratory acidosis may be an elevation of blood pressure. However, when the conditions producing the inadequate ventilation are removed and the excess carbon dioxide of the alveolar air and arterial blood is rapidly blown off, profound cardiovascular changes may ensue. It has been demonstrated that dogs will usually tolerate 30 to 40 per cent carbon dioxide in oxygen for four hours or longer with little evidence of cardiovascular damage as evidenced by electrocardiograms and blood pressure.[8] However, when these dogs are returned to air breathing, marked cardiac arrhythmias appear within a few seconds; the blood pressure falls, and ventricular fibrillation and death follow within three to seven minutes in a high percentage of the animals. The only procedures which have thus far been effective in preventing the cardiovascular complications in these animals in our hands are: (1) lowering the inhaled carbon dioxide concentration gradually, (2) maintaining the blood pressure by means of sympathomimetic drugs during the interval of sudden carbon dioxide depression.

Figure 1 demonstrates the hypercapnia that developed during a pneumonectomy for bronchiectasis. The anesthesiologist was unable to prevent this carbon dioxide accumulation even by vigorous manual ventilation. When the patient was removed to the postoperative recovery room a condition suddenly developed which was interpreted as a cardiac arrest; blood pressure was unobtainable, and the heart beat could not be heard. Fortunately, by prompt use of artificial respiration and sympathomimetic drugs, a disaster was prevented, and the patient recovered without further difficulty. While this is the only well-documented instance of clinical posthypercapnic arrest, a survey of our hospital death files revealed several cases in which this might have been the underlying mechanism. Schumacker and Hampton[9] reported a series of deaths which occurred either during the introduction or removal of the intratracheal tube, a period during which one might suspect that mechanisms similar to the "posthypercapnic phenomenon" were operating.

In his series of patients with hypercapnia during cyclopropane anesthesia, Buckley[6] showed that most of them exhibited an alarming hypotension following the cessation of surgery and anesthesia. This probably merely represents a milder facet of the cardiovascular impairment associated with the "posthypercapnic phenomenon." In fact, Dripps[10] feels that the so-called cyclopropane shock is always a resultant of the associated respiratory acidosis rather than any specific toxic property of cyclopropane. This idea is well substantiated in Buckley's data which showed only two of fifteen patients exhibiting significant hypotension following cyclopropane when adequate respiratory ventilation was carried out and carbon dioxide accumulation was prevented during the period of anesthetization.

Conclusions

Hypercapnia can and does occur clinically in association with conditions of respiratory depression when high oxygen mixtures are utilized to combat the ventilatory deficiency. When such a condition develops it is capable of producing a serious cardiovascular impairment if it is rapidly reversed. The best method of combating this potentially disastrous phenomenon is the assiduous supplementation of respiration so as to prevent the respiratory acidosis from occurring. However, when it does develop the carbon dioxide tension of the alveolar air should be lowered slowly rather than rapidly.

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Acute Pulmonary Edema

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THE currently widespread clinical belief that pulmonary edema is always due to isolated or disproportionate failure of the left ventricle is not actually supported by the experimental studies that purport to confirm it. As was shown elsewhere,[1] the reported work is either inconclusive or else faulty in conception or interpretation. An excellent negative study, that of Meyers et al.,[2] showed that experimentally produced necrosis of the left ventricle causes no increase in pulmonary blood volume. Similarly, the volume has not been consistently found to be increased in patients with cardiac failure generally believed to be due to left-ventricular failure.[3]

There are also certain clinical facts that raise doubts concerning the validity of the left-ventricular-failure theory. Among these facts are: (a) that acute pulmonary edema occurs in chronic cor pulmonale or with pulmonary embolization, (b) that the incidence of the syndrome is much higher with aortic regurgitation than with aortic stenosis, (c) that its incidence is higher in hypertensive crises than in chronic hypertension of similar degree, et cetera. Re-examination of the available clinical and physiologic data should lead to a better understanding of the nature of the syndrome.

Clinical Manifestations

The very beginning of an attack of pulmonary edema is usually not detected or is recognized only in retrospect. When an attack is developing, the patient may have no premonitory sensations; on the other hand, he may first notice a feeling of marked anxiety several minutes (occasionally, several hours) before dyspnea develops. The physician may sometimes misinterpret this anxiety as the cause rather than as the earliest symptom of the attack. Physical examination at such times reveals pallor, an anxious look, and increase in pulse rate and arterial pressure above the patient's normal range; the skin is usually cold and slightly sweaty. The hypertension may be misinterpreted as evidence of a hypertensive crisis that might have caused the attack. It is evident that these signs and symptoms are not specific for pulmonary edema; they can be established as part of the syndrome only if roentgenographic studies show typical areas of haziness close to the hilar regions of the lungs.

The interstitial phase occurs next. In this phase the nonspecific manifestations of the first phase persist, but they are masked by the development of dyspnea, which increases rapidly in severity, and orthopnea, which is usually pronounced. Examination reveals pallor of the skin, now combined with cyanosis. The pulse rate is still accelerated; the arterial pressure either is elevated, as at the onset, or has returned to the patient's normal level. The neck veins may be distended. There is striking prolongation of the expiratory phase of respiration. Wheezing, accompanied by musical râles and rhonchi, is marked in many cases but may be absent in others. Crackling râles are few and inconstant or are absent entirely.

This phase of the attack may last for many minutes or a few hours; on the other hand, the period in which the lungs remain free of detectable intra-alveolar fluid may be so brief as to be unmeasurable. The interstitial phase may proceed abruptly into the phase of alveolar transudation or may be terminated by spontaneous remission or by treatment.

Laboratory studies show rises in hematocrit, erythrocyte count, and hemoglobin level; a lesser rise in plasma protein level may be noted. The circulation time is slowed; a few observations of cardiac output have revealed lowered values for blood flow through the lungs. The vital capacity is lowered markedly, and the arterial blood oxygen saturation may fall to between 70 and 85

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per cent. The right auricular and peripheral venous pressures are elevated, but not necessarily above the upper limit of normal.

The intra-alveolar phase occurs next in patients in whom remission did not occur in the previous phase. The severity of intra-alveolar accumulation of fluid varies markedly; in some patients there is only a little fluid, which is revealed by fine scattered râles, whereas in others bubbling sounds may be audible. The time of onset of this phase varies also; it may occur a few seconds or several hours after the first feeling of anxiety, or in some cases it may not appear at all. The accumulation of fluid in the alveoli and small airways usually aggravates dyspnea and causes coughing. The amount of sputum produced ranges from a few cubic centimeters to a liter or more. Small amounts of mucus, varying quantities of blood, and considerable air are mixed with the plasma that is coughed up.

Examination of such patients shows moderate to marked cyanosis. Tachycardia is present; the arterial pressure is approximately at the patient's usual level. The neck veins are distended. Expiration is prolonged, and crackling or bubbling râles are audible over most of the chest. Wheezing, with its musical râles, may be present also. The crackling and musical râles are not necessarily uniform in distribution; they may be more numerous on one side or over one lobe.

This phase of the attack may be followed by remission—either spontaneous or due to therapy. In many instances, however, shock develops. This too may be either spontaneous or due to the use of tourniquets, positive pressure respiration, or venesection. In a few cases the bubbling râles disappear and the patient reverts to the interstitial phase, with or without wheezing.

Laboratory studies on patients in this state are few and fragmentary. The changes are similar to those described for the interstitial phase but may be more marked.

Shock is common in pulmonary edema. It may be caused by the factor responsible for the edema—myocardial infarction, for example—or may be a consequence of either the pulmonary edema or its treatment.

The development of shock is ushered in by clouding of consciousness in the patient. Although hyperpnea is still evident, dyspnea is not prominent and orthopnea disappears. The pulse rate increases and arterial pressure falls. The veins are narrow and flat. Pallor, grayish cyanosis, and clammy sweating are marked. Examination of the lungs shows signs similar to those of the earlier stages of the attack.

A few measurements of venous pressure have been made in patients with shock associated with pulmonary edema. Values in the normal range occur with mild shock, but very low pressures are found in severe collapse.

The mortality rate is high in patients who have both pulmonary edema and shock. A few patients recover spontaneously; there is no strong evidence that available therapy predictably influences the outcome. The longer the shock persists, the poorer the prognosis.

In the terminal phase deepening of shock, development of coma, and the appearance of cardiac and respiratory arrhythmias indicate impending death.

Basic Physiologic Considerations

The following factors apparently constitute important influences on pulmonary fluid dynamics.

Plasma Protein Level.—There is ample evidence that hypoproteinemia causes increased pulmonary lymphatic flow. No experiments have yet demonstrated that lowered plasma protein level of itself causes overt pulmonary edema. This factor is probably of contributory importance in the pulmonary edema observed in nephritis, cirrhosis of the liver, beri-beri, and some cases of longstanding cardiac decompensation.

Pulmonary Blood Flow.—No experimental study has shown that pulmonary edema is caused solely by increases or decreases in pulmonary blood flow within a reasonable range. On the other hand, there is good evidence that changes in pulmonary blood flow strongly influence the amount of pulmonary edema. Increase in inflow into or decrease of outflow from the lungs aggravates pulmonary edema; changes in the opposite direction ameliorate it. These phenomena are of the greatest importance in clinical pulmonary edema in that they are basic to some of the most effective forms of therapy; tourniquets, venesection, and positive pressure respiration decrease pulmonary inflow, whereas vasodilator drugs increase outflow. In some cases of mitral stenosis, when chronic congestive failure has caused diminution of flow into and through the lungs, overt attacks of pulmonary edema may cease.

Pulmonary Filtration Pressure.—It is well established that increases in pulmonary capillary pressure aggravate pulmonary edema. However, there is no exact correlation between capillary pressures and the occurrence of the syndrome; this circumstance demonstrates that other factors also are important. Although any rise above the normal capillary pressure causes increased transudation into the tissues, the fluid so filtered does not accumulate unless the rate of transudation is excessive. This excessive rate may be the result of *extremely* high capillary pressure or of a variety of other factors. Increase in capillary pressure is always a contributory cause of pulmonary edema; if extremely large it may be the sole cause. As far as is now known, rises in pulmonary capillary pressure are due solely to rises in pulmonary venous pressure, although pulmonary arteriolar dilation theoretically might have some similar effect. Pulmonary arteriolar constriction tends to lower capillary pressure—or at least minimize its rise. Pulmonary capillary pressure is known to be elevated in three groups of clinical syndromes: chronic congestive failure, mitral valvular disease, and pericarditis. It may possibly be elevated in other diseases.

Pulmonary Venular Pressure. — Pulmonary venular pressure largely determines the pulmonary capillary pressure; the latter must always be slightly higher than the former. Elevation of pulmonary venular pressure by partially ligating the pulmonary veins or inflating balloons in the left auricle causes congestion of the lungs. Detectable pulmonary edema does not develop unless the degree of venous obstruction is extreme—presumably because stasis causes a decrease in pulmonary blood flow.

Other mechanisms of pulmonary venous hypertension that have been revealed by studies in animals are the influence of neurogenic stimuli on the vasomotor system and the effect of histamine on pulmonary venules. These are discussed below.

Available measurements show high pulmonary venous (or left auricular) pressure in patients with mitral disease, and similar—though commonly less marked—changes are found in chronic congestive failure. Their etiologic importance in pulmonary edema is evident. In addition, it is possible that they constitute an important mechanism in the pulmonary edema associated with a variety of intracranial lesions.

Pulmonary Vasomotor Changes.—Increased pulmonary arterial pressure, evidently caused by vasoconstriction at least part of the time, occurs in some forms of experimentally induced pulmonary edema, in anoxia, and during intravenous infusions. This phenomenon may lower the capillary pressure slightly or at least prevent it from rising. More importantly, it causes the right ventricle to expend so much work against pressure that it is unable to increase its output. It is evident, therefore, that pulmonary arteriolar constriction protects the lungs against the development or exacerbation of pulmonary edema. Recent experiments by van Bogaert et al.[4] indicate that this mechanism may operate where pulmonary venous pressure rises. It is not established whether pulmonary arterial hypertension, like peripheral hypertension, depresses the heart and lowers its output through reflex action.

Some types of experimental pulmonary edema —such as that induced by phosgene—are not accompanied by pulmonary arterial hypertension except terminally. Moreover, it is not certain that the observed rises in pulmonary arteriolar pressure in other types of pulmonary edema are entirely due to vasoconstriction—that is, are significantly greater than rises in pulmonary capillary pressures.

It is not yet established that pulmonary venular constriction can be .caused by vasomotor activity. Some data obtained in animals with pulmonary edema reveal increases in pulmonary venous pressure that might be due to venoconstriction. Additional fragmentary data pertinent to this problem are to be found in observations that pulmonary capillary pressure may fall after administration of drugs that dilate smooth muscle (i.e., aminophylline) or paralyze autonomic reflexes and thereby presumably dilate pulmonary venules.

Recent work on histaminergic nerve fibers may prove highly important in relation to pulmonary edema. Histamine, normally present in the lungs in a combined form, is freed during the experimental production of pulmonary edema. Epinephrine has this effect; it also increases the blood histamine content. Histamine constricts the pulmonary venules but not the arterioles, in contradistinction to epinephrine, which constricts both. Acetylcholine resembles histamine in this respect.

The fact that adrenolytic agents inhibit the development of pulmonary edema in animals strongly suggests that vasomotor influences may be important in the precipitation of the syndrome. Many studies of these phenomena must be made before valid conclusions may be drawn.

The pathways that carry vasomotor impulses to the lungs have not yet been delineated. Evidence has been published that the pre-optic hypothalamic nuclei are involved in the production of pulmonary edema in animals, but this work awaits corroboration. The concept that vasomotor centers in the brain may play a role in pulmonary edema raises the possibility that reflexes originating in the viscera and passing through these centers might play a significant role in the development of pulmonary edema. In addition, it is theoretically possible that damage to these centers by infection, hemorrhage, anoxia, or toxins might distort such visceral reflexes, thereby precipitating pulmonary edema, or might cause edema solely through irritation of the centers themselves.

Capillary Permeability.—Currently available methods cannot yield precise data *in vivo* on the physical characteristics of a capillary bed as extensive as that of the lungs. Accordingly, published information on the degree of its permeability to protein in experimental pulmonary edema is based on indirect evidence. The pulmonary edema of experimental alpha-naphthyl-thiourea intoxication and of phosgene inhalation is generally believed to be due to capillary damage. In the case of the latter, available data apparently rule out increased capillary pressure as the chief factor in its production. Burns cause pulmonary edema by damaging capillaries in the lungs. Anoxia also causes increased pulmonary capillary permeability—at least it causes an increased elaboration of fluid in the lungs that is not due to vasomotor changes; a recent study suggests that there may also be some venular constriction in anoxia.[5] At any rate, pulmonary arteriolar constriction, the only vasomotor change definitely shown to occur in anoxia, minimizes tendencies toward formation of pulmonary edema.

Increased capillary permeability seems established as an important causative mechanism in pulmonary edema due to anoxia and to inhalation of toxic gases. Whether it contributes to pulmonary edema in inflammatory lesions of the lungs is a matter of conjecture.

Area of Pulmonary Capillary Filtering Surface.—No data are available concerning how filtration is affected by changes in the area of the total capillary filtering surface in the lungs. It may be taken for granted that filtration is altered by these changes.

Significant increases in pulmonary capillary filtering surface are probably caused by stasis due to high pulmonary venous pressures, as in some of the conditions enumerated above. In addition, increases in total circulating blood volume, as in chronic congestive failure, might have similar effects. It is also possible that blood redistribution resulting from peripheral vasoconstriction in the absence of equal (or any) pulmonary vasoconstriction increases the amount of blood in the lungs and hence also the area of filtering surface. This phenomenon might possibly occur in hypertensive crises or during a febrile reaction; increases in thoracic-duct and cervical-duct lymph flow have been described in animals with fever.

Pulmonary Lymphatic Function.—Experiments in animals have demonstrated that obstruction of pulmonary lymphatic flow aggravates pulmonary edema. One known cause of such obstruction is elevation of pressure in the peripheral venous system, into which all lymphatics ultimately drain. Extensive inflammatory lymphatic thrombosis is almost certainly another cause, but no observations have been published on this phenomenon.

Pulmonary edema in chronic cardiac decompensation is probably aggravated by elevation of peripheral venous pressure, with its secondary consequence of increased pulmonary lymphatic pressure. In addition, inflammatory thrombosis of lymphatics probably contributes to the pulmonary edema encountered in pneumonias—especially those of the interstitial variety.

Bronchial Obstruction.—Several authors have shown that bronchial obstruction accelerates transudation. The mechanism of this change is not known. However, one suggested explanation is based on the fact that forceful inspiration against partial bronchial obstruction increases the negativity of the pressure within the parenchyma—that is, lowers tissue pressure (which ordinarily resists transudation). Bronchial obstruction causes only a small or moderate increase in extracellular fluid. It is interesting that bronchospasm may be caused by reflexes from the carotid sinuses.

It is possible that the pulmonary edema encountered in some patients with cor pulmonale is partially due to bronchospasm. However, the latter cannot be a potent factor in this respect, since pulmonary edema is rare in other types of bronchial obstruction.

Pulmonary Extracellular Water.—No studies are available on the normal variations in pulmonary extracellular fluid volume; a considerable increase occurs in cardiac decompensation.[6] Even larger increases are grossly evident in pulmonary edema of any origin. It is reasonable to believe that a generalized bodily increase in extracellular fluid favors the development of pulmonary edema to some extent. Extracellular water accumulates in collagenous tissue; it is established that the proportion of collagen to other tissue in the lungs is one of the two highest in the body, being exceeded only by that of collagen to other tissue in the skin.

A generalized increase in body water is found in chronic cardiac decompensation and in some cases of nephritis, malnutrition, and hepatic cirrhosis; this factor probably contributes to the development of pulmonary edema in some patients with these conditions.

Systemic Arterial Hypertension.—Many animal experiments have been made in attempts to study the effects of increased peripheral resistance on pulmonary edema. The procedures used were of several types: ligation of the aorta, administration of epinephrine and related amines, and irritation of the brain. The first group are worthless in the study of the role of the left ventricle in pulmonary edema, since the techniques used also caused marked impairment of cerebral blood flow.

The use of sympathomimetic amines also fails to yield definite conclusions, since resultant increases in left-ventricular work amount to only 100 or 200 per cent—that is, not enough to cause failure of the ventricle. This is shown by the fact that the left ventricle does not fail when its work is increased 1,000 per cent by other means. Some other mechanism must be sought to account for the pulmonary edema caused by these amines. In this regard it is noteworthy that when the same degree of hypertension is experimentally produced by both epinephrine and nor-epinephrine, the latter is found to be less likely to produce pulmonary edema. When it does produce the syndrome, cardiac failure is not the cause.[7] Among those additional effects of sympathomimetic amines that may be important causative factors in pulmonary edema are the pulmonary vasomotor changes induced by these substances and the arrhythmia-producing effect of epinephrine. Moreover, epinephrine causes the release of histamine in the lungs. Theoretical considerations raise another point: it is possible that hypertension conduces to pulmonary edema through reflexes arising within the ventricles that might act on the pulmonary vasomotor mechanisms. The study of reflexes from within the ventricles themselves has only recently begun, many decades after von Bezold first demonstrated them; information on them is fragmentary at present.

The third group of experiments on the relation of peripheral arterial hypertension to pulmonary edema involves the effects of irritation of the brain on peripheral vascular mechanisms. Interpretation of the results of these experiments is impossible because of the simultaneous pulmonary vasomotor changes that occur after the brain has been damaged.

The etiologic role in pulmonary edema of agents or mechanisms that cause peripheral arterial constriction remains to be clarified. It is established, however, that pressor drugs may be given to patients with pulmonary edema without aggravation of that syndrome. Whatever mechanisms are ultimately found, it is clinically evident that hypertension is important in the genesis of pulmonary edema.

Ventricular Damage.—The earlier experiments involving ventricular damage showed that lesions of the left ventricle produced pulmonary edema in animals. Early work also showed that lesions of the right ventricle had the same effect. The more recent work of Meyers et al.[2] showed no increase in pulmonary blood volume after induced necrosis of the left ventricle. Whether myocardial failure occurred in these experiments cannot be ascertained; it is known that the heart cannot be made to fail by any but the most extreme traumatic damage. In addition, the edema observed in all these experiments cannot be interpreted as due to cardiac failure, since pulmonary edema may occur with experimental lesions of either ventricle. There is as yet no explanation for this edema; reflexes of the von Bezold type, acting on the pulmonary circulation, might account for the experimental results reported. Considerable work remains to be done along these lines.

State of the Respiratory Centers.—Published reports have described the amelioration or prevention of experimental pulmonary edema by means of respiratory depressant drugs; the view has been expressed that the state of the respiratory center may in some way influence the vasomotor mechanisms in the lungs. On the other hand, respiratory depression produced by experimental cerebral damage appears to have no inhibiting effect on the development of pulmonary edema.

The single most useful agent in the control of pulmonary edema in man is morphine; its action in this respect has not yet been explained.

Summary.—Available physiologic data suggest that the pulmonary edema encountered clinically is caused by the summation of submaximal changes in several complexly interrelated physiologic phenomena, with one mechanism or another playing a dominant part in different situations. The fundamental disorder seems to be a rate of transudation from the pulmonary capillaries that exceeds the reabsorptive capacity of the pulmonary lymphatics. The factors that contribute to this imbalance are:

I. Increased transudation
    A. Elevated capillary pressure in lungs
        1. Cardiac decompensation and mitral disease
        2. Venular constriction
            a. Neurogenic
            b. Histamine?
    B. Increased filtering area in lungs
        1. Increased blood volume
        2. Redistribution of blood
            a. Peripheral vasoconstriction
    C. Large blood flow in lungs
    D. Lowered plasma protein level
    E. Increased capillary permeability
        1. Anoxia
        2. Histamine?
        3. Toxins
    F. Bronchospasm
II. Decreased reabsorption
    A. Impaired lymphatic function
        1. Elevated systemic venous pressure
        2. Inflammatory thrombosis?
III. Increased total extracellular fluid volume

Pathologic Physiology

Dyspnea.—The dyspnea that occurs in patients with acute pulmonary edema is caused by several mechanisms. The accumulation of fluid in the interstitial tissues renders the lungs abnormally rigid; hence greater respiratory effort is required. This difficulty involves both inspiration and expiration, but is more pronounced in expiration because of the loss of pulmonary elasticity that occurs. When the lungs become inelastic, expiration necessarily becomes a forcible process instead of one consisting largely of relaxation of the respiratory muscles. Rigidity and inelasticity of the lungs impair mixing, and this circumstance may cause some degree of anoxia. Carbon dioxide is more readily diffusible than oxygen; hence carbon dioxide retention usually does not occur.

The bronchospasm that often occurs in acute pulmonary edema causes dyspnea not only by imposing a need for increased respiratory effort but also by causing anoxia. Lungs in which bronchospasm is present become temporarily emphysematous owing to trapping of air; mixing is accordingly impaired. In severe bronchospasm the amount of air reaching the alveoli may be diminished also.

The accumulation of edema fluid in the alveoli and airways greatly aggravates the anoxia by preventing ingress of air. In addition, trapping of minute air bubbles in the edema fluid results in the formation of a foam that resists expulsion.

Nocturnal Occurrence of Attacks.—The fact that many attacks of pulmonary edema occur at night (or, more accurately, during sleep) has not been completely explained. Several mechanisms that are important in this regard are known, but others may operate.

The circulating blood volume increases during sleep. During waking hours—and more particularly if the patient is up and about—the venous pressure is elevated above the basal level found during sleep, largely as a consequence of muscular activity. The increase in venous pressure, transmitted back to the tissue capillaries, causes the loss of about half a liter of fluid from the circulation into the tissues. The muscular relaxation that occurs during sleep is accompanied by a fall in venous pressure, and the fluid lost returns to the circulation over a period of two or three hours; the plasma volume increases, the hematocrit falls, and the vital capacity decreases. In effect, the patient gives himself an intravenous infusion during the early hours of sleep; this may precipitate pulmonary edema if a tendency toward the condition is present because of some underlying disease.

Another factor that favors the occurrence of nocturnal attacks is an abnormal vasomotor change that occurs during sleep in patients with congestive heart failure or mitral stenosis. The pulmonary arteriolar vasoconstriction present in these conditions relaxes during sleep; pulmonary blood flow therefore increases, whereas in sleeping normal subjects it decreases. Thus it is evident that the relaxation of the arteriolar constriction removes one of the mechanisms that protect against pulmonary edema.

A third factor is the depression of the respiratory center that occurs during sleep, especially during the earlier hours. Even normal persons show mild or moderate anoxia at such times; patients with impaired pulmonary function probably develop it in more severe degrees. The fact that anoxia causes increased transudation from the pulmonary capillaries is well known.

Fever.—The development of an abnormally elevated temperature is a general indication that heat production exceeds heat dispersal in the body. About five sixths of the heat normally formed in the body is ordinarily dispersed via the skin. Experimental studies in normal persons have shown that diminution of blood flow in the skin is followed after an interval by elevation of the rectal temperature; the cutaneous temperature, of course, falls to or toward that of the ambient air. Cutaneous vasoconstriction occurs regularly during attacks of pulmonary edema; it may be very severe in some cases. In addition, cutaneous flow is slowed, owing to an over-all decrease in cardiac output. It is probable that these phenomena are the cause of the fever that sometimes develops when pulmonary edema persists for many hours.

Shock.—A large amount of blood plasma, containing varying numbers of blood cells, becomes incarcerated in the interstitial tissues of the lungs and is exuded into the alveoli during attacks of pulmonary edema. As much as a liter or more of plasma may be lost from the circulation. Although the shock of acute pulmonary edema is usually due to oligemia, other mechanisms participate in its development. Experiments in animals have shown that severe, protracted anoxia produces shock through some mechanisms not as yet completely characterized. In addition, the shock that occurs in some patients with acute pulmonary edema is due more to the underlying disease—such as myocardial or pulmonary infarction—than to the pulmonary edema itself.

Shock in pulmonary edema may be precipitated by treatment. Morphine may create a tendency toward collapse. Tourniquets, venesection, and positive pressure respiration may also precipitate shock. These procedures are beneficial in pulmonary edema because they decrease the return of blood to the heart; in some instances the fall in cardiac output so induced may precipitate shock in a patient who is already somewhat oligemic and whose cardiac output is low.

Wheezing.—Unilateral novocainization of the vagus nerves in patients with pulmonary edema and wheezes causes the wheezes—but not the crackling rales—to disappear on the side injected. These findings indicate that the wheezing is due to neurogenic bronchospasm. The ultimate origin of the impulses causing the phenomenon is not known.

Spontaneous Remissions.—Nearly all patients suffering from the recurrent type of acute pulmonary edema have spontaneous remission of at least the milder attacks; such remission may also occur in patients who have single attacks of the syndrome in association with conditions such as acute glomerulonephritis, myocardial infarction, hypertensive crises, or acute intracranial lesions. In many instances the remissions are not spontaneous at all, but are due to the patient's accidentally or purposely instituting a therapeutic procedure without medical advice. When a patient with pulmonary edema stands up or sits with feet dependent, the resultant physiologic changes favor amelioration of the edema; these changes are decreased return of blood to the lungs and, after a time, diminution in circulating blood volume.

Another mechanism may also cause spontaneous remissions. The intrapleural pressure probably rises considerably during attacks, owing to loss of pulmonary elasticity and to the increased force and duration of expiratory effort. The return of blood to the heart and lungs is regulated to an important degree by the intrapleural pressure. Loss of the normal negative pressure impedes the venous return; therefore the attack itself causes changes that favor cessation of the attack. Obviously the circulatory changes so induced are not always great enough to cause remission.

Patients who cough up large amounts of sputum during an attack are, in effect, performing a venesection on themselves. This tends to diminish the severity of the attack, although some patients die despite the benefit of this physiologic phenomenon.

Treatment

Morphine, oxygen, aminophylline (by vein), tourniquets, and venesection constitute the traditional therapy of individual attacks. To these have recently been added anti-foaming agents and ganglion-paralyzing drugs. Hilden[8] has recently reported good results with tetraethyl ammonium bromide, and Wilson[9] has found hexamethonium effective. These drugs should, of course, be used with great caution. Digitalis, salt restriction, and mercurial diuretics serve better in the prevention of recurrent attacks of pulmonary edema in patients with chronic cardiac decompensation than in the treatment of the attacks themselves. Acute hypertensive crises accompanied by pulmonary edema are usually relieved by the injection of veratrum derivatives.

The use of the therapeutic measures discussed here is commonly attended by remission of the syndrome. However, a disappointingly large number of patients fail to respond satisfactorily. Because knowledge of the disorder is still incomplete, its treatment requires close observation of the patient, understanding of the mechanisms of therapy, and avoidance of rigid routines—especially those based on *a priori* theories.

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FACTS ABOUT "MIKE SHOT" OF "OPERATION IVY"

1. It was a ground test of a thermonuclear device, not a bomb dropped from the air.

2. It took place at Eniwetok Atoll in the Pacific in November, 1952.

3. The fireball measured 3¼ miles in *diameter.*

4. The *radius* of total destruction was 3 miles.

5. The *radius* of heavy to medium damage was 7 miles.

6. The *radius* of light damage was 10 miles.

7. The blast destroyed the tiny island of Elugelab, leaving a crater roughly 1 mile in diameter and 175 feet deep at the deepest part.

8. After twelve minutes, the cloud was nearly 20 miles high and 100 miles across.

9. Some damage from any similar device would be suffered over an area of somewhat more than 300 square miles—about the area of metropolitan New York City . . . *but*

10. Complete destruction would cover less than 30 square miles—1/100,000th of the surface area of the continental United States.—*Federal Civil Defense Administration.*

Idiopathic Pleural Effusion

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THE ACCUMULATION of fluid in the pleural cavity is a complication of many conditions. In most of these, such as heart disease, renal disease, obvious pulmonary tuberculosis or an acute pneumonic process, careful study will reveal the cause of the effusion. Hydrothorax due to an intrathoracic malignant tumor, pulmonary infarct, subdiaphragmatic disease or lymphoma may be more difficult to recognize but a definite etiologic diagnosis can usually be made. However, effusions may arise without any of these causative factors being present, and these have been called "idiopathic pleural effusions." Experience has shown that they are usually tuberculous and that the physician's responsibility to the patient demands that he regard and treat them as such unless they can be proven to be due to other causes. This conclusion is based on four observations:

1. The tuberculin reaction in patients with idiopathic effusion is almost invariably positive although in normal members of the same age group the positive reaction rate is low.
2. Special studies show tubercle bacilli in the pleural fluid in up to 70 per cent of these patients although routine cultures for tubercle bacilli are positive in only 25 to 30 per cent.
3. Pathologic studies have shown definite tuberculous lesions in the pleuras of such patients.
4. Active pulmonary tuberculosis occurs within five years after such an effusion in from 20 to 60 per cent of reported cases.

Pathogenesis

In young adults, the majority of pleural effusions are of this "idiopathic" type.[1] However, with increasing age, the picture changes so that in patients over the age of fifty, carcinoma and congestive heart failure become the two most common causes of fluid in the chest.[2] Lloyd[3] recently reported the thoracoscopic findings in twenty-three patients with hydrothorax and found malignant tumors producing the fluid in seventeen. He did not, however, give evidence that these effusions were actually "idiopathic" before the thoracoscopic studies, nor did he state the age or tuberculin reaction of his patients.

The pathogenesis of pleural effusion in tuberculosis has been a matter of speculation in much of the recent literature on the subject. Mention has been made of the possibility of a subpleural focus of tuberculous infection involving the pleura either by lymphatic drainage or by direct extension. Caseous mediastinal lymph nodes have also been suggested as the source of the infection, and the possibility of hematogenous spread to the pleura has been proposed.[4-8] However, until recently no good anatomic evidence has been advanced to support any of these theories.

Stead and his co-workers,[9] in a recent study, have done much to clarify this problem and to improve our understanding of the mechanism by which these effusions arise. They reported a series of fifteen patients with tuberculous pleurisy who underwent thoracotomy within a few months of their acute effusions. In each case, they found a chronic granulomatous pleuritis with complete obliteration of the pleural space. Typical caseous tubercles were seen within the thick fibrous peel which encased the lung; and in twelve of the fifteen, acid-fast bacilli were seen in the stained sections.

On examination of the underlying lung, they found in twelve of their cases a caseous tuberculous focus immediately beneath the pleura at which point the peel was so intimately attached to the lung that decortication was impossible without removal of the parenchymal lesion. In the other three cases tuberculous foci were also present within the lung, but the source of pleural contamination was not identified. They postulated that the site of rupture of the parenchymal

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lesion might have been buried in one of the interlobar fissures.

These authors also called attention to some significant articles in the literature which had received little attention previously. These earlier papers described the thoracoscopic appearance of the infected pleura and the results of autopsies performed on several patients with acute idiopathic effusions. Definite tuberculous lesions were regularly found in the lungs at autopsy, and the thoracoscopic appearance in the acute cases was typical of tuberculosis.

The significance of these findings can readily be appreciated in the light of Paterson's work on the experimental production of tuberculous pleural effusion in guinea pigs.[10] The injection of tubercle bacilli into the pleural cavity of a tuberculin-sensitive guinea pig produced a vigorous pleural reaction and an effusion which were followed by a fibrinous pleurisy. A similar intrapleural inoculation in a tuberculin-negative animal resulted in very little pleural reaction but a much higher incidence of disseminated tuberculosis.

The lesions which they saw at surgery plus the experiments of Paterson led Stead and his co-workers to the conclusion that tuberculous effusion is caused by the rupture of a subpleural parenchymal caseous focus into the pleural space. This discharge of infected material into the pleural space of a tuberculin-positive subject results in an actual infection and a vigorous inflammatory reaction of the membrane. This concept was supported by their finding of tubercle bacilli in caseous lesions within the thickened pleura. The possibility still remains that in a few cases effusions may arise by hematogenous seeding of the pleura, especially in those occasional instances where the effiusion is bilateral. There seems to be little doubt, however, that most arise by the sequence of events which they describe.

Clinical Picture

As stated previously, idiopathic pleural effusion is primarily a disease of young adults. The peak incidence is at about age twenty, about ten to twelve years earlier than the peak incidence of pulmonary tuberculosis.[4,11] The fact that many of these effusions occur within a few months of the conversion of the tuberculin reaction is evidence that effusion may frequently be a complication of a primary tuberculous lesion in young adults who have escaped such a primary infection in childhood. In contrast to this is the unexplained observation that pleural effusion rarely develops in children with primary tuberculosis. Primary tuberculosis in the adult is uncommonly associated with enlarged hilar nodes on x-ray.

The clinical picture accompanying an effusion varies widely. There may be an actue fulminating onset with high fever, chest pain, cough and dyspnea, or the patient may be totally asymptomatic and his effusion discovered on a routine chest x-ay. In the majority of cases the disease begins with a mild to moderate chest pain, fever up to 103 degrees, and a slight irritative cough. An acute onset may suggest a bacterial pneumonia, but this can usually be recognized by the clinical picture and bacteriologic findings.

The usual physical findings of dullness and decreased or absent breath sounds and fremitus are well known, but Bernstein and White[12] have called attention to the frequency with which the physical examination may be atypical. They described seventeen patients with pleural effusion of whom eight also had signs of pulmonary consolidation. They postulated that these findings were caused by compression of the underlying lung by massive effusion.

Routine laboratory findings are those of a chronic inflammatory process. Mild leucocytosis may occur but the leucocyte count is usually normal. The sedimentation rate is usaully elevated and correlates roughly with the vigor of the pleural reaction. The pleural fluid is an exudate, usually yellow-amber in color, and may be clear or cloudy. The specific gravity is usually between 1.015 and 1.020 with a protein content of approximately 3 to 5 gm. per 100 ml. Bloody fluid is rare in the white race, but may occur in 2 to 3 per cent of cases. The cells of the exudate are chiefly mononuclear although neutrophils may be present in an early effusion. It has been shown recently that fluids from which tubercle bacilli can be isolated contain significantly less glucose than do fluids from patients with nontuberculous effusions. However, the usefulness of this procedure is limited by the fact that sterile, truly idiopathic effusions show a sugar content in an intermediate, equivocal range.[13,14]

Careful bacteriologic study of the pleural fluid will result in demonstration of tubercle bacilli in only 25 to 30 per cent of cases. Success with routine cultures alone will be considerably less than this, and all such fluids should be inoculated into guinea pigs as well. Repeated attempts will increase the percentage of positives; and it has been suggested that, in cases where the effusion is large, the laboratory be provided with a large volume specimen which can be centrifuged before culture and guinea pig inoculation.[1,4,5,16]

The tuberculin test is of great help as it must either be positive at the time of onset or become so within six to eight weeks, or the diagnosis of tuberculosis can be virtually ruled out. On the other hand, if a history of a previously negative Mantoux can be obtained and an effusion appears coincidentally with conversion to positive, the tuberculous etiology is well established. Roper and Waring[15] found 100 per cent tuberculin reactivity in 141 cases of primary pleural effusion despite the fact that a Mantoux history was usually not obtainable at the time the cases were selected for their series. Oille[1] reported eight of seventy-eight patients Mantoux-negative at first testing, but these all became positive within two months. Kraft[16] described 100 cases in which seventy-seven of the patients reacted to PPD No. 1 and the remaining twenty-three to PPD No. 2.

Since pleural effusion arises from underlying pulmonary tuberculosis, it is also important that bacteriologic study of sputum and gastric washings be done. Mitchell[21] obtained a higher incidence of positive cultures from sputa and gastric washings than from the pleural fluid even though no parenchymal lesions were noted on x-ray.

Lloyd[3] used thoracoscopy and pleural biopsy in cases of pleural effusion where a diagnosis was impossible by other means and found it a safe procedure. He reported no serious adverse effects in twenty-three cases where thoracoscopy and biopsy were done after preliminary aspiration and introduction of air.

Therapy

The therapy of idiopathic effusion may be divided into two aspects: the treatment of the effusion itself and that of the underlying tuberculous process.

The policy as to management of the fluid varies widely in different series. Most authors agree that at least one thoracentesis should be done for diagnostic purposes, but some restrict further aspiration to those instances where the effusion is productive of dyspnea or where the fluid fails to re-absorb in a reasonable time.[1,7] Sibley[5] demonstrated that the incidence of later morbidity and mortality was not affected by aspirations. However, it has been shown that persisting fluid may rapidly result in marked fibrin deposition, pleural thickening, and loss of lung function. This has caused some authors to recommend regular and systematic removal of fluid at least down to the level of the dome of the diaphragm, despite the absence of proof that this favorably effects the incidence of later pleural complications.[4,17,18,19] In the older literature, introduction of air to create an artificial pneumothorax was recommended in an attempt to maintain a free pleural space, but this is now felt to be contraindicated.[6]

Therapy after resolution of the effusion is directed at the underlying tuberculosis. In addition to the subpleural tuberculous foci which gave rise to the effusion, Stead et al[9] found in seven of their cases multiple caseous lesions scattered through the lung. Despite the fact that these were extensive enough to be classed as moderately advanced or far advanced at the time of surgery, they were either unsuspected or their extent was not fully appreciated on preoperative x-rays. This finding, plus the high incidence of subsequent tuberculosis, makes it imperative that these patients receive the same treatment ordinarily advised for a demonstrable pulmonary tuberculosis.

Six months of bed rest is usually recommended after the effusion has disappeared or the fibrotic pleura has become stable on x-ray. This period is followed by four to five months of gradual ambulation, which results in a total of approximately one year in the sanatorium. Roper and Waring showed a reinfection rate of 86 per cent in their patients treated with less than six months of rest, 34 per cent in those treated six months to a year, and only 13 per cent in those with over one year of therapy.[20] Falk and Tucker[22] found found an 8 per cent tuberculous complication rate in patients hospitalized eight to sixteen months as compared with 21 per cent in the group treated less than eight months.

Although less emphasis is being placed on bed rest in the treatment of tuberculosis, it should be stressed that this modification of treatment is still experimental. Most physicians experienced in tuberculosis believe that rest is a very important part of the treatment of tuberculosis. This also applies to pleurisy with effusion since it is simply a manifestation of tuberculosis. Antimicrobial therapy should be used in conjunction with systemic rest in the same manner that it is employed in other forms of pulmonary tuberculosis.

Since the etiology of the pleurisy can be established bacteriologically only in a minority of instances, it may tax the ingenuity of the physician to convince the patient of the need for prolonged hospitalization. This difficulty is further increased by the fact that the fluid usually disappears rather rapidly and the patient frequently feels quite well within a few weeks. Only a physician who is convinced of the tuberculous etiology and the serious significance of such an effusion can sell such a program to the patient.

Prognosis

Frostad[11] reported that 26.4 per cent of patients admitted for tuberculosis gave a history of pleural effusion as the first sign of their disease. Falk and Tucker[22] found a history of previous effusion in 8 per cent of 250 consecutive patients admitted to the hospital for pulmonary tuberculosis, while in patients with skeletal and genito-urinary tuberculosis, they obtained a similar history in 37 per cent and 23 per cent, respectively. These figures again demonstrate the importance of pleural effusions as a precursor of later, more serious, tuberculous infection.

Age seems to be a factor as Falk and Tucker found all of their subsequent relapses occurred in the age group thirty or under. They also showed that non-white patients showed a significantly higher incidence of later tuberculosis, an observation confirmed by Bonilla[8] and by Jones and Dooley.[23] The later authors also found specifically that the subsequent occurrence of extrapulmonary disease was higher in the Negro.

The demonstration of tubercle bacilli in the pleural fluid has been shown by several authors to have little effect on the total number of patients who subsequently develop tuberculosis.[1,5,11,21,22] However, there seems to be a higher incidence of extrapulmonary disease in those patients with positive pleural fluid[22] and also in those bilateral effusions.[7]

Several authors mention the frequent occurrence of extrapulmonary tuberculosis after pleural effusion, the incidence varying between 5 and 25 per cent.[5,8,11,22,23] This indicates that tuberculous bacteremia commonly accompanies tuberculous pleurisy and is probably due to the widespread lymph drainage of the infected pleura. Burke[24] has studied the fate of particulate matter injected into the pleural cavity and has shown that the usual drainage is into the parasternal, para-aortic, and paravertebral nodes. His autopsy data indicate frequent involvement of these nodes in tuberculosis of the chest wall and of the spine following effusion. He has demonstrated the importance of the paravertebral nodes in the pathogenesis of Pott's disease.

The size of the effusion seemed to have little effect on the ultimate development of tuberculosis.[11] However, the presence of a demonstrable pulmonary infiltrate on x-ray was accompanied by a significantly higher morbidity rate.[8] This is probably because in patients with progressive infection demonstrable pulmonary infiltrates tend to develop during the period of observation.

Clinical spread generally becomes manifest within two years of the effusion. Falk and Tucker[22] found that the acute hematogenous complications (miliary, meningeal and peritoneal) occurred within an average of one year after effusion. Comparable average figures for the time of onset of pulmonary, skeletal and genito-urinary tuberculosis were 1.64, 1.85 and 2.57 years, respectively. Frostad[11] found 80 per cent of the morbidity and 84 per cent of the mortality in his patients occurred within five years after the effusion. Mitchell[21] found 93 per cent of recurrences by the end of the fifth year. Frostad quotes Norweign life insurance studies which show that in patients followed for four years after effusion, the mortality from tuberculosis is four times that of the general population. All of these figures tend to emphasize the potential seriousness of this condition and the need, not only for adequate treatment at the time of diagnosis, but also for close follow-up for at least five years.

Summary

1. Many pleural effusions, especially in young adults, arise without an obvious etiology. The evidence for a tuberculous etiology for such effusions is reviewed. "Idiopathic" pleural effusions must be recognized and treated as tuberculosis if the patient is to avoid the danger of subsequent tuberculous complications.

2. Careful laboratory studies, including skin testing and culture and guinea pig inoculation of

pleural fluid, sputum and gastric contents, will frequently point to tuberculosis as the etiology. However, many such effusions never show positive bacteriologic studies, and a negative result does not rule out tuberculosis.

3. Treatment consists of aspiration of all readily obtainable fluid, anti-tuberculous chemotherapy, and bed rest, until the x-ray picture has been stable for at least six months. Patient acceptance of this regimen is dependent on a positive attitude on the part of the physician.

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FARM-CITY WEEK, OCTOBER 23-29, 1955

Each physician and his medical society is invited to plan and participate actively in Farm-City Week, October 23-29, a week-long series of events aimed at bringing about better understanding between rural and urban dwellers.

Under the sponsorship of the Farm-City Conference, an informal alliance of leaders in industry, agriculture, and the professions, Farm-City Week is hoped to be one of the most effective ventures in good citizenship ever undertaken. Member organizations, representing several million people, are going "all out" to make the week national in scope through observances in every community across the country.

The AMA, a conference member, has already sent program plans and suggestions to all medical society officers. Kiwanis International has been selected as the co-ordinating agency, and local Kiwanis club officers have been informed of the invitation to participate in state and community programs.

Because Farm-City Week provides a splendid opportunity for the medical society to assert its civic leadership and to inform the public of its many services, the AMA urges all physicians and societies to get behind this program to help dramatize the mutual need for good relationships between town and country neighbors.

Health and medical care are of uppermost interest in this activity dedicated to urban and rural progress. Medical societies can add greatly to the success of the week by developing health programs for city and farm groups, addressing civic organizations, participating in newspaper, radio and TV interviews, providing tours of medical facilities, and presenting vocational guidance programs in secondary schools. In addition, physicians, as members of the community, should be represented in the general observances of Farm-City Week.

A successful Farm-City Week, October 23-29, depends upon community-wide action. The medical profession—both as individual members and as a professional unit—should accept this challenge.

Individual Contributions

Radioisotope Studies of the Physiology of Calcified Tissues

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On casual view the bones give an impression of rigidity and permanence which belies their dynamic physiology. Prior to the work of Hevesy, who demonstrated[1] about two decades ago, shortly after radiophosphorus first became available, an unexpectedly large and rapid incorporation of this radioisotope in the skeleton, it had been assumed that the bone mineral is resorbed and deposited only as a result of changes in the ionic concentration of the body fluids or of the activity of the osteoblasts and osteoclasts. The studies with radioisotopes have demonstrated that the bone mineral is not a static stone but is a dynamic system in which the elements are constantly turned over and renewed. Because of the rapidity and extent of these processes the skeletal minerals are intimately associated with all other aspects of the transport and metabolism of these elements. The deductions which have been made from the studies with radioisotopes have pointed out an important physiological concept, namely, that the skeleton has a homeostatic function in maintaining and regulating the ionic composition of body fluids which is fundamentally more important than its role as a supporting tissue.

An example of a research which is frequently cited to indicate the high order of rate of turnover of skeletal phosphorus is the work reported by Hevesy, Levi and Rebbe.[2] These workers maintained what was stated to be a constant specific activity of plasma inorganic phosphorus of rabbits over a period of several weeks by spacing of repeated intravenous injections of radiophosphorus as inorganic phosphate. The specific activity* of the bone phosphorus as a percentage of the specific activity of the plasma inorganic phosphorus gave the results in Table I which denote the fractions of the phosphorus in the bones which were renewed in fifty days. For example, about one-fourth of the phosphorus of the femur and tibia epiphyses was replaced in fifty days. Smaller fractions of the phosphorus of long bone diaphyses were replaced but even in this case the quantity of phosphorus involved is considerable.

TABLE I. EXTENT OF REJUVENATION OF RABBIT BONES

In 50 Days	Phosphorus Replaced Per Cent
Femur epiphysis	29.7
Femur diaphysis	6.7
Tibia epiphysis	28.6
Tibia diaphysis	7.6
Ribs	27.5
Scapula	43.8
Incisor dentin (root)	103.0

It is not correct to conclude that bone mineral is dissolved, removed and replaced with newly deposited bone mineral through the operation of the biological processes of bone resorption and bone formation in order to account for the degree of turnover of the bone mineral which is demonstrated by the accumulation of radioisotopes in the skeleton. The mechanism in largest part is one in which the labeled ions, for example phosphate, in the body fluids exchange for and replace an equal number of unlabeled ions in the bone salt. This concept was legitimately drawn by inference from the whole animal experiments but the direct evidence for this conclusion was first obtained from experiments carried out *in vitro*. It was shown that dead calcified tissues rapidly remove large amounts of radiophosphorus from solution without affecting the total concentration of phosphate ion in the solution.[3]

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*Specifiic activity=fraction of injected radioisotope per milligram of the element.

Presented before the Minnesota Academy of Medicine, November 10, 1954.

TABLE II. TURNOVER OF PLASMA CALCIUM AND SODIUM

	C_o C/M/ml.	R_p %
6 animals		
Ca	1184	51.9
Animal No. 14		
Ca	1046	60.1
Na	933	56.4
Animal No. 15		
Ca	613	30.3
Na	586	29.7
Composite of Animals 14 and 15		
Ca	758	38.8
Na	750	43.9

The rate of turnover of skeletal calcium and sodium can be estimated in the living animal without the necessity of maintaining the plasma specific activity constant by following the rate of dilution in the blood of intravenously injected radioisotopes of these ions. This procedure is applicable only to those inorganic ions which are located solely or mainly in bone mineral and extracellular fluid. This method cannot, therefore, be used to measure the rate of turnover of skeletal phosphorus or potassium.

In our experiments[4] dogs received a rapid intravenous injection of a solution containing calcium[45] or both calcium[45] and sodium[22]. Arterial blood samples were obtained, beginning one minute after the injection and at frequent intervals thereafter, from a cannula which had been inserted into the femoral artery (Fig. 1). The concentration of each of the isotopes in the arterial plasma samples was determined and the results are shown by the points on the curves in Figure 1. The concentration of the radioisotopes in the plasma fell rapidly; that of calcium more rapidly than that of sodium and the concentration of radiocalcium continued to fall long after an equilibrium concentration of radiosodium in the plasma had been reached. Obviously equal numbers of the ions must move simultaneously in both directions between adjacent compartments since the concentrations of each of the ions in plasma and interstitial fluid are held constant. The concentrations of the radioisotopes in the plasma fell because of the movement of the labeled ions from plasma to interstitial fluid and from interstitial fluid to bone. The labeled ions in the fluid compartments were thus replaced with a similar number of unlabeled ions.

Equations in three exponential terms for sodium and four exponential terms for calcium were derived from the data to describe the time-concentration curves for each isotope in plasma. The curves shown in Figure 1 are actually the plots of the equations. The success of the curve fitting is shown by the agreement of the observed values, indicated by the points, to the curves.

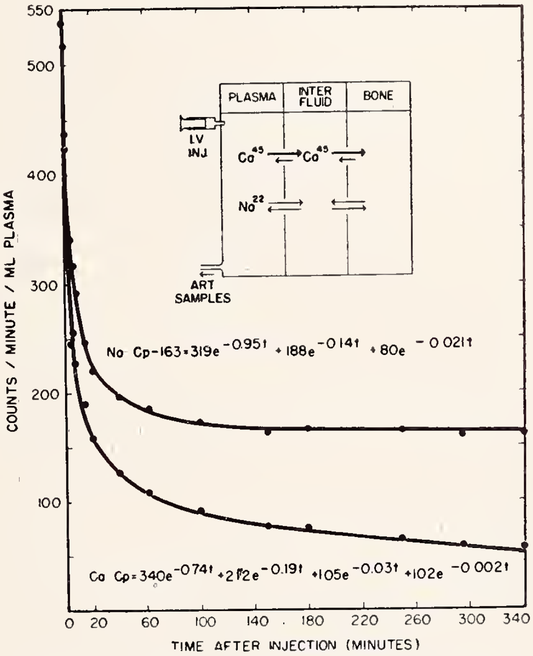

Fig. 1. Diagram of experiment and results showing rates of disappearance of intravenously injected radioisotopes of calcium and sodium.

At any actual time the number of labeled ions in the plasma is determined by the number which have left the plasma and the number which have returned to the plasma. At zero time there are no labeled ions in the interstitial fluid and the change in concentration of the labeled ions at zero time is thus determined only by the number which are transported from plasma to interstitial fluid.

The sum of the first derivatives of the exponential terms in each equation at zero time gives the instantaneous rate of change of the number of labeled ions in the plasma at that time. In effect one obtains by this mathematical device the number of labeled ions which leave the plasma per unit time before any have left. If this number is then divided by the number of labeled ions present in the plasma at zero time the fraction of labeled ions transferred across the vascular membrane per minute is obtained. Since we can assume identical behavior of the labeled and non-labeled ions, this fraction is then identical with the fraction of ordinary plasma calcium and ordinary sodium transferred across the capillary membrane in both directions per minute.

The results as to transcapillary migration rates of calcium and sodium are given in Table II. The averaged results obtained with six animals indicate a turnover and replacement from the interstitial fluid of 52 per cent of the plasma calcium per minute. From the two animals which received both radioisotopes, the not unexpected result of equal rates of transcapillary transport of sodium and calcium was obtained.

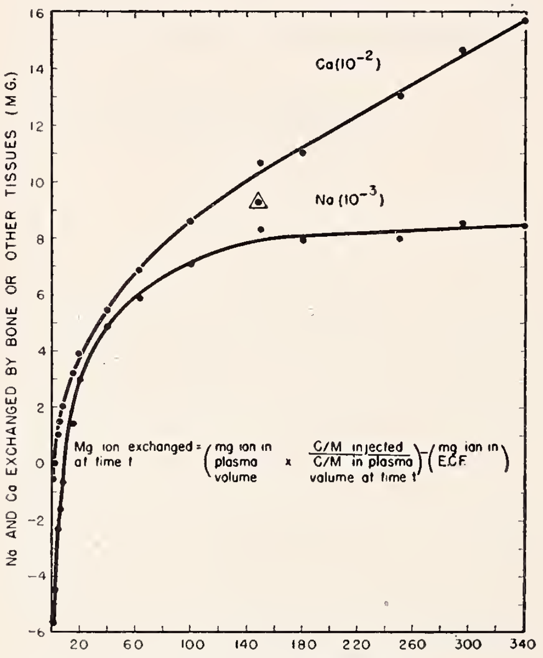

Fig. 2. Turnover of skeletal calcium and sodium in dogs.

As was seen, the intravenously injected radiocalcium and radiosodium in the plasma rapidly became diluted in quantities of calcium and sodium which far exceeded the amounts of these ions present in the extracellular fluid compartments. By use of the isotope dilution equation the data given in Figure 1 were also used to estimate the quantities of skeletal calcium and sodium exchanged at each of the time intervals. It is assumed, upon sound grounds, that bone is the only tissue which can supply significant quantities of calcium or sodium ions to extracellular fluid.

In the equation as used (Fig. 2) the first step was to calculate the total quantity of sodium or calcium through which the injected isotopes were distributed in order to give the observed activity per milligram of each element in the extracellular fluid as sampled by the plasma. The first two terms in the equation gave this result. From this quantity was subtracted the amount of calcium or sodium in extracellular fluid to give the amounts of these elements which had been exchanged by bone at each of the time intervals after the injection.

This equation requires that it be assumed that the specific activity of each labeled ion is equal in plasma and in interstitial fluid at any given time. This assumption is incorrect over the very early minutes after the injection but the error thereby introduced rapidly decreases with time. Certainly by 150 minutes the calculated quantities of exchanged bone calcium and sodium are liable to only a small error.

The results show (Fig. 2) that by 150 minutes the skeleton of a 20 kg. dog had exchanged about 1 gram of calcium and about 8.2 grams of sodium. Both of these quantities are large and indicate that significant amounts of bone mineral calcium and sodium are delivered to the fluid compartments of the body in short periods of time. The quantity of sodium is of the order of half of the entire amount present in the skeleton. The very rapid turnover of skeletal sodium, compared to the slower rate of turnover of skeletal calcium, is consistent with the Hendricks-Hill description[5] of the location of bone sodium as adsorbed ions on the bone mineral crystals.

Through studies with radioisotopes a continual remodeling of bone as a whole and of its mineral phase can be demonstrated. The remodeling occurs by formation and maturation of Haversian systems[6] and by recrystallization of the bone mineral.[7,8] One line of evidence for *in vivo* remodeling of bone, which is at the same time an expression of the result of the remodeling, is the gradual sequestration of originally labile and freely exchangeable radiocalcium into situations in the bone which are remote from the body fluids.

In our experiments[9] radiocalcium was given to mature rats by intraperitoneal injections and the animals were sacrificed at periods of time from one day to 210 days after the administration of the radioisotope. The fractions of the injected radiocalcium present in the entire skeletons of the animals were determined and these results are shown by the heavy descending line in Figure 3. At Day 1 80 per cent of the injected radiocalcium was present in the skeleton. This quantity decreased because of mobilization of the labile skeletal radiocalcium and its excretion until the fiftieth day when about 45 per cent of the injected radiocalcium was still present in the skeleton. After this time there was no further significant decline in the amount of radiocalcium retained in the skeleton. These results indicate that processes of bone remodeling and of bone salt recrystallization which had occurred over the first fifty days had resulted in the relocation of a considerable fraction of the radiocalcium in locations which were inaccessible to the body fluids.

Another constituent of bone mineral which exhibits a rapid rate of turnover is the bone inorganic carbon. Our studies show that the biological half-life of bone inorganic carbon in growing rats is twelve to fifteen days and in mature rats is about thirty to forty days.[10]

A variety of methods have been used to administer radioactive carbondioxide to animals and to build up and maintain the radiocarbon label of body fluid inorganic carbon. In one phase of our work this result was obtained by the continuous intraperitoneal injection of sodium carbonate labeled with radioactive carbon. In one experiment a large rat received sodium carbonate in this manner by injection over a period of 116 hours.[11] When this animal was sacrificed, radiocarbon was found in every tissue examined, and some results are given in Table III. The skeleton as a whole had accumulated about 0.1 per cent of the injected radiocarbon. Since the specific activity of the bone inorganic carbon was higher than that of any other tissue carbon, this carbon had the highest rate of turnover of any fixed carbon in the body. The incisor teeth inorganic carbon is a special case due to the fact that radiocarbon was accumulated by deposition consequent upon growth of the teeth as well as by exchange.

TABLE III. DISTRIBUTION IN TISSUES AND TISSUE COMPONENTS OF RADIOACTIVE CARBON FOLLOWING CONTINUOUS INTRAPERITONEAL INJECTION AS SODIUM CARBONATE

Tissue or Tissue Component	Fraction of Total Dose Per Cent	Specific Activity x 10^6	Relative Specific Activity (Bone Inorg. Carbon = 100)
Bone Inorg. C		715.4 ± 6.25	100.0
Bone	0.1162 ± 0.0006	90.0 ± 0.46	12.6
Bone Protein		25.5 ± 0.41	3.6
Incisor Teeth	0.0016 ± 0.00002	76.9 ± 0.82	10.7
Incisor Teeth Inorg. C.		1138.9 ± 5.3	159.2
Molar Teeth	0.0008 ± 0.00001	51.5 ± 0.68	7.2

Our knowledge of the physiology of the teeth has been notably advanced through studies with radiotracers. The old question of the permeability of enamel and dentin has been answered by the demonstration of a two-way transport of labeled ions and some molecules through these dental hard tissues.

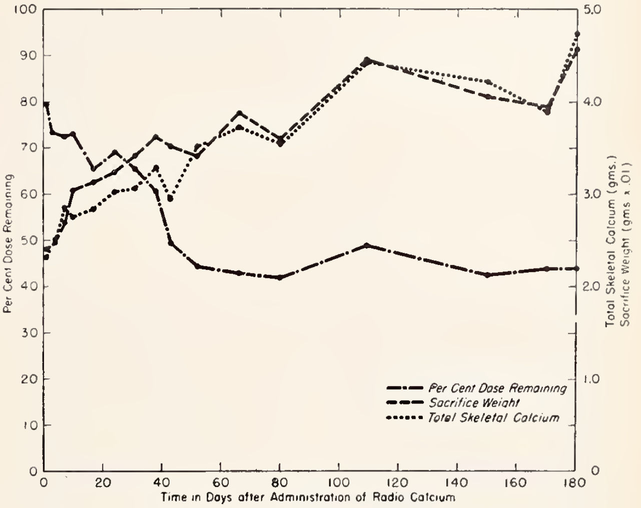

Fig. 3. Retention of radiocalcium by skeleton of rats.

TABLE IV. RADIOISOTOPE UPTAKE BY CALCIFIED TISSUES (RAT—5 DAYS AFTER INJECTION OF Ca^{45} AND P^{32})

	Relative Specific Activity P^{32}	Relative Specific Activity Ca^{45}
Femur epiphyses	80	92
Femur diaphyses	75	82
Molar dentin	14	12
Molar enamel	2.7	1.2

When radioisotopes of phosphorus and calcium are given to experimental animals by injection or by stomach tube,[12] these isotopes can be found in the enamel and dentin. Dentin acquires much smaller amounts of these mineral isotopes than bone and the specific activity of enamel is, in different experiments, of the order of 5 to 20 per cent that of dentin as is shown in Table IV. Whether the avenue of entrance of labeled isotopes into the enamel is from the blood *via* the dentin or from the saliva has been debated. The best evidence is that both avenues are open and operating. In the case of radiocalcium and radiophosphorus it appears that the transport from saliva to enamel is relatively the more important. The migration of ions and molecules through the surface of the enamel and into the teeth can be demonstrated in simple *in vitro* experiments in which the crowns of extracted teeth are immersed in solutions of the radioisotope.

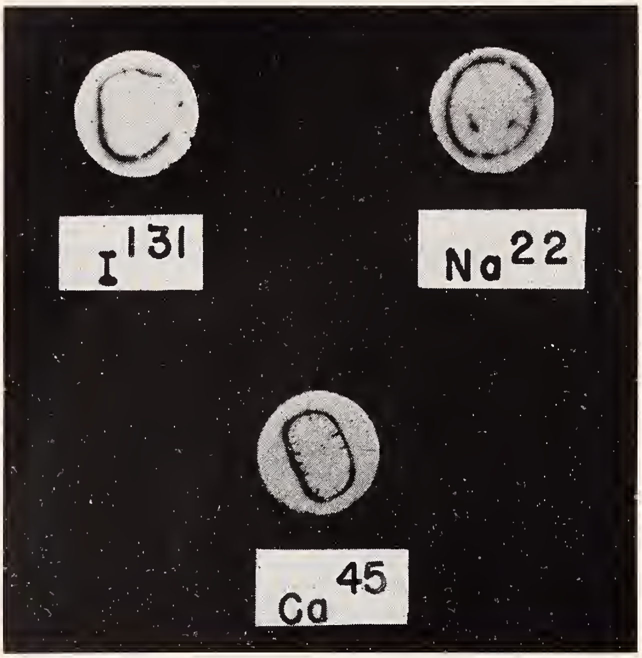

Fig. 4. Radioautographs showing migration of radioisotopes into the enamel of human teeth.

The radioautographs in Figure 4 were prepared from human teeth whose crowns had been immersed in the solutions of the indicated radioisotopes. The migration of these ions into the enamel is evident. These and other studies of the centripetal migration of ions which are part of the enamel mineral indicate that exchange of ions between the enamel and saliva is a normal physiological process.

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BATTLE AGAINST TUBERCULOSIS STILL CONTINUES

One fact which must be acknowledged and is of practical importance is that over the country the annual number of newly reported cases of tuberculosis has declined very little. In fact, the number increased from 1940 until 1948 and, while it has gone down for the past four years, in 1952 it was still slightly higher than in 1940, although the case rate has gone down since it is affected also by the increasing population.

Even though many of these patients do not require hospitalization, all should be investigated, their clinical status determined, their familial contacts studied and other appropriate control measures taken where indicated.

Thus, there has been no decrease in the effort required of health departments and voluntary agencies, and no reduction in personnel or funds should be contemplated. In fact, greater effort is demanded if tuberculosis is to be eradicated, and no lesser goal should satisfy us.—Philip E. Sartwell, M.D., *Nat. Tuberc. A. Tr.,* May, 1954.

Influence of Anesthesia on Maternal Mortality

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THE majority of pregnant women who come to delivery are young, healthy, vigorous patients. Most of them have been watched carefully throughout their prenatal course. A good history and physical examination have been taken, which will aid the person giving the anesthesia to give that type of anesthetic which will be the easiest for the mother and the baby and yet will be the safest. Therefore, the calculated risk of anesthesia for this type of patient should be extremely low. In like manner, most deliveries are relatively simple, requiring but little obstetric manipulation, and therefore the amount of anesthesia required and the relaxation necessary should be minimal. And yet, one is confronted with the fact that anesthesia deaths contribute substantially to over-all maternal mortality.

The position that obstetric anesthesia holds, both in the field of obstetrics and in that of anesthesia, is frequently inferior. In many instances, the administration of the anesthetic is relegated by those who are in charge to the most inexperienced. The opinion seems to be, in a great many instances, that anyone—nurse, intern, resident, practicing physician, anyone that is handy—can competently give an obstetric anesthetic. As long as this type of thinking remains prevalent, the incidence of maternal deaths due to anesthesia will remain high.

In a review of the literature, of 4,894 maternal deaths, 182 (3.7 per cent) were caused by anesthesia.[1-26] This series represents a cross section of the United States. Hingson and Hellman[13] have reported a much larger series, gathered from the literature of both the United States and Europe, with a total of 330 anesthetic maternal deaths. The present report does not include theirs, although I am sure that many of the cases in this report are included in their statistics. Unfortunately, most of the published articles do not break down the anesthetic deaths as to the method of anesthesia, whether it was spinal, inhalation, caudal, local or intravenous. Therefore, it is difficult to ascertain which of the methods used was the most lethal. If only the reports in which the method of anesthesia was stated are considered there were 1,878 deaths, seventy-one of which were due to anesthesia. Of these, twenty-five were due to spinal anesthesia, thirty-nine to inhalation anesthesia, and seven to caudal anesthesia. The maternal mortality rates of Minnesota for 1941[9], 1950[17] and 1951[24] have been published. There were 237 maternal deaths reported in these three years. Of these, ten were due to anesthesia. This gives a proportion of 4.2 per cent, which is higher than that given for the total cross section of the country.

All methods of anesthesia have been used for obstetrics: inhalation anesthesia, conduction anesthesia and, in a small number of cases, intravenous anesthesia. All have been the cause for some deaths. Unfortunately, the vast majority of these deaths were preventable.

Inhalation Anesthesia

Various agents have been used in inhalation anesthesia, including nitrous oxide, ethylene, ether, cyclopropane, trichloroethylene and chloroform. All have their advantages as well as their disadvantages. The agent used should be the one with which the person administering the anesthetic is most familiar. Whenever inhalation anesthesia is used, there is always the problem of vomiting under anesthesia and aspiration of the vomitus. This has caused more deaths than any other factor. The solution of the problem appears to be to avoid giving this type of anesthetic if the mother has eaten within five to six hours before she comes to the delivery room. There appears to be no good reason why these women cannot be instructed by the obstetrician

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From the Section of Anesthesiology and Intravenous Therapy, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

to stop taking food and drink when they feel they are going into labor. The reason given—that these gravid patients must eat to "keep up their strength"—is nonsensical and dangerous. Once an obstetric patient is in the hospital and is in active labor, there is never a good reason to feed her. Intravenously administered fluids can not only provide adequate fluid balance but also maintain the necessary caloric intake. Those patients who have come to delivery with a full stomach should under no circumstances receive an inhalation anesthetic. Other methods of anesthesia are available. Any of the conduction anesthetics can be used with safety if they are properly administered.

In regard to the administration of inhalation anesthetics, not everyone can give an inhalation anesthetic, not everyone can competently work a gas machine and even not everyone can "drop" ether competently. Only those persons who have been trained should be allowed to give an anesthetic. There is no argument who that person should be, whether nurse or physician, but at least he or she should have had training in the giving of the anesthetic. Often the inexperienced person will unwittingly allow the obstetric patient to become too deeply anesthetized, causing cardiac and respiratory failure. Frequently these inexperienced people will allow anoxia to be present and unrecognized, causing perhaps not death but severe brain damage not only to the mother, but also to the baby. Generally the inexperienced will be unable to cope with the patient who has vomited while under the influence of an anesthetic.

Conduction Anesthesia

Conduction anesthesia can be safe if it is properly managed. Among the forms of conduction anesthesia that are available to the obstetric patient are spinal, caudal and pudendal block. Spinal anesthesia is reported in the literature to be the second highest cause of maternal deaths due to anesthesia. Almost without exception, the death was due to too massive a dose of the spinal anesthetic agent.

For vaginal deliveries a minimal dose should always be given. Two and one-half milligrams of heavy solution of dibucaine hydrochloride (nupercaine hydrochloride), 2.5 to 3.5 mg. of weighted tetracaine hydrochloride (pontocaine hydrochloride), 30 to 35 mg. of piperocaine hydrochloride (metycaine hydrochloride) or 40 to 50 mg. of procaine hydrochloride will give adequate anesthesia for vaginal delivery. If larger doses are given, then only complications can be expected.

For cesarean section minimal doses again are an absolute necessity. My colleagues and I use only procaine or metycaine; 60 to 70 mg. of unweighted metycaine or 75 to 85 mg. of procaine will give adequate anesthesia for cesarean section. If a larger dose is used, the anesthesia will extend abnormally high, causing a drop in blood pressure and a sensory level that will indicate that the patient may have respiratory embarrassment due to depression and paralysis of the respiratory muscles.

The technique, as worked out by Adriani and his co-workers,[27,28] by which saddle anesthesia should be performed using a dextrose-weighted anesthetic agent, is a safe and proved method for the administration of this type of conduction anesthesia.

Caudal anesthesia, the use of which is largely due to Hingson and Edwards,[29,30] has proved also to be a safe method of conduction anesthesia, if the technique for the insertion of the needle and administration of the anesthetic agent is properly followed. There are certain inherent dangers and difficulties with caudal anesthesia. The greatest difficulty is the insertion of the caudal needle, which takes a great deal of practice. Caudal anesthesia should not be used unless the person administering it has received instruction from someone who has had experience in this method. The caudal technique can either be continuous, which means the giving of intermittent doses of the anesthetic agent as is necessary, or as a single dose for the expected short delivery. The danger of caudal anesthesia is the inadvertent placement of the caudal needle in the subarachnoid space, which results in giving the patient a massive spinal anesthetic. The height of the caudal anesthesia is so regulated that the level of anesthesia must extend at least to the sensory level of T10, the umbilicus, to insure that the patient will have pain-free contractions. If it extends higher than T6, uterine contractions will be completely stopped. For caudal anesthesia my colleagues and I have used 1.5 per cent solution of metycaine with good results. When the caudal needle is introduced, the patient is given a test dose of 5 to 8 cc. of the metycaine solution and a time interval of at least five minutes is allowed to elapse in order to insure that the patient has not inadvertently received a spinal anesthetic. After this time lapse, the patient is given an additional dose of 20 to 30 cc., which, in the majority of cases, will produce adequate anesthesia with a sensory level between T10 and T11. The caudal anesthesia is then supplemented at regular intervals when the patient experiences pain. An additional dose of 10 to 15 cc. will, in most cases, give adequate relief.

Pudendal block is by far the safest of conduction anesthetic methods and is probably the safest of any of the anesthetic methods for a vaginal delivery. The pudendal nerve is blocked at Alcock's canal, which lies to the medial surface of the ischium in the region between the ischial spine and the tuberosity. This block, which is done bilaterally, plus infiltration of the labia majora up to the mons pubis, will give anesthesia to the lower part of the birth canal.[31] Although this block does not produce the marked relaxation that is found with saddle or low spinal and caudal anesthesia, it does have the advantage that, with a little practice, uncomplicated vaginal deliveries can be done with ease and the episiotomy and the repair can be done without pain to the patient. The most frequent complication of this block is inadvertent introduction of the needle into the rectum. This can be prevented by inserting the index finger of the opposite hand into the rectum and guiding the needle along the ischial tuberosity in the direction of the ischial spine. The second most frequently encountered complication is the intravascular injection of the anesthetic agent. Frequent aspirations on the barrel of the syringe will show whether or not the needle is in a blood vessel.

Cesarean Section

In our institution, the vast majority of cesarean sections are done with the patient under local anesthesia. Once the baby has been delivered, the patient is then given a general anesthetic in the form of intravenous pentothal sodium for the remainder of the procedure. The abdominal wall is thoroughly blocked, using a 0.5 per cent solution of metycaine. This means that the skin and the subcutaneous tissue down to the fascia are thoroughly infiltrated with the anesthetic solution. Most of the pain from a cesarean section appears to come from traction at the lower end of the incision. Therefore, we put a rather large amount of anesthetic agent into the suprapubic space of Retzius. Once the baby has been delivered and the cord has been clamped, the patient is given pentothal sodium for anesthesia during closure of the uterus, peritoneum and abdominal wall. My colleagues and I feel that it is absolutely necessary to instruct the patient that this is the safest possible way for the procedure to be done and, in most instances, the patient is more than willing to go along with our desires.

Prevention of Anesthetic Deaths

What are the things one can do in an attempt to eliminate the anesthetic factor in maternal mortality?

1. Personnel should be trained to administer the anesthetic. In surgery this fact is well recognized and accepted without question. Here one insists that the anesthesiologist or the nurse anesthetist administering the anesthetic must be trained to give adequate relaxation, that he must be able to cope with any emergency that arises during the course of the anesthesia and that he make sure there are no episodes of anoxia. It is just as important in obstetrics as in surgery that this type of person should be at the head of the table administering the anesthetic. Even if there is a shortage of personnel in the hospital, it is still reasonable that one or two people should be adequately trained to administer anesthesia properly.

2. It behooves the obstetrician to instruct his patients that when they are in labor they should not take food nor drink.[32] In this way inhalation anesthetics can be administered with safety. The old idea of trying to wash out the stomach by introducing a lavage tube is of no avail. The only possible good it can do is to induce vomiting by the patient and in this way rid the stomach of its contents.[33] In the hospital, as has been pointed out, there is never a good reason for feeding a patient in labor.

3. It must be remembered that the anesthetic death rate does not tell the entire story. It does not tell how many mothers were near death, nor does it tell how many accidents which were not fatal could have been prevented if good anesthetic management had been instituted. Unfortunately, these cases are never reported and only the fatal cases are drawn to our attention. These

cases of near death and of preventable accidents are just as important and should teach just as good a lesson as the fatal cases. If the obstetrician is convinced that inhalation anesthesia is the type of anesthesia that gives him the best results, then he must instruct his patients of the dangers of a recently eaten meal. On the other hand, if he feels that conduction anesthesia in any of its forms is the anesthetic of choice, then it behooves him to inform his patients of this, so that they will expect to receive conduction anesthesia. If the obstetrician feels that pudendal block is the safest method, and this fact is explained to the patient, making her fully cognizant that she will have some pain, the vast majority will be more than willing to acquiesce in the obstetrician's desire for a local anesthetic. These women are in a good position to bargain with. They realize that not only their own safety is at stake, but also that of the unborn child. Most of them will go to any extent to make sure that nothing unforeseen happens.

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Chronic Hypervitaminosis A

Report of a Probable Case in a Man

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VITAMIN A intoxication is probably a more common disease in the United States than its famous counterpart, vitamin A deficiency.[1,2] In Caffey's opinion routine administration of vitamin A to healthy, well-nourished infants and children constitutes a greater danger to their well-being than its omission.[3,4] In contrast to the wide dissemination of knowedge concerning vitamin A deficiency, the fact that vitamin A has definite toxic properties is frequently not appreciated. Modern texts on medicine and related sciences still emphasize hypovitaminosis A. The sections devoted to the opposite nutritional extreme, vitamin A intoxication, frequently fail to do justice to the importance of this disease in the everyday practice of medicine in this country;[5] some widely used treatises omit hypervitaminosis A entirely.[6] The widespread use of vitamin A preparations as supplements in infant feeding, which are often continued to school age, the massive doses which are on occasion employed for the treatment of some common skin disorders, (5f, 5g, 6c) "therapeutic trials" in conditions for which no specific remedy is known,[7,8] advertising claims and over-the-counter sales make chronic hypervitaminosis A a possible concern to the diagnostician. The true importance of the disease will remain unrecognized until its manifestations become more widely known.

Eskimos have realized, apparently since time immemorial, that the livers of polar bears, bearded seals, and arctic foxes are unfit for consumption by humans or dogs.[9] White men learned about these facts through the bitter experience of polar explorers. There is an account of acute vitamin A intoxication, which occurred on Novaya Zemlya on May 31, 1597: In an effort to find a North East Passage to India the citizens of Amsterdam had equipped an expedition under the command of William Barntzoon, "a most skillful mariner and burgher of Amsterdam." The expedition had reached Novaya Zemlya in August, 1596. There the members "were forced, in great cold, pouerty, misery and griefe, to stay all that winter" (*sic*) as reported by their historian, Gerrit de Veer. By spring the food situation had become desperate and the men took the liver of a bear which they had killed "and drest and eate it; the taste like us well, but it made us all sicke, specially three that were exceeding sicke, and we verily thought that we should have lost them all, for all their skins came of from the foote to the head; but yet they recouered againe, for the which we gave God heartie thankes, for if as then we had lost these three men, it was a hundred to one that we should neuer have gotten from thence, because we should haue had too few men to draw and lift at our neede" (*sic*).[10] Subsequent generations of arctic explorers have attested to the accuracy of Gerrit de Veer's description of polar bear liver poisoning.[11,12] Rodahl and Moore attribute the toxicity of polar bear and bearded seal livers to their high vitamin A content. Ingestion of one pound of these livers provides about 10,000,000 units of vitamin A; administration of 40,000 units vitamin A to a 250 gram rat is definitely poisonous.[13,14]

Polar bear liver poisoning is the prototype of the acute vitamin A intoxication which has been described following accidental ingestion of single large doses of vitamin A preparations or of large quantities of foods which have a high vitamin A content.[2] The abrupt onset of the illness, the gastrointestinal disturbances which accompany it, and the history greatly facilitate the diagnosis. In contrast, the manifestations of chronic vitamin A intoxication begin insidiously, gastrointestinal disturbances are absent, and patients frequently fail to volunteer information regarding their vitamin A intake unless they are questioned specifically. Symptoms referable to the central nervous system (headache, irritability, lethargy) and lesions of the integument are common to both acute and chronic hypervitaminosis A.

The author wishes to thank Dr. C. J. Watson and Dr. B. J. Kennedy for the opportunity to study this case.

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Although carotenoids are precursors of vitamin A in the human body, they will not cause any untoward reactions even if large amounts are ingested. Only vitamin A itself is toxic. Its precursors are apparently harmless. They are not converted into vitamin A in sufficient amounts to cause hypervitaminosis. To the contrary, carotenemia is likely to be associated with diseases which tend to cause endogenous vitamin A deficiency. It occurs in diabetes mellitus, hypothyroidism, Simmond's cachexia, male castrates, chronic glomerulonephritis, acute hepatic disease, and malnutrition. Lipemia, thyroid, and androgen deficiency seem to be involved in its pathogenesis. Carotenemia is characterized by a lemon to orange-yellow discoloration of the skin involving chiefly areas which have heavy stratum corneum (palms, soles, forehead, nose); the sclerae and mucous membranes remain unaffected. The discoloration is due to an excessive accumulation of cartenoids in the body and associated with a high plasma carotene level; the plasma vitamin A level remains normal. Skin pigmentation does not develop in healthy individuals even after ingestion of large amounts of carotene, and it is the only outward manifestation of carotinemia in those who are susceptible. The discoloration will disappear when the underlying disease abates.[15,16]

Chronic hypervitaminosis A, as it is seen in the United States today, was described and recognized in experimental animals thirty years ago: Takahashi, Nakamiya, Kawakami, and Kitasato fed excessive amounts of a crude vitamin A concentrate to mice and rats and found that their animals suffered loss of hair, emaciation, paralyses, and ultimately death; at autopsy fatty degeneration was found in the liver, heart, and kidneys, intestines and lungs were hyperemic and on occasion contained hemorrhages.[17] The possibility that these lesions might have been due to substances other than the vitamin A in their crude "Biosterin" was entertained for many years. But it is now established that vitamin A can produce lesions of the integument, gastrointestinal tract, and skeleton, as the Japanese workers had contended. These lesions are not due to fatty acids, vitamin D or other substances. They are not produced by fish liver oils in which vitamin A has been inactivated, but they can be evoked by administration of synthetic vitamin A acetate.[18,19,20,21] Rats and mice are not the only species susceptible to hypervitaminosis A; the disease has also been produced in guinea pigs, dogs, ducks, and chicks.[9,21] A hemorrhagic tendency, similar to that seen in scurvy, is prominent in many species. The adrenal cortex shows changes akin to those caused by vitamin C deficiency, especially in the guinea pig. But administration of vitamin C neither prevents nor counteracts hypervitaminosis A. Hypoprothrombinemia has been found in rats with vitamin A intoxication; however, it is not a constant feature of the disease and it does not respond regularly to vitamin K administration.[23,24]

Chronic hypervitaminosis A may produce skeletal lesions which are of diagnostic importance.[3,4,9,25] X-ray examination may reveal hyperostoses, mottling of the bones, thinning of the cortices, and decalcification. The roentgenograms may resemble those seen in infantile cortical hyperostosis, syphilis, low-grade infections, or healing fractures. In experimental animals excess vitamin A causes accelerated maturation and degeneration of epiphyseal cartilage, excessive remodeling of bone, increased osteoclastic activity leading to loss of cortical substance and multiple fractures; the fractures heal readily by voluminous callous formation. Hypervitaminosis A, like other metabolic disturbances, produces skeletal abnormalities most readily in young and growing individuals.

Iatrogenic hypervitaminosis A antedates the discovery of the vitamin.[16,17] Czerny, in 1912, reported development of seborrheic dermatitis in children with tuberculosis following administration of large doses of cod liver oil.[28] Nevertheless, Lehman and Rapaport, as late as 1940, maintained that vitamin A may be administered to children in doses of 300,000 units per day without any ill effects.[29] The first well-documented case of chronic hypervitaminosis A was reported by Josephs in 1944.[30] The patient was a three-year-old boy who had received daily 240,000 units of vitamin A in the form of halibut liver oil for almost his entire life. His height was normal, but he was underweight and anorexic. He had no eyebrows, and his hair was coarse, dry and brittle. Roentgenograms revealed thinning of the cortices on his phalanges and metacarpal bones; the proximal epiphyses of his humeri and tibiae were mottled, and skeletal development was advanced. When vitamin A medication was withheld, the patient showed rapid improvement; normal hair growth resumed after two months, his appetite became normal, and he gained weight.

In the past ten years over twenty cases of chronic hypervitaminosis A have been reported in the pediatric literature in the United States.[2,3,4,30,31,32,33,34] The children were one to four years of age and had ingested at least 75,000 units vitamin A per day for a minimum of six months. The most conspicuous feature of the disease were bony, tender, subcutaneous tumors. Pain in the lower extremities prevented some of the patients from walking. Alopecia, rhagades, dry skin, hepatomegaly, and jaundice were common. The clinical picture suggested vitamin deficiency rather than vitamin intoxication, but the disease responded dramatically to the withdrawal of vitamin A medication. Bone pain subsided within days although the plasma vitamin A level required weeks to return to normal; the hyperostoses regressed only over a period of months. No permanent sequelae have been reported.

The relatively large doses of vitamin A required to cause hyperostoses in children may lead one to believe that vitamin A is rather innocuous after all, but hyperostoses develop quite late in the disease. They are undoubtedly preceded by months of malaise which may be difficult to evaluate in a young patient. A substance purported to be as beneficial as vitamin A is not likely to be thought of as the cause of a child's vague complaints.

Although the onset of chronic vitamin A intoxication should be easier to recognize in the more articulate older patient, the first well-documented case of chronic hypervitaminosis A in an adult was reported only three years ago.[35] Two more cases have been recorded since.[36,37] All of them occurred in women who had taken 100,000 to 600,000 units vitamin A daily. Initial symptoms appeared after about a year and included insomnia, irritability, malaise, headache, nausea, anorexia, weight loss, depression, diplopia, blurred vision, dysuria, urinary incontinence, arthritis, dysmenorrhea and hypomenorrhea. Loss of hair, including eye brows, eye lashes, pubic and axillary hair, desquamation of the skin and chloasma developed later. Exophthalmos, papilledema, nystagmus, unequal reflexes, positive toe signs and abnormal electroencephalograms have led physicians to mistake chronic vitamin A intoxication for brain tumor, meningitis and encephalitis. One of the patients had been on 500,000 units vitamin A per day for eight years; during this time she had undergone a craniotomy and developed some skeletal deformities and muscular atrophy. When chronic hypervitaminosis A was finally suspected and the fasting plasma vitamin A concentration determined it was found to be 2,000 microgram per cent.*

Despite occasional statements to the contrary, especially in the older literature, there can now be little doubt that excessive intake of vitamin A is toxic to man. Assessment of the optimal nutritional status of man with respect to vitamin A is beset with difficulties. The desirable vitamin A intake for an adult is variously estimated to lie between 2,000 and 7,000 units per day. In the United States, 5,000 units are recommended for normal adults, and it is believed that this dose should be increased to 6,000 units during pregnancy, and to 8,000 units during lactation; the recommended doses for children are 1,500 units to age one year, 2,000 units from age two to three years, 2,500 units at age four to six years, and 3,500 units at age seven to nine years.[38] One of the most careful studies concerning the vitamin A requirements of man was carried out in England during World War II on young adults.[39] One thousand three hundred units vitamin A per day were sufficient to maintain these individuals free of any signs of deficiency, and 2,500 units constituted a safe margin. Only three of eleven subjects who were placed on a vitamin A deficient diet developed night blindness. It began after they had been on the diet for seventeen to twenty-two months, and it was cured by administration of 1,300 units vitamin A per day; no massive doses of vitamin A were required.

Carotenoids are less effective in the prevention of hypovitaminosis A than vitamin A itself and their effectiveness depends in large measure on the form in which they are administered. Ingested as solutions in oil they seem to be quite readily utilized, but in form of carrots they are rather inactive. Diseases of the liver and possibly diseases of the intestine interfere with the conversion of carotenoids to vitamin A. Where absorption of fat is impeded, for instance in sprue and celiac disease, absorption of vitamin A and carotenoids is likely to be faulty, also. Patients with diseases of this type may require an increased intake of vitamin A to forestall deficiency.

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*1 microgram vitamin A corresponds to 3.3 international or USP units vitamin A. The normal range of fasting serum or plasma vitamin A concentration is in the order of 20 to 60 microgram per cent.

Understanding of the processes involved in the absorption and utilization of vitamin A and its precursors is still only fragmentary. Until more pertinent information along these lines becomes available, it will be impossible to determine the optimal nutritional status of man with respect to vitamin A with certainty. As far as the normal individual is concerned, there has been a tendency to overestimate the vitamin A requirements and to underestimate the dangers inherent in overdosage. The margin of safety between preventive and toxic doses may be appreciably smaller than is generally believed. Gerber, Raab, and Sobel[37] have observed symptoms indicative of chronic hypervitaminosis A in an adult who took as little as 50,000 units vitamin A per day for a period of eight months.

At the present time, the diagnosis of both vitamin A deficiency and intoxication must be made essentially on clinical grounds. Biophotometric measurements and determinations of the serum vitamin A concentration are of value only when they are interpreted in conjunction with the history and clinical findings. The cardinal manifestations of hypovitaminosis A are seen in the eye and the integument. They consist of follicular keratosis, nyctalopia, xerophthalmia, Bitot's spots and keratomalacia. The diagnosis should be made only if a reliable history of low vitamin A intake can be obtained, or if the patient suffers from a gastrointestinal, metabolic, or hepatic disease. The serum vitamin A level may be within normal limits if the patient has ingested vitamin A containing foods in the recent past.[41] On the other hand, a low fasting serum vitamin A level is not in itself diagnostic of vitamin A deficiency. The disease should respond to a normal vitamin A intake. Chronic hypervitaminosis A manifests itself chiefly by signs and symptoms referable to the central nervous system, the integument, and the skeleton. They may include headache, malaise, insomnia, irritability, nervousness, increased intracranial pressure, anorexia, weight loss, loss of hair, coarse hair, desquamation of the skin, hyperpigmentation of the skin, pruritus, joint and bone pain, skeletal deformities, hyperostoses, and decalcification. Roentgenograms are valuable adjuncts in the diagnosis when they do reveal skeletal abnormalities. A reliable history of high vitamin A intake is of paramount importance. The vitamin A level should be elevated before excess vitamin A medication is withdrawn, but it may return to normal before the last vestiges of the disease have disappeared. The patient should recover when given a normal diet without vitamin A supplement.

Probable Case in a Man

A fifty-four-year-old white interior decorator and real estate broker was admitted to the University of Minnesota Hospitals in May, 1954, with the chief complaint of pain in his left leg.

He had been in good health until nineteen months prior to his admission when he began to experience periodic episodes of indigestion. These episodes occurred usually in the afternoon, were not related to meals, and were relieved by belladonna, sodium bicarbonate, "cokes", or plain water. Seven months prior to his admission he consulted a physician for his symptoms of gastric distress. Physical and neurological examinations, roentgen studies of his gastrointestinal tract, and laboratory tests failed to reveal any abnormalities. The patient weighed 136½ pounds and there had been no weight loss. Symptomatic treatment resulted in some improvement.

During subsequent months the patient became mildly depressed. Two months before his admission he consulted the physician again because of severe neuritic pain in his left hip and leg. The pattern of pain corresponded to the distribution of the femoral nerve. The sedimentation rate was normal. Treatment with salicylates was instituted. A few days later weakness, headache, anorexia, and a low-grade fever developed. Physical examination was again negative, except for a decreased patellar reflex on the right; this abnormality persisted for only a few days. Roentgenographic examination of the chest and pelvis failed to reveal any disease process. The white cell count was 16,300 per cubic millimeter, with 22 per cent neutrophiles, 68 per cent lymphocytes, 5 per cent eosinophiles, and 5 per cent monocytes. The cephalin flocculation test was 3+. Other laboratory determinations, including urinalysis and bromsulfalein excretion, were within normal limits. Within one week the patient seemed to have largely recovered from his acute illness, except for some residual pain in his left thigh and a general feeling of malaise.

The patient was admitted to the University of Minnesota Hospitals (two months after the onset of his acute illness) for further diagnostic study. He related that he had had an attack of "arthritis" in his left hip "many years" previously and a moderately severe alcohol intake until about seven years prior to his admission. No history of exposure to heavy metals could be elicited. The patient thought that he might have had some undue exposure to solvent vapors in the months immediately preceding his acute illness. Questioned specifically concerning his vitamin A intake, the patient stated that he had had a mild eczema on his leg three years prior to admission. At that time his physician prescribed vitamin A by mouth in doses of 50,000 units per day. The eczema seemed to respond to this treatment, although it recurred on occasions on either leg, especially during the winter months. The patient had continued to take vitamin A daily in doses of 50,000 units until the onset of his acute illness two months prior to admission when he stopped the medication on his own volition.

Physical examination revealed a well-developed, thin male in no acute distress. His temperature was 98.0°, pulse 72 per minute, respiration 18 per minute, blood pressure 120/70, weight 127 pounds. The tongue showed a mild tremor. The liver was palpable two fingerbreadths below the right costal margin. A small inguinal hernia was present on the right. The prostate was moderately enlarged (2+), symmetrical, non-tender, and of normal consistency. The left patellar reflex was absent; there was some weakness and atrophy of the quadriceps muscle and weakness of the tibialis anterior on the same side. Both ankle reflexes were diminished. No other abnormalities were found on physical or neurological examination.

A roentgenogram of the chest, an electrocardiogram, and urinalysis were within normal limits. The sedimentation velocity was 2, 6, and 9 mm. at 20, 40, and 60 minutes, respectively. The hemoglobin concentration was 15.2 gram per cent. The white cell count was 17,200 per cubic millimeter with 59 per cent neutrophiles, 28 per cent lymphocytes, 9 per cent monocytes, and 4 per cent eosinophiles. The stool gave a 4+ guaiac test on two occasions (on a diet containing meat). The bromsulfalein retention was 1 per cent, serum bilirubin 0.1 milligram per cent at 1 minute and 0.3 milligram per cent total. Cephalin flocculation was negative, thymol turbidity 3 units. The total lipids were 29 units. The prothrombin time was 13.2 seconds (control 13.1 seconds). The blood glucose concentration was 95 milligram per cent. The Mantoux test was positive at a dilution of 1:1000. The Kline test was negative. The erythrocyte protoporphyrin was 65 microgram per cent, the erythrocyte coproporphyrin 0.5 microgram per cent, the hematocrit 48 per cent. Urinary coproporphyrin excretion was 224 microgram in twenty-four hours. The Addis count (on a twelve-hour urine specimen consisting of 470 milliliters) was: casts 13,050, erythrocytes 1,720,000, leukocytes 11,750,000, negative for protein. A fasting serum vitamin A level was 69 microgram per cent with a carotene level of 228 microgram per cent (normal values for our laboratory are 20 to 40 microgram per cent for vitamin A and 120 to 200 microgram per cent for carotene).

The patient was placed on a regular diet. Since his discharge seven months ago he has continued to improve gradually without any further treatments.

Except for periodic outbreaks of eczema on his lower legs this fifty-four-year-old man was apparently in good health until eighteen months after he had begun to take 50,000 units of vitamin A daily. At that time ill-defined gastrointestinal symptoms developed which responded partly to non-specific measures. He became moderately depressed and finally contracted neuritic pain in his left hip and thigh and a mildly febrile illness with leukocytosis, eosinophilia, and a normal sedimentation rate. The outstanding findings on physical examination were hepatomegaly, an absent patellar reflex on the left, and atrophy of the quadriceps muscle. His hemoglobin concentration was normal, but there was some bleeding from the intestines. When vitamin A was discontinued he improved rapidly for one week and more slowly in subsequent months.

The essential signs and symptoms which this patient manifested have all been recorded in similar combinations in hypervitaminosis A. All three adults with chronic hypervitaminosis A described in the literature thus far had anorexia, weight loss, headache, malaise, depression, arthropathies, and neuropathies.[35,36,37] Gastrointestinal disturbances are prominent after ingestion of excessive amounts of vitamin A. Hepatomegaly is common in hypervitaminosis A in children; it has also been described in the adult.[37] Various alterations in liver function tests, usually minor and inconsistent, and fatty metamorphosis of the liver have been seen in this disease.[36,37] Hemorrhagic diatheses[36] and intestinal bleeding[17] may be features of this illness, as may leukocytosis,[35] eosinophilia,[36] febrile episodes,[37] and normal sedimentation rates.[37] Following withdrawal of vitamin A medication and on a normal diet, most of the patients reported on showed a brief period of rapid improvement, followed by a more gradual decline of signs and symptoms. Loss of hair, which was a conspicuous sign in the three other adults, was not prominent in our patient.

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Psychiatric Out-patient Treatment

A Questionnaire Survey

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IN A previous paper published in MINNESOTA MEDICINE, a report was made of a questionnaire survey of 342 patients who had received out-patient psychiatric treatment in one of the state-supported mental health centers.[1] Since this report was published, 500 additional patients have been referred to the Southern Minnesota Mental Health Center* for diagnosis and/or treatment.

As stated in the previous article, this state-supported out-patient center has a three-fold purpose: (1) to provide follow-up care for patients provisionally discharged from state mental institutions, (2) to provide treatment for psychiatrically indigent residents of Minnesota upon referral by a licensed medical doctor, and (3) to act as educational centers in applications of mental health.

The staff consists of a part-time psychiatrist serving as medical director, a clinical psychologist, a social worker and a receptionist-stenographer. Treatment consists primarily of eclectic psychotherapy in which reassurance, interpretation and re-education play a major role. The clinical team participates in treatment under the direction of the psychiatrist.

As a result of improved statistical accounting procedures of the Department of Public Welfare, valuable data was collected on the 234 new patients accepted for treatment during the 1953-54 fiscal year. In the opinion of the staff, an opportune time existed for an evaluation of the reactions of these new patients to treatment received. It was felt that patient attitudes, plus the statistical data on such variables as their age and sex, number of therapeutic contacts, psychiatric diagnoses, prior treatment and source of referral might provide a more complete appraisal of the services rendered by the Albert Lea installation.

Statistical Characteristics of the Patient Population

The population included in this study are the 234 new patients referred for services from the period July 1, 1953 to June 30, 1954.

Age.—The 234 patients ranged in age from three years to eighty-three years. The median chronological age was 18.9 years.

Sex.—Fifty and nine-tenths per cent of new admissions were males while 49.1 per cent were females. In the child population (eighteen years and under) about twice as many boys as girls were referred. Among the adults, the proportions were reversed.

Source of Referral.—Table I contains the primary sources of referral, i.e., the persons or agencies who originally suggested that the patient seek psychiatric help.

TABLE I

Referral Source	No. of Children (Under 18)	No. of Adults (Over 18)	Percentage of Total Case-Load
State hospital staffs	0	10	4.3%
Self, family, friends, relatives or employer	7	5	5.2%
Public welfare agencies	25	3	11.9%
Private welfare agencies	1	0	.5%
Licensed physicians	46	114	68.3%
Courts	1	0	.5%
Schools	16	0	6.9%
Ministers	1	1	.7%
Others	1	3	1.7%
Total	98	136	100. %

With the exception of patients referred directly by licensed physicians, all patients were required to obtain a medical examination prior to admittance to the center.

Prior Treatment.—Of the 234 new patients, 81.6% had received no prior treatment or psychiatric consultation of any sort. A total of 7.7%

From the Minnesota Department of Public Welfare, Albert Lea, Minnesota.

*Formerly the Community Mental Health Center.

of the population had been hospitalized in a state mental hospital prior to admission. Only 6.5%, or fourteen patients, had received prior psychiatric treatment on a private basis. Six of the fourteen were referred by the private psychiatrist because of the inability of the patient to pay for private care. The remaining 4.2% had received prior help from such sources as traveling psychologists from the Department of Public Welfare, the University Hospital and VA out-patient clinics.

Number of Therapy Contacts Per Patient.—An average of 8.3 treatment contacts were recorded for each adult patient and an average of 4.4 treatment contacts per child patient were made.

Psychiatric Diagnoses of Patients Seen.—The largest percentage of patients were diagnosed as suffering from some form of psychoneurosis (35. 3 per cent). About 5 per cent of the cases were diagnosed as having a psychosis upon admission. Learning difficulty and/or mental deficiency were the diagnoses applied to 17.4 per cent of the cases. Transient situational personality disorders comprised 11.9 per cent of the cases. Behavior disorders and character disorders comprised 7.8 per cent of the case load. The remaining 22.6 per cent were distributed among trait disturbances, special symptom reactions, and chronic brain disorders.

Results of Questionnaire Survey

In order to evaluate the reactions of the new patients to treatment, an eight-item questionnaire was devised and mailed to the 234 patients included in this study. In the directions for completing the questionnaire, the patient was encouraged to indicate freely his personal reaction to the questions. The directions further emphasized that if the patient so desired, the questionnaire could be completed with his or her name omitted.

Of the 234 questionnaires mailed, fourteen were returned because of improper addresses, leaving a total of 220 questionnaires which were presumably received by the patients or guardians of patients. From the total of 220 mailed questionnaires, 112 returns were received by the time of our self-imposed cut off date, one month later. This yielded a 51.4 per cent return. Many questionnaires were returned blank with a note that their child was seen only for testing, not "treatment."

Not all the respondents answered each question. Each question is, therefore, treated separately in compiling the results.

Question I	No. of Responses	Percentage of Responses
I visited the Mental Health Center:	110	100.0
A. Only once	21	19.1
B. Less than 5 times	30	27.3
C. Between 5 and 10 times	28	25.4
D. Between 10 and 20 times	20	18.2
E. I am continuing to visit the Center	11	10.0

Question II	No. of Responses	Percentage of Responses
As a result of my visits to the Center, I feel:	97	100.0
A. Greatly improved	24	24.8
B. Much improved	49	50.5
C. Only slightly improved	14	14.4
D. That no improvement was made.	10	10.3
E. That my condition is worse	0	00.0

Question III	No. of Responses	Percentage of Responses
I feel that coming to the Center:	97	100.0
A. Has upset me greatly	0	0.0
B. Made me more uncomfortable	5	5.2
C. Reduced some of the fears I had previously	31	31.9
D. Helped me in solving some of my problems	47	48.5
E. Actually prevented further mental or nervous difficulties	14	14.4

Question IV	No. of Responses	Percentage of Responses
In terms of help received, I feel I was (or am):	100	100.0
A. Aided greatly	25	25.0
B. Helped considerably	54	54.0
C. Helped some, but wanted more aid	18	18.0
D. Wasting my time	1	1.0
E. Actually harmed by visits	2	2.0

Question V	No. of Responses	Percentage of Responses
If any of my friends or relatives needed psychiatric help, I would:	105	100.0
A. Highly recommend the Center in Albert Lea	68	64.8
B. Suggest the Center as a possible place where help could be received	26	24.7
C. Mention the Center to them	11	10.5
D. Tell them of the Center, but only as a last resort	0	0.0
E. Tell them to seek help elsewhere	0	0.0

Question VI	No. of Responses	Percentage of Responses
For the treatment received, I would:	101	100.0
A. Resent having to pay any fee	1	1.0
B. Would pay a minimum fee if required	40	39.6
C. Would pay a reasonable fee if required	53	52.5
D. Would feel better if I could pay for services received	6	5.9
E. Feel that all patients should pay for treatment	1	1.0

Question VII	No. of Responses	Percentage of Responses
In the future, if I felt I needed further psychiatric treatment, I would:	84	100.0
A. Certainly not come back to the Albert Lea Center	0	0.0
B. Maybe go back to the Center, but only as last resort	4	4.8
C. Go back to the Center, but prefer to have private care	4	4.8
D. Come back willingly	40	47.6
E. Be eager to return because of help received in the past	36	42.8

Question VIII	No. of Responses	Percentage of Responses
Since my last contact with the center:	78	100.0
A. I was hospitalized for my condition	3	3.8
B. I have sought further psychiatric help	4	5.2
C. I have obtained help from others such as doctors, ministers, welfare workers, etc.	14	17.9
D. I am troubled only occasionally by my condition	36	46.2
E. I am feeling well and healthy	21	26.9

The last two questions were to be answered only by patients who had no further scheduled appointments with any staff member of the Center.

Conclusions

The 234 patients referred to the Southern Minnesota Mental Health Center during a twelve-month period were statistically studied and found to have a median age of 18.9 years. The case load was equally divided between male and female patients. Eight of ten patients referred had never received prior psychiatric help. About 68 per cent of these patients were referred directly by physicians of the community. About eight treatment contacts were made with each adult patient, while about four and one-half treatment contacts were made with each child patient. As for psychiatric diagnoses, the largest percentage of the case load falls in the psychoneurotic category with only 5 per cent diagnosed as psychotic.

An eight-item questionnaire was devised and mailed to the 234 patients in an effort to determine their reaction to treatment. In the directions for the questionnaires, provisions were made for patient anonymity. Of the 220 questionnaires presumed to have been received, 112 or 51.4 per cent were returned by our self-imposed cut off date.

The following conclusions appear warranted:

1. 75 to 79 per cent of the respondents indicated their condition as much improved or greatly improved. An additional 14 to 18 per cent indicated slight improvement (Questions II and IV).

2. 31 per cent indicated that some of their fears have been reduced while 48 per cent felt they have received help in solving some of their problems (Question III).

3. 14 per cent of the respondents felt that further mental or nervous difficulties have actually been prevented (Question III).

4. 65 per cent would highly recommend the Center to friends or relatives, while none of the respondents indicated that they would disparage the Center (Question V).

5. As for payment of fees for treatment received, 92 per cent indicated that they would pay

(Continued on Page 650)

Hypertensive Encephalopathy versus Schizophrenia

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THIS case history is presented as an example of how an organic illness, hypertensive encephalopathy, was mistakenly diagnosed schizophrenia.

A man, aged fifty-six, was referred to the Anoka State Hospital on December 14, 1954, as a severely disturbed schizophrenic patient. For the four days before admission, he had been hospitalized elsewhere because of his destructive behavior. He required restraints and presented such a severe problem that the management insisted he be transferred to the state hospital.

On admission, the patient was ambulatory, did not appear acutely ill and gave the following history of the presenting illness. "I went to the neighbors about a week ago and they asked me to stay with them for a few days and I went to bed, awakened, and they told me my closest friend had just died. I was upset because he was my best friend, but I went completely to pieces when he walked in the door and returned from the dead. Then there was a lot of excitement and a lot of people were running in and out, and there were a lot of automobiles and strange lights."

He remembered being examined by a doctor and of going to a local hospital. After arriving there, he felt that others talked about him and threatened him. He went on to say, "They told me that if I married one of the nurses, my life would be spared. I refused and they shot at me and I ran away from them and they chased me through fields and over church steeples. They finally captured me and took me back and put me in a cage." He felt that he needed to come to Anoka State Hospital, as he had gone through so much that he was close to a nervous breakdown.

He gave a scrambled version of his past history with an involved story about different insurance examinations and various doctors who had treated him. Nevertheless, he had been in comparatively good health and able to do farm work with supplementary labor on the highway until a few months before the present illness. He had some discomfort in the back of his head and neck, pains in his chest, had trouble with his vision and some loss of memory. He had been taking medicine for several weeks for high blood pressure and kidney trouble. During the initial examination the patient's level of awareness fluctuated. At times, he seemed in good contact, talked coherently and evidenced good motor control. Then, again, he was confused, disorganized, incoherent and inco-ordinated.

He was seen by our consultant in internal medicine who noted the following: Blood pressure was 200/140. The heart was enlarged, with A-2 about three times as loud as P-2. There was a Grade III retinal arteriosclerosis with hard white spots (edema residues), with marked narrowing of lumen of arterioles to ⅓ or ¼ the lumen of the retinal veins. A slight choked disc and a few white spots which looked like cotton wool exudates were noted. The examination of the retina of this patient gave a clue to the probable cause of his symptoms. The arteriolar lumen of all of his retinal arteries was from ½ to ⅓ the normal diameter. Poiseuille's Law states that the quantity of fluid flowing through a vessel under a given head of pressure is proportional to the 4th power of the diameter. Therefore, if the diameter of a tube is reduced ½ only 1/16 of the previous volume flow is present. The retinal arterioles are part of the brain arterial system, and if the same condition of spasm would exist in the arterioles of the brain or even one part of the brain, the blood supply to this part of the brain would be enormously reduced. In consequence, there would be an inadequate supply of blood sugar, oxygen and other substances necessary for normal metabolism. Products of normal as well as abnormal metabolism would pile up, causing altered cerebral function. The exudates of the retina, as well as hemorrhages of the retina, would probably be reproduced in these inadequately nourished portions of the brain. The urine concentrated to a specific gravity of 1020, and there was some renal insufficiency, with blood-urea-nitrogen of 28 mg. per cent."

A diagnosis of hypertensive encephalopathy was made, and the patient was started on Serpasil orally. As his blood pressure decreased, he evidenced continuous improvement in his mental status. He remained cooperative with no disturbed behavior, and reassurance was given freely to help overcome the relatively mild confusion, delusions and illusions which presented at intervals the first two weeks of hospitalization.

After three weeks of hospitalization, the patient was discharged symptom-free, on a maintenance dose of Serpasil and a protein limit of 50 grams daily.

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Minneapolis Surgical Society

PREOPERATIVE USE OF A COMBINATION OF LEVO-DROMORAN TARTRATE AND A NEW NARCOTIC ANTAGONIST

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Fromherz and Pellmont[1] suggested, on the basis of experiments in animals, that the respiratory depression produced by Levo-Dromoran® Tartrate could be overcome by doses of a new narcotic antagonist, levallorphan tartrate,* which are smaller than those which inhibit analgesia. This leads to the interesting supposition that a ratio of these two morphinan derivatives might be found which, when given to man, would provide good analgesia without hazard of respiratory depression. The pharmacology of these drugs was investigated further by Benson et al.[2] Gross and Hamilton[3] were the first to study these agents in normal human volunteers. Utilizing the Wolff-Hardy-Goodell technique they found that, on simultaneous and subcutaneous administration, less levallorphan tartrate was required to prevent respiratory depression than was necessary to affect the analgesia produced by Levo-Dromoran Tartrate. Interestingly, when the drugs were given simultaneously by the intravenous route, there was respiratory depression in four subjects. Hamilton and Cullen were the first to report on the use of levallorphan tartrate in opiate-induced respiratory depression in man[4] and on its merits as a supplement to nitrous oxide-oxygen anesthesia.[5] Cullen and Santos[6] used a combination of the drugs (10 parts Levo-Dromoran Tartrate and 1 part levallorphan tartrate) in patients with chronic pain. This 10:1 ratio provided good analgesia without respiratory depression when given either subcutaneously or intravenously. In view of these findings, the present study was undertaken to determine the characteristics of the 10:1 mixture as a preoperative medication in comparison with morphine.

Procedure

The patients in this study were divided into two groups so that concurrent data with morphine could be obtained to serve as a standard for comparison. Ninety-eight patients received morphine sulfate subcutaneously as preoperative medication. Three hundred and sixty-four were given a premixed solution containing 5 mg. Levo-Dromoran Tartrate and 0.5 mg. levallorphan tartrate per cc. also subcutaneously.† For the sake of brevity, this mixture will be referred to in the text as "combined drugs" and the doses given expressed as mg. of Levo-Dromoran Tartrate. Special individual protocol forms were prepared so that all data relating to the pre and postoperative periods could be recorded.

The routine preoperative procedure was as follows: All adults, except those over sixty years of age or those for whom there was a specific contraindication, received 1½ grains of Nembutal® the evening before surgery. This dose was repeated in one hour if the patient was not resting comfortably. One and one-half grains of Seconal® were given 90 minutes preoperatively. Scopolamine (preserved with mannitol) or atropine and the narcotic were given subcutaneously forty-five minutes preoperatively. The dosage schedule for these drugs is shown in Table I.

TABLE I. PREOPERATIVE DOSAGE SCHEDULE

Age (Years)	Atropine (mg)	Scopolamine (mg)	Morphine (mg)	Combined Drugs (mg 1-Dromoran)
Under 1	0	0	0	0
1- 3	0.2	0	0	0
3- 6	0.3	0	0	1.0
6-10	0.3	0	*	1.5
10-12	0	0.2	8.0	2.0
12-60	0	0.3	10.0	2.5
Over 60	0.3	0	*	2.0

*Morphine given intravenously in operating room, p. r. n.

The dosage schedule of the combined drugs was arbitrarily chosen on the basis of our earlier experience with Levo-Dromoran Tartrate in the recovery room.[7] It will be noted that patients under ten or over sixty years of age did not receive morphine preoperatively in this study. In contrast, of those who were given the combined drugs, 4 per cent were under six and 11 per cent were seventy years of age or older. The oldest patient on the combined drugs was ninety-four. The poorer clinical status of those who received the combined drugs is shown by the following facts: In the morphine group 97 per cent were considered good and 3 per cent fair operative risks. On the other hand, only 83 per cent of the combined drug group were classified as good, 13 per cent as fair and 4 per cent as poor risk cases. Again, 4 per cent of the patients in the morphine group, as against 9 per cent in the combined drug group, had diseases other than those which required surgical intervention. We were not concerned over these disparities in the groups because we were of the opinion that if the combined drugs had significant advantages over morphine, they would become apparent in spite of the poorer clinical condition of the test group.

When the patients were brought to surgery, notes

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Presented before the Minneapolis Surgical Society, December 2, 1954.

*Levallorphan tartrate is the generic designation for levo-3-hydroxy-N-allylmorphinan tartrate (RO 1-7700).

†The premixed solution of Levo-Dromoran Tartrate and RO 1-7700 was made available for this study in 10 cc multiple dose vials by Dr. M. J. Schiffrin, Hoffmann-La Roche, Inc.

TABLE II. PREOPERATIVE AND POSTOPERATIVE CONDITION

	Morphine	Combined Drugs
Total Number of Cases	98	364
Preoperative reactions		
Drowsy	56%	71 %
Sleep	8%	19 %
Nausea	2%	0.6%
Emesis	0	0.6%
Respiratory depression	11%	2 %
Hypotension	0	2 %
Hypertension	2%	0.6%
Bradycardia	2%	2 %
Tachycardia	1%	3 %
Euphoria	4%	7 %
Apprehension	9%	4 %
Vertigo	0	1 %
Others	0	0.3%
Reactions during surgery	3%	2 %*
Condition in recovery room		
Good	94%	96 %
Fair	6%	3 %
Poor	0	0.3%
Awake	52%	54 %
Drowsy	45%	45 %
Depressed	3%	0.3%
Total recovery room reactions	28%	9 %
Time in recovery room		
90 minutes or less	60%	69 %
91-120 minutes	17%	15 %
More than 120 minutes	23%	16 %

*Including death of cardiac arrest during surgery on the heart.

TABLE III. REACTIONS IN RECOVERY ROOM

Reaction	Morphine (Per Cent of Cases)	Combined Drugs (Per Cent of Cases)
Cardiovascular	13%	5.0%
Respiratory Depression	5%	0.3%
Euphoria	2%	0.4%
Nausea or Emesis	2%	2.0%
Disorientation	2%	0.3%
Miscellaneous	4%	1.0%
Total	28%	9%

were made of their condition with particular attention being given to such reactions as: drowsiness, sleep, nausea, emesis, respiratory depression, hypotension, hypertension, bradycardia, tachycardia, euphoria, apprehension and vertigo. All inhalation anesthesia was given with assisted or controlled ventilation. Careful records were kept of reactions during surgery as well as of the amounts of other agents used during the operative procedures. Similarly, notes were made of reactions in the recovery room and of the length of time the patient remained there. Our standard procedure to keep patients in the recovery room for at least one hour, regardless of their condition, was followed in this study.

Results

An analysis of the results obtained is shown in Table II. The immediate preoperative condition of the two groups did not differ appreciably with reference to the incidence of nausea, emesis, hypotension, hypertension, bradycardia, tachycardia and vertigo. The combined drugs produced more sedation (90 per cent vs. 64 per cent) and euphoria (7 per cent vs. 4 per cent) than did morphine. The combination yielded less respiratory depression (2 per cent vs. 11 per cent) and less apprehension (4 per cent vs. 9 per cent) than did morphine. The anesthetists, who did not know which drug the patient had received preoperatively, could pick out those who were given the combined drugs because they were more relaxed and had better skin analgesia than the morphine group. There was no significant difference in the incidence of reactions during surgery (2 per cent for the combined drugs, 3 per cent for morphine). The only death in the two groups was a poor risk cardiac patient who had received the combined drugs and died of cardiac arrest during surgery on the heart.

There was no significant difference in the percentage of patients who were in good condition on admittance to the recovery room. It was significant, however, that in spite of the poorer preoperative clinical condition of those cases which were given the combined drugs, the incidence of depressed patients was ten times as great in the morphine group as in the combined drug group (3 per cent vs. 0.3 per cent). Moreover, there was a significant difference in the frequency of reactions in the recovery room; as can be seen from Table III, listing these reactions, their over-all incidence in the morphine group was 28 per cent, as contrasted by an incidence of 9 per cent in the combined drug group.

This difference in the frequency of reactions in the recovery room is paralleled by the difference in the time the patients of the two groups had to be kept in the recovery room. As appears from Table II, a higher percentage of patients in the combined drug group had a significantly shorter stay in the recovery room. This is all the more remarkable since these were the cases whose preoperative condition was generally poorer.

An analysis of the frequency with which various agents were used during surgery indicates a similar distribution in both groups. The frequency distribution in the combined drug group was as follows: Pentothal® was given in 325 cases; nitrous oxide and oxygen in 271; curare in 263; cyclopropane in 95; intrathecal Pontocaine®-ephedrine-dextrose in 71; Tensilon® in 35; ethyl ether in 19; combined drugs (intravenously) in 17; morphine (intravenously) in 15. Other agents used on five occasions or less were: Wyamine,® vinethene, atropine, cortisone, cocaine, insulin, Cedilanid,® procaine, and aminophylline. The average amount of Pentothal per patient in the morphine group was 682 mg. and in the combined drug group, 631 mg. Of the patients who in the combined drug group received Pentothal, 42 per cent required less than 500 mg., whereas only 32 per cent of the morphine group were in this category.

The advantages of the combined drugs were most apparent in poor risk patients, particularly in those in whom major surgery was performed. The following cases may serve as examples: A seventy-four-year-old woman, a very poor operative risk with a vital capacity of 1.2 liters, was given preoperatively 1.5 mg. Levo-Dromoran Tartrate (in combination with 0.15 mg. levallorphan tartrate) and 0.3 mg. atropine. She was relaxed but not sedated and had no preoperative reactions. An exploration of the right chest was performed using 750 mg. Pentothal, 21 mg. curare and endotracheal nitrous oxide-oxygen. Respirations were controlled throughout the procedure. There were no reactions during surgery, and she was awake and responding when admitted to the recovery room. All vital signs were stable. She was given 1 mg. Levo-Dromoran Tartrate (in combination with 0.1 mg. levallorphan tartrate) for pain relief and was released from the recovery room after sixty minutes. The second example is that of an eighty-seven-year-old woman who had a cerebral accident with hemiplegia (four days' duration), a mediastinal tumor and who had swallowed an open safety pin. In spite of her very poor condition, 1.5 mg. of Levo-Dromoran Tartrate (with 0.15 mg. levallorphan tartrate) and 0.3 mg. atropine were given preoperatively. She was drowsy on arrival in surgery. An esophagoscopy was performed under Pentothal and curare, supplemented with endotracheal nitrous oxide-oxygen. Ten mg. Tensilon were given by the intravenous route immediately after completion of surgery. She was awake and comfortable when admitted to the recovery room. All vital signs were stable. She was returned to her room in sixty minutes. The oldest patient who received the combined drugs was ninety-four years of age. She had an uneventful and successful repair of a fractured hip using intrathecal Pontocaine-ephedrine-dextrose. The preoperative disorientation disappeared following the administration of 1.5 mg. Levo-Dromoran Tartrate (with 0.15 mg. levallorphan tartrate) and 0.2 mg. atropine.

Discussion

Our experience with the combined drugs indicates that they are safe, effective, predictable and have advantages over morphine as preoperative medication. There was with this combination better sedation and greater freedom from apprehension than with morphine. This is particularly advantageous in children. The combined drugs produced less respiratory depression preoperatively and postoperatively than did morphine. Pain and local reactions at the injection site were absent when the combined drugs were given. Occasional reactions of this type were seen after the administration of morphine sulfate.

The combined drugs provided for easier management of the patients during surgery. Intrathecal injections were more easily made. Pentothal and intravenous opiate requirements were reduced.

The wide variety of agents used during surgical procedures following the administration of the combined drugs attests to the safety of this new type of preoperative medication. In seventeen instances the combination was given intravenously as a supplement to nitrous oxide-oxygen anesthesia. The results were uniformly satisfactory.

The postoperative course of those patients who had received the combined drugs was better than that of those who had been given morphine. The former were more co-operative, better oriented, free of pain and easier to manage in the recovery room. The opiate requirements and the number of reactions in the recovery room were reduced in the patients who had received the combined drugs. Children were more sedated and easier to manage. The secondary effect on the parents was of no little consideration. Elderly patients did not "climb the walls," indeed, they were more mentally alert at times following the administration of the combination than they were beforehand. The over-all shortening of time in the recovery room was an important factor economically. The addition of 1 part levallorphan tartrate to 10 parts Levo-Dromoran Tartrate made it possible to provide intense analgesia without depression. Our experience has been so rewarding that the combined drugs have been made standard preoperative medication in this hospital.

Summary

1. A premixed combination of 10 parts Levo-Dromoran Tartrate and 1 part levallorphan tartrate was given preoperatively by the subcutaneous route without adverse effects to 364 patients. The results were compared with the effects of morphine in ninety-eight patients.
2. The combined drugs provided greater sedation, euphoria and freedom from undesirable reactions than did morphine. There was less apprehension and respiratory depression both pre and postoperatively when the combined drugs were used.
3. The amount of Pentothal and intravenous opiates required during surgery was reduced following administration of the combined drugs. The use of the combination preoperatively was compatible with a wide variety of agents commonly employed during surgical procedures.
4. Patients who received the combined drugs had fewer reactions in the recovery room and were sent back to the floor sooner than those who were given morphine.

Acknowledgment

The excellence and completeness of the individual records could not have been possible without the wholehearted interest and co-operation shown by the nursing staff on the floors and in the Department of Anesthesia. My sincere thanks to them all.

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Addendum

Since the establishment of routine use of the combined drugs, further experience has shown that scopolamine with the combined drugs can be given up to age seventy without danger of depression. No Seconal is given preoperatively to these patients.

When this paper was written, we did few tonsillectomies because of the polio season. Since then, the combined drugs have been used routinely preoperatively for tonsillectomy in children.

Our staff in the recovery room has reported that these children respond more quickly, have little or no nausea, require airways for a shorter time and need much less postoperative sedation than previously. As a consequence, the minimum time in the recovery room, post-tonsillectomy, has been reduced from sixty to thirty minutes.

Current Concepts

THE VALUE OF THE ELECTROCARDIOGRAM IN DIAGNOSIS

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Nearly fifty years have passed since Einthoven developed the electrocardiograph, and a quarter of a century since it became generally available. Today, thanks to the development of small, lightweight instruments, an electrocardiogram is regarded as a routine laboratory request in most general medical examinations and a requirement in any cardiac evaluation.

In the diagnosis of myocardial infarction, the usefulness of the electrocardiogram has been greatly extended by a more thorough precordial study using Wilson's unipolar leads. The six positions, V-1 through V-6, are generally agreed upon as necessary, with back leads or leads in higher or lower interspace levels desirable in certain doubtful cases. Smaller areas of infarction are now being detected with these leads. As a result, the over-all prognosis of myocardial infarction has doubled in the last fifteen years.

Coronary insufficiency remains a problem in diagnosis. An electrocardiogram taken at rest may be normal in the presence of definite coronary sclerosis. Various tests such as Master's two-step exercise test, are useful in detecting coronary insufficiency, but false negative, and, occasionally, false positive tests add to the problem.

Increased accuracy in the diagnosis of ventricular hypertrophy has followed more complete precordial lead studies. Many difficulties remain, however, especially when right and left ventricular hypertrophy are both present. The thoracic roentgenogram may reveal cardiac enlargement undetected by electrocardiography, and vice versa. A left ventricular strain pattern may appear before x-ray evidence of enlargement of the left ventricle in the early stages of hypertensive heart disease, and occasionally concentric left ventricular hypertrophy in aortic stenosis is more evident by electrocardiography than on fluoroscopy.

Arrhythmias remain the special province of electrocardiography. Here, the precordial lead V-1, overlying the right atrium, is especially useful. More or less continuous tracings are advisable in the conversion of ventricular tachycardia to normal sinus rhythm when intravenous Procaine amide is employed, allowing for better control and early recognition of toxic signs.

The rapid growth of cardiac surgery in congenital and acquired heart disease has called upon electrocardiography as an aid in correct presurgical diagnosis. For example, the presence of right axis deviation should raise immediate doubt as to the presence of uncomplicated patent ductus arteriosus. Similarly, left axis shift and evidence of left ventricular strain may point to significant mitral regurgitation in a case of mitral stenosis being considered for mitral commissurotomy.

Thanks to much electrocardiographic and pathologic investigation, the diagnosis of myocarditis is again fashionable, and myocardial involvement in the course of many types of viral disease and a host of other bacterial diseases, including pneumonia, diphtheria, and rheumatic fever, is recognized. Isolated, or Fiedler's, myocarditis is a particularly fatal form of unknown etiology. Myocardial involvement in the collagen diseases—lupus erythematosis, panarteritis nodosum, and scleroderma—may also be demonstrated by electrocardiography.

In differentiating primary idiopathic pericarditis, by no means a rare disorder, from acute myocardial infarction, the electrocardiogram is most valuable. The benign character of the former and the grave prognosis of the latter demand a correct differential diagnosis.

The electrocardiogram may also be useful in the diagnosis of a number of other diseases, including myxedema, thyrotoxicosis, beri beri, acute and chronic cor pulmonale, primary or metastatic tumors of the heart. It may also reveal effects of quinidine, digitalis, and electrolyte imbalance associated with hypokalemia and hyperkalemia and hypocalcemia.

The limitations of the electrocardiogram in diagnosis, unfortunately, are not generally recognized. Only a few of the patterns mentioned above are truly diagnostic. Left ventricular strain and coronary insufficiency are often impossible to differentiate, and both may be confused with digitalis effect. Beri beri, pericardial effusion, and myxedema may produce similar patterns. Even myocardial infarction is not necessarily a specific pattern, a similar one being produced by stab wounds of the heart. The boundary between normal and abnormal electrocardiograms may also be obscure and open to varying diagnoses by different electrocardiographers. The following current classification of electrocardiograms reflects these difficulties: within normal limits; probably normal; borderline ECG; probably abnormal; definitely abnormal ECG.

Finally, let us not forget that an electrocardiogram cannot be substituted for a well-taken history and a thorough physical examination.

Laboratory Aids

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George G. Stilwell, Editor

LABORATORY AID IN THE DIAGNOSIS OF LUPUS ERYTHEMATOSUS

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Systemic lupus erythematosus (LE) is a disease of unknown cause. Aggravating factors are recognized, such as infection, exposure to sunlight or cold, allergic reactions and surgical procedures. The general symptoms occurring during activity of the disease are fever, weakness, fatigue, prostration and loss of weight. Arthralgias and joint symptoms are seen in many patients, and often it is difficult, without prolonged follow-up, to distinguish between systemic LE and rheumatoid arthritis. Various cutaneous manifestations have been described, among them the classic open "butterfly wings" on the face. Evidence of disturbances of the central nervous system or of lesions of the pulmonary and cardiovascular systems occurs frequently. Lymphadenopathy and splenomegaly occur in more than 50 per cent of patients.

It has been stated that LE has assumed an importance disproportionate to its incidence as a clinical problem. The main factors responsible for this emphasis are (1) recognition of multiple systemic manifestations in LE in addition to cutaneous lesions, (2) development of technical procedures and criteria for more exact diagnosis, (3) increased interest in the entire group of so-called collagen diseases and (4) discovery of cortisone and corticotropin (ACTH), which offer a limited approach to treatment.

Laboratory findings include anemia, leukopenia, proteinuria and hematuria. Elevation of the erythrocytic sedimentation rate is frequently observed and is roughly proportionate to the activity of the disease. False positive results of serologic tests for syphilis may occur.

The LE Cell

In 1948, Hargraves and associates described a peculiar cell in the buffy layer of bone marrow taken from patients who had acute and subacute lupus erythematosus. This cell, the well-known LE cell, is a mature polymorphonuclear leukocyte containing an ingested or lysed mass of homogeneous, pale-purple material thought to be of nuclear origin. It was noted also that several polymorphonuclear leukocytes might be attracted to a single large mass of lysed nuclear material, forming a "rosette." A cell similar to but not identical with the LE cell was described in the bone marrow of patients who had a variety of chronic disorders. This was called a "tart" cell, which usually consisted of a monocyte with a secondary nucleus that apparently had been phagocytized. This same nucleophagocytosis also was noted in polymorphonuclear neutrophils. This cell is rarely a source of confusion to experienced observers.

Since the discovery of the LE cell in bone marrow, various tests have been developed using peripheral blood. Comparative studies have been done by many investigators, who now agree that the production of LE cells as seen in the various preparations of blood and bone marrow is an *in vitro* phenomenon occurring in the presence or absence of anticoagulants but depending on three elements, namely (1) the lytic, or LE, factor contained in the gamma globulin of the plasma, (2) nucleoprotein to react with the lytic factor and (3) active polymorphonuclear neutrophils to engulf the lysed material.

Clot Test for the LE Cell

One of the most satisfactory laboratory procedures for the demonstration of LE cells is the so-called clot test, as it has been demonstrated that formation of LE cells is enhanced by coagulation. The greater sensitivity of the two-hour blood-clot technique (to be described) as compared with results of examination of bone marrow should not be construed to indicate that the LE factor is in greater concentration in blood than in bone marrow. Two factors in the routine handling of

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This is the thirty-first in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

bone marrow tend to limit formation of LE cells; these are the inhibition of clotting produced by use of heparin and the fact that preparations of smears usually are made within twenty minutes after aspiration.

Technique.—Venous blood (10 ml.) is withdrawn and allowed to clot for two hours in a clean glass tube. The clot is removed and placed on a special sieve made of stainless-steel wire gauze, 40 meshes per inch, fitting over a Petri dish. By means of a pestle, the clot is mashed through the screen, which removes the fibrin. A Wintrobe sedimentation tube is filled with the blood that accumulates in the Petri dish under the sieve. The tube is centrifuged at 2,000 r.p.m. for five minutes. Smears are made from the buffy coat after the serum is aspirated down to the layer of leukocytes. The smears are stained by Wright's method and examined for LE cells.

Comment

Although the procedure for making the LE preparations is relatively simple, their study should be done by a trained observer because certain difficulties may arise in the interpretation of results of the LE clot test. Histiocytes and, not infrequently, neutrophilic polymorphonuclear leukocytes may contain a secondary nucleus that has a sharp nuclear membrane, and there may be a relatively sharp differentiation between chromatin and parachromatin. This nucleophagocytosis ("tart" cell) is identical to that seen in bone marrow. As already indicated, such cells may be confused with LE cells by an inexperienced microscopist. LE cells found in the clot test are derived from mature neutrophilic polymorphonuclear leukocytes in the peripheral blood containing pale-purple intracytoplasmic masses of lysed chromatin. Lysis must be sufficient to impart a digested, smoky or ground-glass appearance. Such cells resemble in all respects the LE cells of bone marrow. In preparations made from clotted peripheral blood, clumps of leukocytes may be present at the edge of the smear. Some of the neutrophils in these clumps undergo distortion, which results in nuclear disintegration resembling the lysis of nuclear material seen in LE cells. One must be careful not to mistake damaged polymorphonuclear neutrophils for LE cells.

Small aggregations, or rosettes, of polymorphonuclear neutrophils are frequently seen in preparations containing LE cells. These rosettes should be distinguished from the clumps of leukocytes just described. Extracellular, pale-purple, amorphous, nuclear masses are often seen in preparations containing LE cells. The presence of rosettes and of free amorphous nuclear masses creates suspicion, but positive results of the test should not be reported unless true LE cells are seen.

In a correlation of the results of the LE clot test with the clinical diagnoses in a series of 347 patients, it was noted that the results were positive in all patients who had subacute to acute disseminated LE, whereas the results were negative in those who had chronic to subacute lupus erythematosus or discoid lupus erythematosus.

A small percentage of patients with rheumatoid arthritis have LE cells in the clot test. Also, certain patients receiving hydralazine (apresoline) hydrochloride for hypertension may present symptoms not unlike those of LE, and clot tests may reveal LE cells. When use of the drug is discontinued, the symptoms clear up and the LE cells disappear from the clot test, although such disappearance may lag behind the clinical improvement.

LE cells have been reported by some workers in disorders such as periarteritis nodosa, dermatomyositis, hyperglobulinemia not related to LE, pernicious anemia and reactions to drugs. Other workers have been unable to find LE cells in these conditions.

The consensus is that the LE clot test is a valuable laboratory aid in the diagnosis of lupus erythematosus. The test cannot be used for prognosis in spite of the fact that the cells may disappear during remission. There are factors regarding the sensitivity of the test that are not clearly understood at present. Results of the clot test may be negative in an occasional case of acute disseminated lupus erythematosus.

Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

FREEDOM FOR PHILANTHROPY

Freedom to exercise one's will is a right which men have fought to exercise—and when lost, fought to restore—since time immemorial. We, as Americans, are engaged today in such a struggle to retain that elusive quality, most often called "liberty," on many fronts.

This may seen an unusual introduction to a discussion of independent vs. federated fund-raising for health and welfare agencies. Yet, to many, free will is the heart of the question.

Philanthropic giving is a unique characteristic of this country. In no other nation have private citizens rallied to support medical research and provide aid for the handicapped and unfortunate on the scale that we have achieved. In other countries, where such programs are carried on at all, they usually become a function of governmental agencies supported through taxation. Any step which may threaten this desirable American tradition should be taken cautiously.

Which agencies one should support is a question that no person can answer for another. Each individual differs in his approach to a given cause. Some may think one far more worthy than another—and give in that ratio. Some, through personal experience, may feel that an agency is not worthy of his support. That is, and should be, his decision to make.

If an agency cannot or does not impress the public with the merit of its cause, that agency should not be supported long. Thus, free will as expressed by the public becomes a "police force" for weeding out groups which do not merit support or duplicate the work of other agencies.

In the case of the so-called "federated" or "united giving" plans, the expression of one's free will is easily dissipated. By default, the individual's decision as to which agencies he will support and in what amounts, is all too often passed on to others. Thus, the very heart of philanthropic giving is touched.

Philosopher John Dewey once observed that we should not destroy one institution, in this case—philanthropic giving, unless we are certain we replace it with one that is superior.

Independent thinking is a painful process, no matter how educated the person. It is much easier to be persuaded by catch-phrases or appealing rationalizations than to think a problem through.

Before you, as a practicing physician and a leader in the community, make a decision concerning independent vs. federated fund-raising, satisfy yourself that you have the correct answers to the following questions—just a few of the many involved:

1. Do you decrease the number of agencies by pooling them into a single fund drive? Or the total amount of the funds sought?
2. Why, if combined fund-raising is a panacea, are there so many federated drives in this state experiencing difficulty in maintaining adequate public support?
3. Isn't it possible to budget the support for health and welfare agencies as one would do in his home or business and reduce the irritation of haphazard giving?
4. Are the individuals who control federated funds elected in a manner consistent with democratic principles?
5. Do you favor such a socialized approach to the solution of our community and national problems?
6. Are the reasons for conducting such a plan strong enough to warrant the action?

The question of independent vs. federated fund-raising may be troublesome—but not a menace. The loss of freedom to exercise one's will is!

G. RAY HIGGINS,
Executive Secretary
Minnesota Heart Association

FAMILY SERVICE—WHAT IS IT?

Across the United States and Canada, in every city of any size, there are Family Service agencies. Altogether, there are 245 of these agencies, if we include those in Alaska and Hawaii. Approximately 45 per cent of the entire population of the United States is accessible to this service.

Family Service agencies are members of the Family Service Association of America and must

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First editorial in a series on "Family Service."

meet required standards which include: A staff of trained social workers, most of whom have a master's degree in Social Case Work; a lay board serving without pay; adequate financing from private funds; proven ability in offering skilled case work to families and children. In addition, statistical reporting must be uniform so that valid comparison between agencies and communities can be made.

In Minnesota, five agencies meet these qualifications. These are: The Jewish Family Service of Saint Paul and Minneapolis, and the non-sectarian Family Service of Saint Paul, Minneapolis and Duluth.

The historic background of many of these agencies is similar. In Saint Paul, Family Service began in 1892 as the Associated Charities, for coordinating voluntary relief. Later, it became the United Charities, and tried to work on the cause of poverty while still giving financial aid. Now, as Family Service, its responsibility is to help families with social and personal problems make the best use of their own and community resources, so that they can live full and satisfying lives, and raise happy, well-adjusted children.

Tensions arising out of personal relationships are the most common cause of application for our service. Marriage partners who have lost their direction and find themselves growing apart; parents who no longer understand their children and who are concerned because their children are no longer in harmony with them; emotional problems which interfere with social well-being—these problems, or some aspect of them, are the area that Family Service agencies are prepared to serve.

The case workers in Family Service agencies are in a position to see the effects of illness, both physical and emotional, on the entire family, and also to see the effects of family tension, as they aggravate physical and mental illness. For this reason, family agencies and physicians have much in common and can often be mutually helpful by working together for maximum use of both resources in helping families with problems.

As a private non-sectarian family agency, people of all religions, races, and economic and social levels, are eligible for service from Family Service of Saint Paul. The only requirement is that they live within the area which supports the Greater Saint Paul Community Chest which raises the funds that support the service.

In order to help with the often complicated and diverse family needs, special services have been developed which include foster care for children, homemaker service, financial and home management counseling and others, which will be described in later issues. The Family Service of Saint Paul welcomes this opportunity to make its service better understood by the medical profession. The family agency and the physician can both give greater service if they understand and work together for the benefit of man.

Dawson Bradshaw, *Executive Secretary,*
Family Service of Saint Paul

STALL THE EXECUTIVE HEART ATTACK

While walking in a cemetery recently, I came upon the ornate head stone of an executive friend of mine. He had died recently from overwork. This was the inscription.

> "Here lies Joe,
> A nice guy to know.
> He couldn't get his people doing
> Work without his always stewing."

Yes, lots of executives stew about their men. "Why don't they handle responsibility?" "Why don't they carry through?" "Why don't they plan?" "Why don't they get things done?" And it's this stewing—this feeling of having personally to stay on top of every detail all of the time—that gradually exhausts the executive.

But how do you get men to do a job? How do you get them to do it without overburdening the executive?

Delegate Details—We often find executives stewing over details that other people should handle. I remember a president who was doing detailed accounting and credit work. Another president was passing on all buying. The net result—they had less time for planning and stimulating men. As a consequence, problems were constantly coming to their desks. They were working twelve to fifteen hours a day.

Get Men to Plan.—All too often, an executive hasn't forced planning down the line onto his men. He has permitted them to bring all planning up to him.

For example, a sales manager should be forced to plan his territories in such a way that he can meet competition. If he comes in crying, "Competition is getting rough," and gets by with it, he has simply dumped all his problems on his boss's desk. The executive who permits this starts worrying about things that he really should have others handle.

Let "Them" Do It.—Sometimes a company is afflicted with "committee-itis"—all executives are in one committee meeting after another. Endless time is spent trying to get things done through committees. The difficulty with this is that committees generally don't take responsibility. In addition, they often develop into buck-passing bodies. What happens? More and more problems come up to an executive's desk. It was assumed the committee would handle them. But no one in the committee took real responsibility!

A firm may have a management committee, sales committee or a special production committee that reviews every little problem, but no one takes any real action. Key people spend a lot of time in meetings. But problems are still dumped in the lap of an executive for him to worry about. *And* other people could easily have solved the problem!

Oh, For a Staff in Time.—As a company grows it becomes evident that staff people are needed in addition to line. (Ordinarily, we would define a staff man as "anyone who has to get something done for the company through someone else who doesn't report to him." For example, personnel people, cost accountants, production control men, and market research men would be staff. Salesmen, production operators, and foremen would be line.) It's easy to set up one staff position after another. Very often, the need for the staff people isn't really there. The line could have handled it. *But every time you add a staff person, you add more problems of co-ordination.*

Sometimes the staff people are needed for a temporary problem, but after the problem disappears, the staff carries on. These staff people, then, look for things to do to justify their existence. Generally, they look in other people's bailiwicks. This usually leads to trouble. And where does this trouble come home to roost? Right on some executive's desk! And so he has one problem after another trying to solve conflict between staff and line.

Often staff people start taking over responsibilities that belong to the line. The line, then, won't take responsibility. For example, the personnel department may handle employe problems. The inspection department may relieve foremen of quality problems. The market research department starts to relieve sales managers of sales planning. This leads to weak line people and usually to poor operation. The staff people actually *cause* more and more problems, because the line people have relaxed their own attention to them. This results in more problems coming up to the executives, problems that could have been solved by men below them.

You Can Prove It with Figures.—As a company grows, it may easily succumb to a creeping paralysis of figure men. One report after another is developed, because it's "essential" to the business. Each of these reports goes to the executive's desk. This forces a lot of reading and concentration on his part to study them. In addition, these record keepers get in the hair of the other people by demanding so much of their time for making up reports. All of these things add to the problems of co-ordination of the executive.

How should you set up record keepers? Each record man should be responsible, not for records, but for getting some constructive action in the company as a result of the records. You then find that you relieve the executive considerably. You force the records to produce value. You help prevent their excessive growth. And you force the records to go to subordinates to make decisions. Without this you have an added burden for the executive—something else to stew about.

"You've Got Full Authority."—Too often, people assume that simply saying, "You've got full authority" solves all problems of authority. Many an executive worrying about the fact that men don't take responsibility has fallen into this trap. But authority is a very complex thing. It means nothing unless the man "feels" that he has it. What does authority mean? It means the right to make minor mistakes and to make them individually with impunity. In this connection, we suggest the rule of errors:

> "If a man has responsibility for a result, he should be allowed to make the necessary decisions *and the normal errors* that those decisions might result in in arriving at that result."

Too often, an executive has become concerned about every little error that people make along the way. He calls these to the attention of the man. As a result, nobody will stick his neck out and take any action. Things don't move. In other words, he has not really given authority.

Authority really means the right to make some errors. Actually, the executive that does not allow his men to do this starts stewing about every little error that comes along. Once he realizes the importance of the rule of errors, it becomes much easier for him to tranquilly accept the little things that go wrong along the way.

Make Them Accountable.—The very life blood of an organization is accountability. Very often, the reason a very fine set-up doesn't work is that people individually are not accountable. In other words, individual men do not get credit or discredit if certain results are or not achieved.

When you don't have accountability, you develop an excessive burden on the executive to keep pushing and pushing to get things done. This alone can easily result in pushing the executive beyond his powers or his energy. Problems occur because people have been allowed to make decisions without being accountable for them.

For example, the sales department may be allowed to exert a great deal of pressure to get research done on crackpot ideas but not be accountable for the cost of that research. But the executive is! Perhaps a superintendent does not feel accountable for costs, but the executive at the top is quite concerned about the cost trend in his company. Until the superintendent is held accountable for the cost of operation of the plant so that he feels that it's his job to keep it actually in line, there is just one more item for the executive to worry about.

This also applies in product development departments. Development men love to turn out a beautiful "well-engineered" product. But perhaps it's one that will sell for $30 when the market will only take a $15 item! Until you make the development man accountable for producing a product that can be made at a salable price, you find you have numerous problems of co-ordination in production, sales, and development. This adds to the already heavy burden of the executives at the top.

Let's Make Men Successful.—Many executives haven't developed the spirit in their organizations that it's the job of every supervisor to make his men successful. To the extent that an executive permits men to pass the buck downward, people do not take responsibility. Duds show up. Action isn't taken. He should establish the attitude in the company that it's the job of every supervisor to make his people successful, and that a man's failure is probably the supervisor's fault. He will then get better action down the line. Fewer of the problems have to come up to the executive.

How Many Can You Supervise?—Too many executives want to keep their fingers on everything. They're in constant turmoil trying to keep in touch with all angles of the business.

For example, I know of a president who had fifteen people reporting to him. People had to stand at the door waiting to get in. This put excessive pressure on him. He had so much to do that it was hard for him to keep on top of everything. He had to put in long hours in order to supervise his men. At that, he did it poorly. Problems were always popping up. He became a worry wart.

Keep Your Balance.—One of the most difficult principles for an executive to follow is that of balance. In other words, he must constantly put pressure on all functions to be sure they're all coming along. Otherwise, one will outstrip the other and problems will develop.

For example, one company put a lot of effort into sales, and it was productive. The only trouble was that competition was developing new products and production methods to such a point that this company was not competitive in price. This president was at his wit's end trying to keep his company afloat.

I know of another company that put all of its eggs in the plant production basket. This was fine! They kept costs down! Then sales dropped 35 per cent. Little effort had been put into sales. It wouldn't help to tell this president not to worry. With a 35 per cent drop in sales, what president wouldn't worry?

Often executives don't realize that some pressure on all functions is better than excessive pressure on any one at any one time. When a president gets the right balance of effort in all of the functions under him, he usually finds he can operate in a much more relaxed way. All areas of the company go ahead. He isn't caught short nearly so often.

These are only a few of the management problems that lead to excessive worry and stewing on the part of an executive. They lead to low production, high waste, and trouble. And it doesn't do any good to tell an executive, "Don't worry." He has to worry, or he feels he has to. He's got problems. But if he learns to straighten out these problems by following good management and organization principles, he finds that he can relax. He can plan better. The cogs of the organization are meshing. The whole company goes ahead on a smooth, easy basis, and the executive, himself, is able to find time to relax and to play a little golf.

Edward C. Schleh, *President*
Schleh Associates
(Management Consultants)

UTILITIES AS AN INVESTMENT

"Who owns the electric utilities?" is a question that is asked every utility executive many times and is the subject of a great deal of popular misconception. We believe that the answer to that question is good evidence of the standing of utility securities for investment purposes.

The electric utility industry in the United States is owned by approximately 3.7 million stockholders who, of course, are direct owners. They come from every walk of life including doctors, lawyers, merchants, farmers, laborers, office workers, teachers and housewives. Nearly every American has an indirect financial interest in the electric utility companies. There are, of course, also thousands of individuals who own bonds, debentures and notes of electric utilities who could be classified as direct owners. The 90 million life insurance policyholders and the more than 20 million depositors in mutual savings banks are examples of such indirect ownership, let alone those interested in pension funds, fraternal organizations, religious and educational institutions, and others that are heavy investors in utilities.

The Institute of Life Insurance points out that the nation's life insurance companies have nearly $13 billion invested in the securities of public utilities, and from that investment the policy holders are drawing about $400 million annually in interest and dividends going toward the enhancement of the value of their policies, the payment of dividends on their policies, or defraying the cost of their insurance. The Institute also shows that the $13 billion utility investment is the equivalent of nearly one-third of the total assets of all privately owned electric, gas, telephone and telegraph companies in the country; and that the long-term bonds, debentures and notes of such public utility companies owned by the life insurance companies represent two-thirds of such securities of all of these companies now outstanding.

As a general rule, all of the utilities of the United States have experienced a sound and steady growth for many years. As the electric light and power utilities represent by far the largest segment of the public utility industry, and as that segment is the part of the industry which I know best, I shall confine the rest of my remarks to the electric light and power companies.

This industry is only seventy-five years old, and during all that time it has supplied good service and stayed ahead of the electrical requirements of its customers. It has successfully withstood the rigors and trials of two world wars, several depressions, and a period of years when the government itself seemed bent on "duplicating it" out of business. The industry now stands in full stature, with an enviable record of continually improving its efficiency of operation, paying a heavy load in taxes, and continuously reducing the average cost per kilowatt hour for service to its customers.

Since World War II, the electric industry has grown at a rate that doubles itself every ten years and present indications are that it will do so again in the next ten years.

Although the federal government has expended many millions of public funds for investment in electric generating facilities, it still supplies only about 13 per cent of the total electrical requirements of the nation. We have enough faith in the American system and in the American people to believe that they will not permit our industry to be destroyed or duplicated out of business.

The privately owned electric utilities are taking an active part in studies looking toward development of a cheap and reliable method of using nuclear fuel for the generation of electricity and will be quick to convert to this new type fuel once it is convinced it is cheaper than other means of generation. In our opinion there is no reason to be concerned about the obsolescence or replacement of present electric generating plants because of developments in the utilization of atomic energy in the generation of electric energy because it will only replace present fuels used for that purpose. About 83% of the present investment in a utility plant would still be required and we believe the change over, if it ever comes, will be so gradual that it will not materially affect the useful life of present or conventional plants.

It therefore appears that the electric light and power industry should continue to prosper and provide fair earnings for the payment of interest and dividends on the monies invested in its securities. The future looks good.

Allen S. King, *President*
Northern States Power Company

THE PRESENT STATUS OF THE BALLISTOCARDIOGRAPH

Interest in the ballistocardiograph as a diagnostic tool has increased rapidly since the end of World War II. There are now available at least three monographs[1-3] describing the several kinds of recording apparatus and giving clinical experience. Approximately 300 papers have been written on the subject since 1947. This interest in the ballistocardiograph is justified in part because it measures something related to the power or strength of the cardiac muscle[4] and therefore is concerned with a component of cardiac function which is not measured by the electrocardiogram or any other tool of the cardiologist.

The ballistocardiogram may be recorded with the patient lying supine on a moving bed which may, or may not, be restrained with springs.[5-9] or the patient may be on a fixed bed and the motion between the patient and the bed may be recorded.[10] A major problem is presented by the fact that the dorsal tissues have elastic and damping properties which impose distortions on records taken with both the popular direct body recording introduced by Dock and with bed systems restrained to a significant degree by springs.[11] A system has been recently designed which appears to have overcome most of these difficulties,[8] but this instrument has not been studied in the clinic. Ballistocardiography is currently going through a phase of instrumentation development which will not be over for some time.

In spite of the fact that records obtained with spring-restrained beds and the direct body pick-up devices contain important distortions both of amplitude and of timing, a great deal of useful information has been accumulated with these instruments. Starr[4] and his associates studied the effects of ejecting known quantities of blood of different rates into the aortae of warm cadavers. It is possible to produce both normal and abnormal ballistocardiographic patterns. This work demonstrated that abnormal ballistocardiographic wave forms *may* be produced by "weakness" of cardiac contraction. However, one should not lose sight of the fact that extracardiac factors, such as changes in the elastic properties of the large vessels and the tendons which anchor them, have not been properly evaluated. Heart disease, in which the myocardium has been damaged, is usually associated with abnormal wave forms and/or low measurements of force (usually computed from the IJ limb). In the case of conges-

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From the Laboratory of Physiological Hygiene, University of Minnesota, School of Public Health, Minneapolis, Minnesota.

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tive heart failure, abnormal patterns have been shown to improve with treatment and general improvement in the patients condition. However, the only abnormal pattern that appears to be diagnostic is that associated with coarctation of the aorta. Much work has been done on coronary heart disease where a high incidence of abnormal wave forms has been found with the use of the high frequency (Starr-type) bed and with the Dock apparatus. When groups of male coronary disease patients are studied with the low frequency critically-damped bed of Nickerson, the coronary groups are found to have mean IJ force values which are substantially below the mean of "healthy" men of the same age.[12] There is, however, a large overlap of values between the normal and coronary group. Study of the normal male population shows a striking decline in the IJ force with age, regardless of the method used to study it,[13-15] and a marked increase with age in the numbers of the otherwise healthy individuals who have apparently abnormal patterns obtained with either the high frequency (Starr-type) bed or the Dock body pick-up apparatus.[13,15] For example, in 174 businessmen, aged fifty to fifty-nine, who were studied in this laboratory with the high frequency (Starr-type) BCG, only 44 per cent were judged to have normal patterns. This fact places a very heavy burden of proof on the ballistocardiographer who suggests that an abnormal wave pattern in an otherwise normal individual has prognostic significance and makes current ballistocardiographs almost useless, as far as identifying significant coronary disease in the middle aged male is concerned. The interpretation of an abnormal pattern in an otherwise healthy individual is one of the major problems facing ballistocardiography today.[16] Until this problem is solved, the clinical usefulness of the ballistocardiogram taken with any of the instruments in common use will remain extremely limited.

It is much too early to give a final judgment on the value of the ballistocardiograph to the clinician. Much work remains to be done. The current improvements in instrumentation must be evaluated in the clinic. Detailed physiological studies must be carried out to describe the magnitude and type of errors which arise in the transmission of force through the internal network of tissues. In the meantime, there is evidence to support the concept[17] that the study of groups of individuals with the spring restrained models which are in current use will yield information which is of value to the clinical investigator.

HENRY LONGSTREET TAYLOR, Ph.D.

REINSURANCE IS NOT REASSURANCE

The old bogey-man, M. M. Davis, is on the loose again larruping the medical profession with might and main in his latest work entitled "Medical Care for Tomorrow," a book which regardless of one's views is packed with information. Dr. Davis has been known to many of us for years, to quote Albert Deutsch, as ". . . the unrelenting gad-fly on the back of the AMA mule." That the American Medical Association isn't completely mulish is shown by the fact that where it once objected to voluntary health insurance, it is now aggressively in favor of it. Dr. Davis is equally unmulish, as may be seen by his change of view. As a member of the Committee on the Cost of Medical Care back in 1932, he was against compulsory health insurance; now he is for it.

Sheer stubbornness is one thing; intelligent objection to a proposal is another. An unchallengingly negative attitude is not enough, and yet to object intelligently one must have sufficient knowledge to spot errors or misleading statements in the other's argument. There is much opposition in medical circles to federal reinsurance, and an understanding of Oveta Culp Hobby's plan requires more space than is available here. It may be briefed, however, in Mrs. Hobby's own words as follows:

> "If the XYZ Insurance Company decides it does not want to assume all the risk in this fleet of cars, it may—by paying a fee—assign, or in other words rein-

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[12] Laboratory of Physiology Hygience: Unpublished data.

[13] Scarborough, W. R., Davis, F. W., Jr., Baker, B. M., Jr., Mason, R. E., Singewald, M. L., Lore, S. A., and Fox, L. M.: A ballistocardiographic study of 369 apparently normal persons. Am. Heart J., 45:161, 1953.

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[15] Taymor, R. C., Pordy, L., Chesky, K., Moser, M., and Masters, A. M.: The ballistocardiogram in coronary artery disease. J.A.M.A., 148:419, 1952.

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[17] Nickerson, J. L., and Mathers, J. A. L.: A study of the physical properties of the ballistocardiograph. Am. Heart J., 47:1, 1954.

> sure, part of that risk with another insurance company or a group of insurance companies. In this way, should the insured fleet have excessive losses, the XYZ Insurance Company's liability would have been substantially reduced. This is straight reinsurance. . . . How do the terms apply to the federal insurance proposal? The proposal specifies that before the federal fund is liable for any claims, a reinsurance company must itself assume some loss above and beyond that anticipated in the calculation of its premiums. . . . A company can, by reinsuring with the federal reinsurance service, spread the risk of excessive aggregate losses in the same way as does the XYZ Insurance Company, which I have used as an illustration. The reinsurance proposal is not a subsidy. And it is not, of course, compulsory."

This all sounds quite plausible and convincing and no doubt it is true. However, there is another side to the matter. Read what Mr. Edwin J. Faulkner, president of the Woodmen Life and Accident Company, has to say:

> "Advocates of the measure (federal reinsurance) have noted the considerable contribution made to the development of life insurance, fire insurance, and other lines by adequate reinsurance facilities and, noting further the relatively small volume of health insurance business done, have argued that expanded reinsurance facilities in this field would have a stimulating effect. Such a conclusion can proceed only from a misunderstanding of the essential function of reinsurance, which is the spreading of risks larger than the initial insurer is willing to carry. It is true that in life insurance, for example, reinsurance makes it possible for an insurer to assume larger risks than it could without reinsurance. Here there is almost never a concentration of risk large enough to require spreading a potential loss. Even with catastrophic or major medical expense insurance, which presents the maximum amount of risk usually found on one life, the insurer's potential loss is seldom as much as $10,000, a sum well within the capacity of all but the smallest insurace company to carry alone."

He also says:

> "Reinsurance can distribute losses among insurers just as insurance distributes that among policy-holders, but no matter how far this distribution is carried, it must be sound to succeed. Reinsurance does not increase the ability of the insurer to sell protection to the unwilling buyer. Reinsurance does not reduce the cost of insurance. Reinsurance does not make the insurance available to any class of risk or geographic area not now within the capabilities of voluntary insurers to reach."

Mrs. Hobby views with alarm the fact that in 1948, $6.6 billion of total medical expense were not covered by insurance and that by 1953, $8.0 billion were not covered. There was thus an increase in the amount uncovered of $1.4 billion. That this is true, there is no question. It is also true, however, that $6.6 billion represented in 1948 92 per cent of the total medical bill, while $8.8 billion in 1953 represents about 80 per cent of the total. This might even be considered by some as an improvement. The percentages themselves are high, because the total medical expense figure used admittedly includes a large amount which under no conceivable insurance plan would be covered.

A recent editorial in one of our daily papers states:

> "The in-between middle class does not have the means to meet such costs (catastrophic illness) without hardship, and cannot at present get adequate protection."

It is, of course, true that the large group making up the so-called middle class consists of all variations between the moderately affluent and those in extremely straightened circumstances. To say at once that catastrophic insurance is not available to all or any of this class, is simply not true. There are forty companies in this country writing catastrophic insurance, with 1,500,000 persons covered. I have before me a policy submitted by one of the largest insurance companies in America, offering medical protection up to $5000, with the first $300 of expense deductible, for a man eighteen to thirty-nine years of age, together with his wife and a son and daughter, for $53 a year. It may thus be seen that this type of insurance is not only available but also accessible to millions who do not bother about getting it. With twenty-five million television sets throughout the land, the cost above-mentioned does not appear to be exorbitant.

Harold E. Hullsiek, M.D.

PSYCHIATRIC OUT-PATIENT TREATMENT

(Continued from Page 635)

a minimum or reasonable fee if required (Question VI).

6. 90 per cent of the respondents indicated that they would return willingly or be eager to return to the Center in the event of future psychiatric difficulty (Question VII).

7. 72 per cent of the respondents indicated they are well and healthy or bothered only occasionally by their condition; 18 per cent have received additional help from other non-psychiatric sources and 5 per cent have sought further psychiatric help (Question VIII).

8. 3.8 per cent have been hospitalized (Question VIII).

Reference

1. Kram, Charles, and Berkwitz, N. J.: Patients' impression of a community mental health center. Minnesota Med., 36,:591-593, 1953.

President's Letter

SOCIAL SECURITY FOR PHYSICIANS

Your officers and councilors are frequently asked why the House of Delegates of the AMA has recently voted to oppose the inclusion of physicians in the Old Age and Survivors Insurance Program, except on a voluntary basis. A wealth of material has been published in *The Journal of the American Medical Association,* both editorially and by Dr. Frank G. Dickinson of the AMA staff, and we all should be well informed on the subject if we take time to read the material available. However, I suppose the pressure of time has led many of us to neglect what should be compulsory reading and study. Dr. Walter Judd, a member of the Congress of the United States, recently wrote to me that he was surprised at the number of letters he received from doctors, urging that they be included under the social security program.

The subject of social security is particularly important to members of the medical profession. It is for this reason that the AMA has strongly supported the Jenkins-Reed-Keogh bills, which have appeared in various forms in Congress during the past sessions. These bills would establish a voluntary retirement plan for the self-employed and the pensionless-employed. They would amend the internal revenue code, but would not change the Social Security Act. They are in the public interest. The amount of each person's pension would be determined by how much he set aside for old age, and not one penny would be added to his fund by the Government. He would be permitted to defer taxes on the annual amount set aside each year until he retired and began to receive his pension, which would be currently taxable. The provision of these bills for tax deferment until retirement are a counterpart of those now enjoyed by employed persons covered by more than 20,000 tax-exempt pension plans. Dr. Dickinson has repeatedly pointed out in committee hearings in Congress that without such tax deferment, physicians are discriminated against.

Opposition to the trend toward the Welfare State might be called the fundamental reason for the opposition of the AMA to the extension of the Old Age and Survivors Insurance Program.

An ostensible reason for physicians to disapprove compulsory coverage OASI warrants only a brief comment. The median age at which self-employed physicians over age sixty-four are currently retiring is seventy-four. The retirement age of self-employed lawyers, dentists, farmers is probably about the same. Under the so-called work rule, no eligible person can draw Old Age and Survivors Insurance Pension if his earnings are over a stipulated amount. This would obviously mean that half of the self-employed physicians, under the President's proposal, would be required to pay social security taxes but not receive one penny of benefits until age seventy-four. But relaxing the work rule to make coverage more attractive to these ten million gainfully employed would, unless accompanied by a sharp increase in the OASI taxes, merely increase the get-something-for-nothing potential of the Act.

The basic objection to bringing an additional ten million persons under the OASI is that such an action by the Congress would be another long step toward big Government and the Welfare State. Social Security taxes, counting both employers' and employes,' are currently only token payments, and will defray only a portion of the costs of the OASI pensions. The tax rates of employers and employes should be doubled, and also graduated according to age—a change that is much more urgent than modification of the work rule. The unpaid balance of the pension cost as older persons receive their monthly pension checks must be paid by another generation.

Those who oppose the steady march toward the Welfare State must also refuse the bait—in this case pensions at cut-rate prices. They must oppose the doctrine of individual irresponsibility, except for the care of those in dire need, which, in fact, is the essence of the principles that guide the life of a physician in his practice. They must stand firmly against the exploitation of youth by those who were born many years earlier. They must insist on standing on their own feet and providing their own retirement needs. Those who can pay should pay their own way, those who cannot pay must, of course, be helped. It is apparent to students of our Federal social security system that some change in the present mechanism is necessary in order to show plainly to the entire country what the real cost of the existing level of benefits is, and then to have the present generation of the working population pay the full cost of the system in the same way we expect the next generation to pay its costs. There is every reason to believe that many advocates of the Welfare State are deliberately concealing the true cost of our Old Age and Survivors Insurance Program, in order to foist a much greater Federal system on the country before the people wake up to its real nature and cost.

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

HEALTH REPORT ON STATE RELATIONS ISSUED

The report by the Commission on Intergovernmental Relations, submitted to the President on June 28, 1955, deals briefly, but sympathetically, with Federal assistance in public health projects, hospital construction, research and professional training programs. Dr. Franklin D. Murphy, chancellor of the University of Kansas and former dean of its medical school, headed the fourteen-member committee.

According to the report, the Murphy committee agreed that public health grants by Washington are wise and desirable, then set down certain fundamental principles. These principles are:

1. Primary responsibility for meeting health problems rests with the community;
2. Aid grants should supplement, not supplant, local effort;
3. Community need is the first criterion for grants-in-aid;
4. Federal help should be of temporary nature;
5. Assistance funds should be matched, but with flexibility of formula;
6. Evaluation program is essential.

The report is quoted in a recent issue of "Washington Report on the Medical Sciences," which states:

> "The advisory committee recommended continuance at present level (about $57 million per year) of Federal grants to states for maternal and child health, tuberculosis, venereal disease, cancer and heart control, and general public health activities. It indorsed Hill-Burton hospital program but urged that voluntary health agencies be called upon for counsel before Congress grants this undertaking another extension (it is due to expire in 1957)."

The report further states that Federal financial support of medical research was heartily indorsed. In the matter of professional training, the committee recommended establishment of public health fellowships but warily avoided taking a stand on subsidies for medical schools, pending "an entirely objective study of this problem." Like the Hoover Commission and its medical task force, the Murphy committee favored creation of a Federal health council to co-ordinate.

CONGRESS ADJOURNS WITH HEALTH BILLS PENDING

The 84th Congress adjourned, leaving considerable unfinished business as far as health measures are concerned. Some issues, although they are lying dormant, will be considered by the second session which convenes in January. These issues include reinsurance of health insurance plans, guarantee of mortgages for health facilities and a series of bills dealing with U. S. grants-in-aid.

However, a recent issue of the AMA Washington Letter carried with it a special report of legislation status as the first session was nearing its close, and the actions taken are as follows:

Salk Vaccine

Many bills were introduced regarding control, distribution and use of biologicals. Last-minute compromise on these measurses provided $30 million to carry out the program of providing for inoculations of needy children.

Disability Insurance

This bill, providing cash benefits for disability, lowering the pension age for women and liberalizing the Social Security Act in other ways, was reported out of House committee and was passed by the House. Senator Byrd, chairman of the Senate Finance Committee which would consider the bill, is reported as being opposed to passage of this bill at this time, and would prefer a thorough study of the proposal before any action is taken. This will be an issue in the second session of Congress. The AMA is opposed to this legislation because (1) machinery at the Federal level to supervise the certification of disability would put the Government firmly in the middle of medical practice, and (2) pressures would be applied on physicians for speedy certifications.

Doctor Draft Extension

The doctor draft remains in effect another two years, until July 1, 1957. In the two major changes from the old act, the new law (1) lowers from 51 to 46 the age limit for call-up of physicians, and (2) provides that any physician rejected for a medical commission for solely physical reasons shall no longer be liable on reaching age 35. The $100-a-month special pay for physicians in uniform is continued for four years. The AMA has opposed extension. It feels that until this discriminatory law ends, firm steps will not be taken by the services to attract physicians to military medicine as a career.

Military Medical Scholarships

As a partial answer to military medical problems, the Defense Department proposed full scholarships for medical students. They would be obligated for three years' active duty if scholarship is a year, or five years if scholarship exceeds a year. This bill is still pending. The proposals are supported by the AMA but with three suggested changes: (1) a student not be approached by the military until fully matriculated, (2) no student so selected to receive any preferential treatment, and (3) number of scholarships in a school not to exceed 5 per cent of any year's class or total enrollment.

Military Dependents

Still high on the Administration's priority list is the plan for a broadened and uniform application of medical care for dependents of all servicemen. This care, within limitations of the military, would be provided in the main by uniformed physicians and military hospitals; where such care and facilities were not available, local physicians and resources would be called on. Progress has been slow, and this bill will await action until the next session.

The American Medical Association feels that the Defense Department's emphasis is in the wrong place. The Association says that in this country military dependents, like other civilians, should be treated by private physicians and in private hospitals unless these resources are inadequate. Then, and only then, should uniformed physicians and military facilities be employed. Also, the Association objects to the use of a special draft law to provide physicians to care for civilians within this country.

Federal Aid to Medical Education

Most prominent are identical House and Senate bills for a five-year, $250 million program of grants to schools for construction, expansion, modernization and maintenance. These bills have had hearings completed and await action until the second session. Up to two-thirds of new school construction cost would be supported by the Government, and up to one-half of existing school costs; if an existing school increased its freshmen enrollment 5 per cent over last year, it also could get up to two-thirds.

The AMA favors this measure in the belief that an emergency condition exists in medical schools. But it proposes dropping out the 5 per cent enrollment incentive and making certain that the advisory Medical Education Council include six "leading medical authorities" and specialists in finance and construction.

Mental Health Survey

This became public law and provides Federal grants totalling $1,250,000 to non-government groups for financing an extensive three-year survey of national mental health problems. The American Medical Association gave its full support to this bill.

Federal Employe Health Insurance

Most U. S. Civilian workers would be eligible for coverage. They would choose between (a) an over-all Federal plan, to be contracted for nationally by the Civil Service Commission, or (b) a local, employe or other type of plan. Major medical expense coverage also would be available. The U. S. would pay up to $1.50 per month for a single person, or up to $4 for one with dependents, or one-third of the premium, whichever is the smaller. Benefits that would have to be allowed are spelled out specifically in the bill. This bill was pending at adjournment.

Tax Deferments

Proposals for deferring taxation on money put into restricted retirement plans have been before Congress for quite some time. Generally they would allow physicians and other self-employed to pay income tax on the amounts when received as annuity payments. The American Medical Association has pressed hard for this type of legislation. Hearings were completed on this legislation, but action will await the second session of Congress in January.

Bricker Amendment

Extensive hearings have been held on the resolution regarding the treaty-making powers of the President. The American Medical Association is a strong advocate in principle of the Bricker concept. This legislation is still pending.

PHS TO HANDLE INDIAN MEDICAL CARE RESPONSIBILITY

Effective on July 1 of this year, the United States Public Health Service took over responsibility for the medical care of American Indians, a duty that up to then had been performed by the Indian Bureau in the Department of the Interior. The program affects about 350,000 Indians living on reservations. Transferred to PHS from the Bureau are 3,600 employes and 970 buildings. Fifty-six hospitals, twenty-one health centers, thirteen boarding school infirmaries and numerous other health structures are included in the transferred real estate, which is estimated to be worth about $40 million. This was reported in a recent issue of the AMA Washington Letter.

According to the Letter, "the shift in responsibility is a result of legislation passed last year. It was supported by the Department of the Interior, the Budget Bureau and many medical groups, including the American Medical Association."

Personnel Continues

To handle the new task, the Public Health Service has established a Division of Indian Health in the Bureau of Medical Services. Dr. James R. Shaw, who has handled the program for the past two years on loan from PHS, will continue to direct it as chief of the new division. Dr. Frank French and Dr. Joseph Dean will remain as his top assistants.

TWO WORLD MEDICAL GROUPS DISCUSSED

A recent article in *The Journal of the American Medical Association* discusses two organizations which work toward world health, understanding and peace—the World Medical Association and the World Health Organization. The article was written by Drs. Harold S. Diehl, Minneapolis; Leonard W. Larson, Bismarck, N. D.; and Franklin D. Murphy, Lawrence, Kansas.

The two groups are "a team that is making one of the greatest contributions of our time to the improvement of mankind—a must for the prevention of the spread of communism," the article states.

An accompanying editorial states:

> "Each country, of course, has its own problems, but there are many aspects of the control of disease and the providing of medical care that should be viewed internationally. Sometimes the view prevailing in one country is helpful when disseminated elsewhere. . . . Such value is readily apparent to everyone."

Goals Explained

The world Medical Association, a professional organization of physicians, developed an International Code of Medical Ethics, sponsored the first World Congress on Medical Education, and now is working in the field of world occupational health.

The World Health Organization, with 84 member nations, is within the general orbit of the United Nations, but administered separately. It has compiled and distributed information on many phases of health and disease; led in the control of malaria and yaws, and assisted in training thousands of persons from countries without health-training facilities.

The article states that the World Health Organization is valuable to the U. S. "both as an arm of foreign policy of our government and as a contributor to our domestic health." Both organizations have worked together, collaborating on projects and exchanging representation. "It is essential that these organizations continue to co-operate in order to assure the greatest possible competence," the article says. It concludes:

> "The individual physician can help in developing understanding within his own community of the importance of the development of publc health services in underdeveloped areas of the world as a barrier to the spread of communism."

In Memoriam

PERCIVAL HALE BENNION

Dr. Percival H. Bennion died June 30, 1955, in White Plains, New York, where he was visiting his daughter. Dr. Bennion retired from active practice in St. Paul in 1947.

Born in 1876 in Grove City, Minnesota, Dr. Bennion spent his boyhood in Litchfield. He graduated from the medical school of the University of Minnesota in 1902 and interned at St. Mary's Hospital in Minneapolis.

During his years of practice in St. Paul, Dr. Bennion was active in civic affairs. He was a member of the Midway Club, the Osman Temple of the Shrine, and on the Merriam Park library board.

Dr. Bennion was on the staff of Midway Hospital, a member of the Ramsey County Medical Society and a "Fifty-Club" member of the Minnesota State Medical Association.

He is survived by his two daughters: Mrs. Kenneth Huntzider, Miles City, Montana, and Mrs. Lester C. Tuttle, Winter Haven, Florida, and five grandchildren.

ALLOYS F. BRANTON

Dr. Alloys F. Branton, former Willmar physician and surgeon, died July 26, 1955, at the Baroness Erlanger Hospital in Chattanooga, Tennesse, where he had been administrator for nine years until his retirement a year ago because of illness.

Born in Minneapolis in 1895, Dr. Branton spent his youth in Willmar. He attended the University of Minnesota and graduated from the medical school in 1920. After interning at Minneapolis General Hospital, he affiliated with the Willmar Clinic.

Dr. Branton was president of the Central Minnesota County Medical Society and a member of the Minnesota State Medical Association. A specialist in hospital administration, he served as executive secretary and president of the Minnesota Hospital Association. He was a member of the American College of Hospital Administrators.

His many activities included a term as vice commander of the district American Legion, vice president of the Kiwanis International, and president of the Willmar Chamber of Commerce.

He is survived by his wife Helen T. Branton and two children: Alloys, Jr., of Philadelphia, and Mrs. Helen Wilhoit of Seattle.

HAROLD WILLIAM HAVEL

Dr. Harold W. Havel, former Jordan general practitioner, died July 23, 1955, in Omaha, Nebraska. He had been in practice there since 1949.

Dr. Havel was born in Montgomery, Minnesota, in 1909. He spent his boyhood in that town. He obtained his medical education at the medical school of Creighton University, Omaha, Nebraska, and interned in that city at St. Catherine's Hospital. After completing his training, he entered general practice in Jordan, Minnesota, in 1933.

While in Jordan, Dr. Havel was active in community affairs. He was president of the Jordan Commercial Club in 1939-40 and a member of the Knights of Columbus. He was president of the Scott-Carver County Medical Society in 1939-40, a member of the Minnesota State Medical Association and the American Medical Association.

He is survived by his wife and two sons, Thomas J. and Harold, Jr., of Omaha; his parents, Mr. and Mrs. T. N. Havel of Montgomery, Minnesota; two brothers, Dr. Thomas E. of Santa Monica, California, and Joseph of Glencoe; and two sisters, Mrs. John Petricka of Northfield and Mrs. Bernice Powers of Mankato.

HENRY EMIL ROKALA

Dr. Henry E. Rokala, physician of Virginia, died June 17, 1955. Dr. Rokala was born in Nashwauk, Minnesota, in 1903 and spent his youth there.

In 1924, Dr. Rokala graduated from Carleton College and for seven years taught at Shattuck Military Academy at Faribault. He then entered the University of Minnesota Medical School, graduating in 1937. He served an internship at St. Luke's Hospital in Duluth and then entered practice in Biwabik.

In 1941, Dr. Rokala established a practice in Virginia and remained there in practice until his health failed. He was a member of Phi Beta Pi medical fraternity, the St. Louis County Medical Society, the Minnesota State Medical Association, the Americal Medical Association, Virginia Lodge, Ancient Free and Accepted Masons, Royal Arch Masons, the Virginia Chamber of Commerce and a number of other civic organizations. He was a member of the First Presbyterian Church.

Surviving are his wife; two sons, Dwight and Curtis; a daughter, Winifred; and two brothers, John and Einar, both of Nashwauk.

STELLA LUCY WILKINSON

Dr. Stella L. Wilkinson, one of Minnesota's best-known women doctors, died July 22, 1955, at eighty-one years of age. She had been in poor health for two years.

Dr. Wilkinson was born in Vinton, Iowa. She attended Central High School in Saint Paul and the University of Minnesota, graduating from the medical school in 1903. She practiced in Duluth for many years, at Askov, Newport, at the Moose Lake State Hospital and the Faribault School. Two years ago, she retired from practice.

Dr. Wilkinson had been a member of the St. Louis County Medical Society, the Washington County Medical Society and the Rice County Medical Society. She was a life member of the Minnesota State Medical Association and received her "Fifty Club" certificate in 1953.

Dr. Wilkinson is survived by one sister, Mrs. Cordelia J. Crawford of Saint Paul.

Reports and Announcements

MEDICAL MEETINGS

National

Academy of Psychosomatic Medicine, 2nd annual meeting, Plaza Hotel, New York City, October 6-8, 1955.

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American College of Gastroenterology, annual convention, The Shoreland, Chicago, Illinois, October 24-26, 1955, followed by annual course in Postgraduate Gastroenterology, October 27-29, 1955.

American College of Surgeons, sectional meeting, Milwaukee, Wisconsin, February 27-29, 1956.

American Medical Writers' Association, 12th annual meeting, Hotel Jefferson, St. Louis, Missouri, September 30, 1955, followed by Workshop on Medical Writing, October 1.

Association of Military Surgeons of the United States, 62nd annual convention, Statler Hotel, Washington, D. C., November 7-9, 1955.

Endocrinology and Metabolism, 7th annual Postgraduate Assembly of the Endocrine Society, Indiana University School of Medicine, Indianapolis, Indiana, September 26-October 1, 1955.

Mediclinics of Minnesota, postgraduate course sponsored by Academy of General Practice of Broward County, Fort Lauderdale, Florida, March 5-14, 1956.

Mississippi Valley Medical Society, 20th annual meeting, Hotel Jefferson, St. Louis, Missouri, September 28-30, 1955.

National Society for Crippled Children and Adults, Palmer House, Chicago, Illinois, November 28-30, 1955.

Occupational Skin Problems, course sponsored by Institute of Industrial Health, University of Cincinnati, Kettering Laboratory, Cincinnati, Ohio, October 10-14, 1955.

Omaha Mid-West Clinical Society, twenty-third annual assembly, Hotel Paxton, Omaha, Nebraska, October 24-27, 1955 (see page xxv for program).

Venereal Disease Postgraduate Course, 24th session, sponsored by University of Chicago and Public Health Service, University of Chicago, September 26-30, 1955.

Winston-Salem Heart Symposium, Robert E. Lee Hotel, Winston-Salem, North Carolina, September 30, 1955.

State

Michigan State Medical Society, Pantlind Hotel, Grand Rapids, Michigan, September 26-30, 1955.

International

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

MINNESOTA ACADEMY OF GENERAL PRACTICE

The Minnesota Academy of General Practice will hold its Fifth Annual Fall Refresher at the Radisson Hotel, Minneapolis, on Wednesday, October 19, starting at 8:30 A.M.

Eight hours of concentrated instruction will be given by fourteen authorities, highlighted by a talk by E. H. Rynearson, M.D., Rochester, Minnesota, at the noon luncheon. Also appearing will be Bert Seligman, M.D., Toledo; Gene Stollerman, M.D., University of Chicago; and George Logan, M.D., Rochester, Minnesota.

There is no registration fee, but a five-dollar luncheon ticket is required. Registrations are advisable and should be made through James A. Blake, M.D., 15 Ninth Avenue South, Hopkins, Minnesota.

WORKSHOP IN MEDICAL WRITING

A Workshop in Medical Writing will be held on the second day of the twelfth annual meeting of the American Medical Writers' Association under the instruction of members of the journalism faculties of the University of Illinois, University of Missouri, and University of Oklahoma, Saturday, October 1, 1955, from 8:00 a.m. to 12:00 noon at Hotel Jefferson, St. Louis, Missouri.

CONTINUATION COURSE

The University of Minnesota announces a continuation course in *Fractures for General Physicians* which will be held at the Center for Continuation Study from November 21 to 23. Management of the fractures most commonly seen in general practice will be stressed. The course will include a demonstration session, and registrants will be invited to bring x-rays from their own practices for consultation with members of the course faculty. The course will be presented under the direction of Dr. Wallace H. Cole, Professor, Department of Surgery, and Director, Division of Orthopedic Surgery, University of Minnesota Medical School.

INDUSTRIAL HEALTH COURSE

A postgraduate course in *Modern Considerations and Methods in Handling the Lead Problems in Industry* will be presented during the week of November 7-11, 1955, by the Kettering Laboratory in the Department of Preventive Medicine and Industrial Health of the University of Cincinnati. Physicians and industrial hygienists who are concerned with the lead problem and who are interested in attending the course should write for an application blank to Secretary, Institute of Industrial Health, Kettering Laboratory, Eden and Bethesda Avenues, Cincinnati 19, Ohio.

AMERICAN CANCER SOCIETY

The American Cancer Society has announced that its program of clinical fellowships, which was begun in 1948, will continue through the next institutional year, July 1, 1956, to June 30, 1957, with fellowships commencing July 1.

CRIPPLED CHILDREN CLINICS

The remainder of the fall clinic schedule conducted by the Minnesota Department of Welfare, Crippled Children Services, is as follows:

City	*Date*	*Place*	*Counties*
Crookston	Oct. 8	High School	Norman, Polk
Marshall	Oct. 15	East Grade School	Lac qui Parle, Lincoln, Lyon, Redwood, Yellow Medicine
Little Falls	Oct. 22	Charles Lindberg School	Morrison, Mille Lacs, Todd
Virginia	Oct. 26, 27	City Hall, Health Dept.	St. Louis, East Range
Hibbing	Nov. 9, 10	Village Hall	St. Louis, West Range
Willmar	Nov. 19	High School	Chippewa, Kandiyohi, McLeod, Meeker, Renville, Swift
Anoka	Nov. 22	Lincoln School	Anoka, Chisago, Hennepin, Isanti, Kanabec, Ramsey, Sherburne, Washington, Wright

PULMONARY-FUNCTION TESTS

(Continued from Page 601)

in the evaluation of right-to-left shunts (pulmonary hemangioma and certain congenital cardiac defects) and occasionally contributes to the differential diagnosis of primary and secondary polycythemia.

For assessing hypercapnia, no method is both simple and entirely satisfactory. Determination of the carbon dioxide-combining power of venous blood is a test that is widely available but that in itself cannot distinguish between respiratory and metabolic disturbances. It is well to remember that elevated values for the carbon dioxide content of blood accompany both metabolic alkalosis and respiratory acidosis. The accurate definition of certain disturbances of acid-base balance occasionally requires direct chemical analysis of arterial blood.

In recent years, there has been rapid advancement of knowledge of both normal and abnormal pulmonary physiology. Much of this has been obtained with complex techniques which are currently impractical for routine clinical use and which have not been presented here. More extensive discussions and bibliographies may be found in the accompanying list of review articles.

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Woman's Auxiliary

MESSAGE FROM THE PRESIDENT

As our activities begin this fall, I bring greetings from your new officers. We pledge to you our support and it will be our pleasure to serve you in every way possible.

It was my privilege to attend the 32nd Annual Convention of the Woman's Auxiliary to the AMA, which was held in Atlantic City, June 6-10. Minnesota was well represented, with eleven delegates. Stimulating reports of officers, chairmen, and state presidents, gave evidence of the excellent work that has been accomplished throughout the country. It was a most gratifying and interesting experience.

Our aim continues to be Leadership in Community Health. The varied program of the Auxiliary makes it possible for every member to choose the field for her contribution to her community. Be a good member! Be informed!

The Fall Board Meeting and School of Instruction for all state officers, chairmen, county presidents, officers and chairmen and all members of the Auxiliary, will be held at the Curtis Hotel, Minneapolis, Tuesday, October 4. It is the new members, willing to assume leadership, who will be our new county officers and chairmen, and many will continue on to State and National offices. To meet the needs of these new officers, our School of Instruction is planned. Well-informed officers, committee chairmen and members, will produce a progressive organization.

Interesting speakers are planned, including our National Auxiliary President, Mrs. Mason G. Lawson, who will be our luncheon guest speaker. She is most delightful and will be an inspiration for us as we start our new year.

Further details will follow, but save Tuesday, October 4. I will be looking forward to seeing you. I wish you success in all your Auxiliary activities this year—1955-56.

Mrs. H. H. Fesler
President

Of General Interest

Hastings State Hospital acquired a new acting superintendent in mid-July. **Dr. Nelson J. Bradley,** superintendent of Willmar State Hospital, is now serving as acting head at Hastings in addition to continuing his duties at Willmar. Since the resignation of **Dr. Ralph Rossen** last fall, the Hastings post has been filled by superintendents and personnel from the other state hospitals.

* * *

Dr. Alf K. Stratte, Pine City, has been given the state's highest award, the Minnesota Medal of Merit. Presentation of the award was made by Governor Orville L. Freeman at Camp Ripley, where Dr. Stratte was senior surgeon during training exercises for the Minnesota National Guard.

* * *

Miss Caryl Hoyer and **Dr. Deno J. Wedes,** St. Paul, were married at Arlington Hills Lutheran Church, St. Paul, on June 18.

* * *

Miss Mary Lou Mulcahey and **Dr. Eugene C. Ott,** Minneapolis, were married at Annunciation Catholic Church, Minneapolis, on July 23.

* * *

Dr. Charles Bolsta, retired Ortonville physician, was recently presented with a printed testimonial by the Medical Alumni Association of the University of Chicago in recognition of the sixtieth anniversary of his graduation from Rush Medical School.

* * *

Dr. E. S. Palmerton, formerly of Fergus Falls, is now practicing at Rapid City, South Dakota. An ophthalmologist, Dr. Palmerton was associated with the Park Region Medical Center at Fergus Falls for the past two years.

* * *

Dr. George L. Berdez, who retired on July 1 as pathologist for Miller Memorial Hospital, Duluth, was honored by the hospital medical staff at a testimonial dinner the preceding evening. Principal speaker at the dinner was **Dr. E. T. Bell,** emeritus professor of pathology at the University of Minnesota.

Dr. Berdez has served as pathologist for more than a dozen hospitals since he arrived at Duluth in 1923. He plans to continue his service at a few hospitals while "slowing down" for another year.

* * *

Dr. and Mrs. Fred A. Figi, Rochester, left by plane for Europe on July 30. During a six-week tour of the continent Dr. Figi presented a paper at the First International Congress of Plastic Surgery in Stockholm, Sweden.

* * *

Dr. Wilford J. Deweese, Bemidji, provided more than a physician's usual services when he saved the life of a seven-year-old girl at Lake Bemidji on July 9. Marshall Deweese, aged eight, saw the girl drowning and called his father, who was working on a boat nearby. Dr. Deweese plunged into the lake, found the child on the bottom in eight feet of water and brought her ashore unconscious. Artificial respiration revived her.

* * *

Dr. and Mrs. W. G. Benjamin, Pipestone, with their two daughters spent July and August on a trip through Europe. During his absence Dr. Benjamin's practice was conducted by **Dr. Edgar Gamm** of St. Paul.

* * *

Governor Orville Freeman has appointed **Dr. Frank H. Krusen,** Rochester, as chairman of a newly formed advisory committee on vocational rehabilitation. The committee will try to help an estimated 80,000 disabled persons in Minnesota become "contributing members of society."

* * *

A biographical sketch of **Dr. Carl Barton Nelson,** Minneapolis, appeared in the Town Toppers column of the *Minneapolis Star* on July 21.

* * *

Dr. Edwin T. Maitland, Jackson, attended a reunion of all past interns of Broadlawn Hospital at Des Moines, Iowa, late in June.

* * *

Dr. Victor Johnson, director of the Mayo Foundation, Rochester, was the principal speaker at commencement exercises for the first summer session at the University of Minnesota on July 14.

* * *

Resignation of **Dr. Howard L. Horns** as assistant dean of the medical sciences and associate professor of medicine at the University of Minnesota on September 1 has been announced by **Dr. H. S. Diehl,** dean of medical sciences.

Dr. Horns, who became assistant dean in 1949, succeeding **Dr. Myron M. Weaver** in the post, is now associated with the Nicollet Clinic in Minneapolis. He recently completed a two-year tour of duty with the Army. He will continue clinical teaching in the medical school on a part-time basis.

* * *

Dr. John M. Streitz is now associated with **Dr. M. M. Fifield** in the practice of urology in Duluth. Dr. Streitz recently completed a residency in urology at the University of Minnesota Hospitals and the Veterans Hospital, Minneapolis.

* * *

An open house celebration was recently held by residents of Cyrus in honor of **Dr. Herman Linde,** who has practiced in the community for fifty years. Numerous gifts and messages of congratulations were presented to Dr. Linde.

Dr. Robert E. Priest, Minneapolis, filled several speaking engagements this summer. At a meeting of the Lyon-Lincoln County Medical Society at Marshall he spoke on "Treatment of Hoarseness." Later he discussed "Treatment of Ear, Nose and Throat Disorders by the General Practitioner" at a meeting of the Tri-County Medical Society at Litchfield and at the Range Medical Association meeting at Grand Rapids.

* * *

Principal speakers at a meeting of the Minnesota Society of Neurology and Psychiatry in St. Paul on September 13 were **Dr. Robert P. Jeub** and **Dr. Jennings C. L. Peteler**, both of Minneapolis.

* * *

Eighteen students trained as **laboratory aides** are being graduated by the General Extension Division of the University of Minnesota this month. Their training consisted of six months of classes at the university and six months of practical training in selected hospital laboratories. In the one-year course they were trained to do most routine medical laboratory tests and are best qualified to work in a physician's office or to assist a medical technologist in a hospital or clinic laboratory.

Information about students or the training program can be obtained from Verna Rausch, Assistant Professor of Medical Technology, C205 Mayo Building, University of Minnesota, Minneapolis.

* * *

At the annual convocation of the American College of Chest Physicians in Atlantic City this summer, the following Minnesota physicians were awarded fellowship certificates: **Dr. James R. Fox**, Minneapolis; **Dr. William F. Mazzitello**, St. Paul; and **Dr. John R. McDonald, Dr. R. Drew Miller**, and **Dr. Gustavus A. Peters**, all of Rochester.

* * *

Dr. Carl O. Thompson, Hendricks, has been appointed coroner by the local board of county commissioners.

* * *

Lt. Col. William F. Sheeley, chief of psychiatric services at the Air Force School of Aviation Medicine, Randolph Field, Texas, has been appointed superintendent of Hastings State Hospital. It was expected that Dr. Sheeley would take over at the hospital by the end of this month, following his discharge from service. The Hastings institution has been without a permanent superintendent since **Dr. Ralph Rossen** resigned last November. Dr. Sheeley was a resident in psychiatry at the University of Minnesota Hospitals from 1950 to 1953.

* * *

Dr. Frank D. Mann, a member of the staff of the Mayo Clinic, Rochester, since 1947, has become associated with the Palo Alto Hospital, Palo Alto, California. Dr. Mann, a son of **Dr. and Mrs. Frank C. Mann** of Rochester, is a clinical pathologist.

* * *

Principal speaker on "The Doctors Round Table," a television program on WCCO-TV on August 28, was **Dr. John P. Medelman**, St. Paul, who discussed the value of routine chest x-rays for all hospital admissions.

* * *

Dr. and Mrs. H. M. Juergens, Belle Plaine, left on August 24 for a tour of Europe. During the trip they plan to attend the World Medical Congress in Vienna, Austria, during the last week of this month.

* * *

Dr. Donald P. Fox, Minneapolis, has been commissioned a medical missionary by the Lutheran Church. With his family he sailed on September 14 for England, where he will study tropical medicine for five months before going on to Tanganyika, East Africa.

* * *

Dr. Andrew C. Erlanson, St. Paul, has been appointed a pediatrics consultant for the Minnesota Department of Health's maternal and child health section. Dr. Erlanson recently completed a three-year fellowship in pediatrics at the Mayo Clinic.

* * *

Dr. Arthur J. Henderson, St. Paul, has been appointed coroner of Ramsey County to fill out the unexpired term of Dr. Carl Ingerson. Dr. Henderson has been a deputy coroner for ten years.

* * *

Dr. K. W. Covey, Mahnomen, has announced that he will discontinue his practice about January 1 to accept a residency in orthopedics at the Mayo Clinic. Dr. Covey has practiced at Mahnomen since 1944.

* * *

Principal speaker at a meeting of the Rotary Club in Duluth recently was **Dr. Gordon J. Strewler**, Duluth, who discussed "Sidelights on Maturity."

* * *

Dr. and Mrs. H. C. Habein, Rochester, returned in mid-August from a six-week vacation in Europe. During the trip they visited their son, Dr. Peter Habein, who is stationed at an Air Force hospital in Germany.

* * *

Dr. Thomas H. Johnson, a graduate of the University of Minnesota Medical School, took over his duties in mid-August as resident physician at the state prison at Stillwater.

* * *

Dr. Corrin H. Hodgson, Rochester, left on August 22 on a special mission to Africa as medical advisor for an expedition headed by Congresswoman Frances P. Bolton. Object of the group, which will return in November, is to study economic, social and health factors for the House Committee on Foreign Affairs.

* * *

The Minnesota Safety Council has awarded a certificate of recognition of long service to **Dr. F. J. Hirschboeck**, Duluth, medical representative on the council. The award was made at the annual meeting of the council in Duluth on August 19.

* * *

Dr. William M. Feigal has been elected president

(Continued on Page 662)

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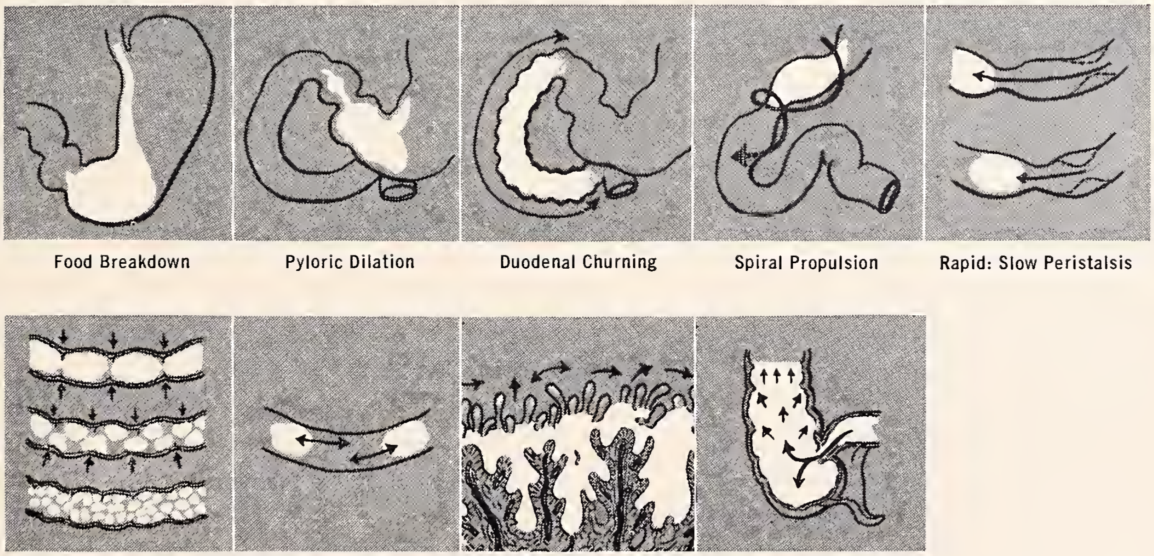

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(Continued from Page 660)

of the medical staff of Northwestern Hospital, Thief River Falls. Named as vice president was **Dr. Daniel Greene,** with **Dr. George Martin** as secretary.

* * *

Mrs. Dorothy B. Leven, wife of **Dr. N. Logan Leven,** St. Paul, died on August 6. For many years Mrs. Leven was active in social and welfare work in the Twin Cities. She was married to Dr. Leven in 1930.

* * *

Dr. and Mrs. F. H. Ellis, Jr., Rochester, left on August 22 for a five-week tour of Europe that was to include two weeks in Russia. Dr. Ellis plans to meet with Russian surgeons in Moscow and Leningrad and exchange information on surgery with them.

* * *

A check for $53,000 for heart research was given to the University of Minnesota by the Minnesota Heart Association on August 16. Accepting the check, which was the first installment of $105,644 allocated for heart research projects, was **Dr. Harold S. Diehl,** dean of medical sciences.

NEW LOCATIONS

Dr. Arnold M. Berg, formerly of Hawley, has become associated in practice with **Dr. D. O. Berge** at Roseau.

Dr. Robert Carlson, who recently completed his internship in Portland, Oregon, has joined the staff of the Davis Clinic in Wadena.

Dr. Robert C. Ahlstrom, who interned at Ancker Hospital, St. Paul, has become associated with **Dr. W. T. Nygren** in the Braham Clinic, Braham.

Dr. G. H. Purves, formerly of Hendricks, has become associated in practice with **Dr. Waldo P. Anderson** in Buffalo.

Dr. Clayton R. Green has taken over the office of **Dr. John P. Cooper** in Excelsior. Dr. Cooper is doing postgraduate work at the University of Minnesota.

Dr. Paul T. Grimes, formerly of Harrisburg, Pennsylvania, has become associated in practice with **Dr. Norman Haugan** in Park Rapids.

Dr. Ernest J. Aulick, formerly of California, has opened offices for the practice of medicine in Belgrade.

Dr. Harry F. Burich, formerly of La Crosse, Wisconsin, has joined the Olmsted Medical Group in Rochester as a general surgeon.

Dr. Charles J. Hedlund, formerly of Los Angeles, California, has become associated in practice with **Dr. F. C. Closuit** in Aitkin.

Dr. Fred Colosey, formerly on the staff of the Shipman Hospital in Ely, has moved to South Bend, Indiana, where he is now practicing.

Dr. Lloyd T. Wood, who recently completed his internship in Detroit, Michigan, has become associated in practice with **Dr. N. M. Mensheha** at Forest Lake.

(Continued on Page 664)

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*Farley, W. J.: Oxytetracycline in Pediatrics, Internat. Rec. Med. *168*:140 (March) 1955.

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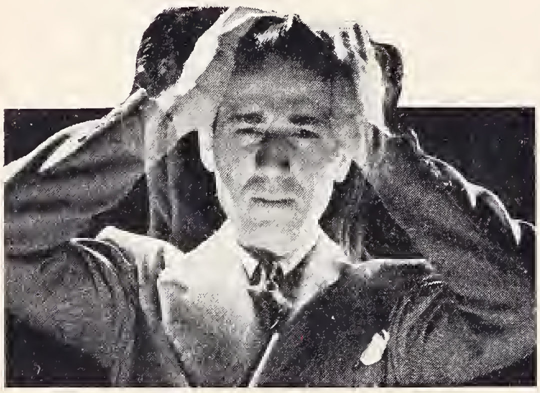

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(Continued from Page 662)

Dr. Theodore M. Gill, who recently completed a residency in ophthalmology at Minneapolis General Hospital, has become associated in practice with his father-in-law, **Dr. F. G. Folken,** in Albert Lea.

Dr. H. W. Henry, after two years in the Air Force, has returned to Hinckley and reopened his offices for the practice of medicine.

MINNESOTA BLUE SHIELD-BLUE CROSS

According to statistics just released from Minnesota Hospital Service Association (Blue Cross) for the month of June, 1955, there were 14,684 hospital cases paid in the amount of $1,678,891 for a total of 91,096.5 days of patient care.

During the first six months of 1955, Blue Cross subscribers have been provided with 521,079.3 days of hospital care. Total benefits paid for this six-month period amounted to $9,524,072.

The figures also revealed a decrease in upper respiratory cases during the first six months of 1955 over the same period in 1954. For example, during the first six months of 1954, Blue Cross benefits were paid for 6,911 tonsillectomies, while there were only 4,507 such cases during the same period in 1955.

Since 1933, 11,000,085.9 days of hospital care have been provided for Blue Cross members, representing over $120,000,000 in benefits.

Within the next two months Blue Cross and Blue Shield "Scotchlite" highway signs will be erected in two-hundred strategic locations throughout Minnesota.

These signs will prominently display the Blue Cross and Blue Shield emblems in blue scotchlite with the words Blue Cross and Blue Shield in silver scotchlite on a blue background. The slogan "You Need Both" is in red letters on a yellow background. These signs will appear as handsome daylight displays, and at night will glow brightly when caught in the beam of passing automombile headlights. They will be constant reminders to all Minnesotans of the importance of Blue Cross and Blue Shield in their daily lives. Located over the whole state, they broadcast the need of effective protection for hospital and doctor bills.

Integrated with this program of highway advertising, 23 daily, and 80 weekly newspapers throughout Minnesota, in addition to Twin City papers, will carry the Blue Cross-Blue Shield story. More widespread use of daily and weekly newspaper advertising by Minnesota Blue Cross and Blue Shield coincides with an expanded national advertising program of the Blue Cross and Blue Shield Commissions. This program is extended to include national general magazines, as well as business and press magazines. Also, radio and television stations throughout the state, in addition to those of Minneapolis, Saint Paul, and Duluth, are carrying spot advertising and announcements concerning the effectiveness of Blue Cross and Blue Shield in meeting hospital and doctor bills.

Insofar as Blue Shield, the doctor's plan, is concerned, the scotchlite highway signs are part of a program to keep before the public the sponsorship of a prepayment medical care plan by the members of the medical profession of the State of Minnesota.

Original Contributions

The County Medical Society

Its Significance and Function

A. O. SWENSON, M.D.
Duluth, Minnesota

THIS annual meeting is especially noteworthy in that we are celebrating with the Hennepin County Medical Society, its centennial. One hundred years of county medical organization in Hennepin County has brought many changes and achievements. A hundred years of service to this community with its accomplishments of increased longevity and decreased morbidity is only a faint reflection of what the next one hundred years of service will bring. Again on behalf of the Minnesota State Medical Association, let me congratulate the Hennepin County Medical Society on its one hundred years of service to this community.

It is especially fitting on an occasion such as this, that we dwell for a moment on the significance of our county medical organization. There is an old saying that "interest does not bind men together, interest separates men." There is only one thing that can effectively bind people, and that is a common devotion. This common devotion is characteristic of our profession, although often lost sight of in our busy work-a-day world.

Our detractors would have us believe that there are two personalities in medicine — the physician, and the physician's organization or medical fraternity. Every doctor knows that the whole of medicine is greater than the sum of its parts; but in certain respects, the individual doctor is much more important than the medical society itself. Therefore, let us concern ourselves with the effect of the county medical society on the individual physician.

I like to think of the county medical society as a character-building organization. When the young doctor in a community joins his local county society, he assumes certain obligations, not only to himself, but to the community of doctors in which he works, and to the public which he serves.

The period of transition from the life of the medical student to that of a practitioner of medicine, is a difficult one. He is exposed to many pressures, not the least of which is the economic necessity of making a living, and not too infrequently the necessity of repaying obligations which had to be assumed in order that he might be able to complete his prolonged course of study. The attitudes developed by the young practitioner in the early and formative months of his medical practice might well affect the rest of his life as a practitioner of medicine. Where else can the young practitioner turn but to the local county medical society for advice and guidance? It is the duty of the local medical society and its members to assist the young practitioner in every way possible, in order that his future career be shaped and guided in such a manner that it bring the most pleasure to himself and benefit to the community which he serves.

During his medical course, his internship, and/or residency, the young doctor has been so concerned with the scientific aspects of medicine that he has had little time or even opportunity to contemplate or visualize the obligations which he assumes on entering the practice of medicine. One criticism of our present medical education is that too little concern is given to teaching the code by which a doctor should live. The Hippocratic oath is supposedly too antiquated for present day purposes, and in only a few schools is this oath even read to the graduating class. The Student American Medical Association has

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Presidential Address presented at the annual meeting of the Minnesota State Medical Association, Minneapolis, May 24. 1955.

done much to remedy this defect. The student's training in this organization makes him better able to take his place in the community, working with the fellow members of his profession and taking his rightful place on the staff of the hospital in which he serves his patients.

Usually, when the young doctor is elected to membership in his local county society, he, for the first time, is exposed to the code of ethics of the American Medical Association. This code of ethics has been evolved over the years, but is based essentially upon the ancient Hippocratic oath and the principles taught by the Father of Medicine. Like the Constitution of our United States, it is a living document and hence is subject to amendments and modifications as indicated by our social and economic changes. Basically, this code of ethics is for the protection of the patient and public. But, adherence to this code of ethics serves the best interest of the physician himself.

The medical society, like any other democratic institution, does not exist for its own sake primarily, but rather as an instrument through which its members are able to do certain things they could not hope to do as individuals. James E. Bryan, in his book on public relations in medical practice, has listed the opportunities which the medical society provides the doctor, to accomplish things for the good of his profession and the public which he serves. I shall briefly list them with some modifications and additions of my own.

First, it enables him to promote and maintain official representation of his profession in all public health and welfare programs. The expert guidance of the medical profession and the development of these various programs is essential. Where else can the public turn for unselfish and critical advice as to the need and extent of the programs involved? Unfortunately, the advice and opinions of the medical society are not always accepted or utilized. We have all seen instances of confusion and misguided effort, one quite recently, when the help and guidance of the medical profession has been ignored.

Our organization aids the individual physician to work for the creation of more favorable attitudes toward the profession, through a better understanding and appreciation of the public contributions of the doctor. It enables him to guide and determine the overall development of medical practice and its relationship with other callings which would obviously be impossible if the doctors did not work as a group.

The protection of the public against exploitation by quacks and frauds and the legal status of medicine are maintained through the watchfulness of the physician's local, state and national organization.

The county society, only, can work toward the mitigation of professional jealousy. The society reduces discord and emphasizes the factors that unite the profession, and works to eliminate those factors which tend to divide it. Through its judicial council, the society can detect, re-educate, and when necessary, discipline those members who bring discredit upon the profession by unethical or abusive practices. This is the most unpleasant, but also one of the most necessary of the functions of the county medical society.

Changes in our social and economic life make it necessary to guard and promote social and economic mechanisms which will enable a patient to pay for necessary medical services regardless of his own cash resources, while avoiding governmental control of medical practice. In this connection, only the profession as a whole can interpret the principles and purposes of the medical profession to the public, thereby helping the people to appreciate the full value of the doctor's services. The protection of the individual members against commercialization or exploitation of professional services is very necessary. Many institutions and outside organizations seek to exploit the profession for their own immediate benefit.

A doctor's education is a continuation course that never ends. Only through community organization can the continuance of medical education be made available to each doctor. It keeps him abreast of the most recent developments in medical science and, by the interchange of ideas, improves his service to the public.

The county medical society tends to promote the association of the individual physician with other members of his profession, thereby stimulating his interest in medicine and the community in which he lives. If the doctor wishes to respect and obtain the respect of his fellow practitioners, he must know them and work with them for the common good.

The integration of the new member into his local county society is one of great importance, but has been sadly neglected. Our national and state organizations have proposed programs of indoctrination which are splendid, but as yet they are not fully utilized. The importance of such a program is obvious to all of you, if we are to educate and make full use of the capabilities of a new member.

This year the Minnesota State Medical Association has entertained the new members at a dinner meeting, giving them an insight into state and national medical organizations. We hope that this program will grow in magnitude and be assumed by each county medical society for local indoctrination of the new member.

Let us also consider for a moment the older member of our association. As in the bee hive, we have the workers and the drones. As in other organizations, the drone is frequently the unjust critic. The uninformed and misinformed critical member of our association is usually the one who never attends meetings, never reads his county bulletins or his state and national publications. He is the grandstand quarterback who never carries the ball and is usually never even present at the game. He speaks learnedly of "cliques who run things" and star chamber activities of the council or executive committees, and calls his county and state officers politicians—and the definition of a politician in his verbiage is an exceedingly "dirty word."

I'm sure there are instances where selfish members of our profession have held office to satisfy a thirst for power or personal aggrandizement. The motives and superficiality of such a person are soon apparent, and if the situation is allowed to continue, it is only through the indifference and indolence of the society members themselves.

The value of a dissenter in an organization is unquestioned, as he tends to make analyses more detailed and complete so that proper action can be taken. The informed and conscientious dissenter is a welcome and valuable member; may every society have at least one!

To satisfy even a majority of any organization is no mean accomplishment; to satisfy all would mean that the millennium had arrived. However, our chief contention should be over who best can serve.

How best to activate every member of our society is a problem that would tax the wisdom of a Solomon. We must, perforce, leave the solution to the intelligence and conscience of each doctor.

"Life is short and the art is long, the occasion fleeting, experience fallacious, and judgment difficult. The physician must not only be prepared to do what is right himself, but also to make the patient, the attendants, and the externals cooperate." Thus runs the first aphorism of Hippocrates, on whom history has bestowed the title of the Father of Medicine. This aphorism points out the need for medical organization. It points out the need for the individual physician to avail himself of the opportunities inherent in our organization. The chain is only as strong as its weakest link. Let us strive to make every member of our organization a good physician and a good servant of the public.

FILMS AVAILABLE THROUGH AMA MOTION PICTURE LIBRARY

A revised list of films available through the AMA motion picture library has been prepared, and copies are available upon request from the Committee on Motion Pictures and Medical Television of the American Medical Association. This catalog lists eighty-three medical films suitable for showing to medical societies, hospital staff meetings and other scientific groups. The catalog also includes thirty-six health films of interest to physicians who may be called upon to speak before lay audiences such as service organizations, parent-teachers' associations, et cetera.

Poliomyelitis Complicating Pregnancy

JAMES R. BERGQUIST, M.D. and
WILLIAM P. SADLER, M.D.
Minneapolis, Minnesota

IN THE last ten years, there has been an increased interest in poliomyelitis. It would appear that in the past it had been thought of as a disease most common in children; however, with better means of diagnosis and reporting, it is clear that the incidence in adults is greater than heretofore supposed.

TABLE I. INCIDENCE IN 1952 EPIDEMIC

Year	No. of Pt. Admitted	No. of Female Age 15-45	No. of Pregnant Females	Per Cent Abortion	Mortality Rate
1952	568	88	24	20.8	12.5%

TABLE II. TYPE OF POLIOMYELITIS BY STAGE OF PREGNANCY

No. Cases	Trimester	Spinal	Bulbar	Non-Paralytic	Bulbar-Spinal Combined
6 (25%)	1st.	2	0	4	0
6 (25%)	2nd.	3	1	2	0
12 (50%)	3rd.	8	0	0	4
24		13 (54.2%)	1 (4.2%)	6 (25%)	4 (16.7%)

Bowers and Danforth, in 1953, reviewed 586 proved cases of acute anterior poliomyelitis occurring during pregnancy and were able to add twenty-four new cases.

Hunter and Millikan, in August 1954, reported on forty-nine cases gathered from 1944 to 1952 at St. Mary's Hospital, Rochester, Minnesota. Baker and Baker, in 1949, reported on thirty cases from the Minneapolis General Hospital. It is the purpose of this paper to report an additional twenty-four cases admitted to Minneapolis General Hospital during the 1952 epidemic and to discuss the obstetrical management and follow-up of these patients.

During the 1952 poliomyelitis epidemic, there were 568 patients (Table I) with acute anterior poliomyelitis admitted to the Contagion Division of the Minneapolis General Hospital between July 3, 1952 and November 19, 1952. Of this group, eighty-eight of the patients were females between the ages of fifteen and forty-five years. Twenty-four (27.3 per cent) were pregnant. This figure is slightly higher than the 21 per cent reported by Anderson in the 1946 epidemic on a state-wide survey, but about the same as reported by Baker (26.2 per cent) in the same year. This can be accounted for, since Minneapolis General Hospital is a referral center and one of the few hospitals equipped to care for the acute poliomyelitic patient in the Minneapolis area. It has been estimated that between 6.2 per cent and 11 per cent of the female population in the child-bearing age will be pregnant at any one time. Thus, it is noted that this figure (27.3 per cent) agrees with the statement that poliomyelitis is about three times as common in the pregnant woman as stated by Priddle, Anderson, Baker and Baker, and Taylor and Simmons. Anderson has proved this fact by three statistically sound methods in his very fine analysis of the 1946 epidemic in Minnesota. He also found that the exposure rate was higher in the nonpregnant woman than in the pregnant.

The ages of these women varied between nineteen and thirty-one years. Stages of gestation varied between eight and forty weeks.

From the Department of Obstetrics and Gynecology, Minneapolis General Hospital, Minneapolis, Minnesota.

TABLE III. SEVERITY OF POLIOMYELITIS BY STAGE OF PREGNANCY

No. Cases	Trimester	Respirator	Mild	Severe	Deaths
6 (25%)	1st.	0	1	1	0
6 (25%)	2nd.	0	2	2	0
12 (50%)	3rd.	4	4	5	3
24		4 (16.7%)	7 (31.4%)	8 (33.3%)	3 (12.5%)

Bower and Danforth, in their review of the literature, found that the susceptibility of poliomyelitis did not vary with the stage of pregnancy. This has been documented by Anderson, Horn, and Hunter and Millikan. In the present study (Table II), it was found that 25 per cent of the patients were in the first trimester, 25 per cent in the second, and 50 per cent in the third. Thus, it would appear that the patient in the third trimester was more likely to be affected than those in the first or second. This must be evaluated in light of the over-all picture.

There were three maternal deaths, giving a maternal mortality of 12.5 per cent. All of these patients had bulbar spinal-type poliomyelitis, and all were in the third trimester. Anderson found the fatality rate in nonpregnant cases to be 14 per cent, as against 11 per cent in the pregnant group. Hunter and Millikan found the mortality to be slightly lower in the pregnant group.

An increase in abortion rate might be expected, as in other severe febrile infections. Five (20.8 per cent) of the women in this group aborted. The length of gestation varied between eight and nineteen weeks. Of this group, three had nonparalytic, one bulbar, and one spinal paralytic poliomyelitis. This figure falls between 23 per cent stated by Taussig and 18 per cent given by Allan.

There is general disagreement in the literature as to the relationship of length of gestation and the severity of the poliomyelitis. Anderson found that the incidence, as well as severity, may be greater during the first half of the pregnancy. Priddle et al found the most severe cases of poliomyelitis in pregnancy occurred in the first six months of gestation. Nearly all of the earlier reviews [4,7-9,13] have shown a greater incidence of residual paralysis and increased fatality rate in the last trimester. In the present study (Table III), six (25 per cent) cases occurred in the first trimester; of these four were nonparalytic, two spinal paralytic, and no bulbar. Only one patient in this group had severe residual, and one mild. The nonparalytic cases are those showing no weakness or paralysis at any time; mild are those who display some weakness or paralysis during the acute stage but have no disabling residual; and severe are those who have disabling involvement. Of the six cases occurring in the second trimester, two were nonparalytic, three spinal paralytic, and one bulbar. In this group, two had severe residual and two mild residual. There were twelve cases recorded in the last trimester; eight were spinal paralytic and four of the bulbar spinal type. Of this group, ten patients (83 per cent) have either died or have severe residual. There were four respirator cases in the entire group, all of which were in the last trimester. These cases are summarized below. Bowers and Danforth state that more than one-half of the maternal deaths occurred in the last trimester and in the puerperium. Cobb found that the mortality rate for poliomyelitis during the third trimester was greater than in the first two trimesters. Thus, in the present study, the most severe cases occurred in the last trimester. All patients with residual have reported some improvement in muscle function. Severe residual resulted in 33.3 per cent of the cases, and mild residual in 31.4 per cent. This represents a variance from the recent studies above; however, it may be one of the many vagaries of this disease or when considered as part of the whole picture may be of little significance.

Our findings (Table IV) agree with those previously published in that there was no statistical correlation between sex of the infant and outcome of the disease.[2,15] There was a slight tendency for those infected late in pregnancy to be the mothers carrying a female fetus. Aycock reported there was a tendency for the disease to occur in the first trimester of pregnancy, if the fetus was a male, and in the third trimester, if it was female.

TABLE IV. SEVERITY OF INFECTION BY SEX OF FETUS

	Male		Female		Undetermined		Total	
Bulbar	0		0		1		1	
Died		0		0		0		0
Severe		0		0		0		0
Mild		0		0		1		1
Paralytic	4		8		1		13	
Died		0		0		0		0
Severe		4		3		1		8
Mild		0		5		0		5
Nonparalytic	3		1		2		6	
Bulbar spinal	3		1		0		4	
Died		2		1		0		3
Severe		0		0		0		0
Mild		1		0		0		1
Total	10		10		4		24	

Management

The obstetrical management of the patient whose pregnancy is complicated by poliomyelitis, in the main, should be that afforded the normal pregnant woman.

The greatest danger to life in spinal paralytic poliomyelitis is involvement of the respiratory muscles, the diaphragm, and intercostal muscles.

In the bulbar form, the involvement of the soft palate gives rise to nasal regurgitation of fluid, absent gag reflex, and voice changes, and pharyngeal weakness is displayed by the pooling of secretions in the posterior pharynx. The above, together with involvement of the respiratory and vasomotor centers, present the greatest hazard to life. It is the patient so involved that is placed in the respirator. Drawing from the experience gained in the 1946 epidemic and that early in the present epidemic, it was found that the respirator patient could be handled much more adequately if tracheotomy was done when the patient was placed in the respirator. Oxygen was administered and the patient was bronchoscoped as necessary through the tracheotomy tube. Patients in the respirator were delivered by inserting an endotracheal tube into the tracheotomy opening and the patient's respirations assisted by manual compression of the oxygen bag. Thus, the obstetrician was able to deliver and repair the patient without hurry.

It was found that the pain threshold of the patients with severe involvement was high. A few patients were found to have advanced dilatation when first examined. The anethesia of choice was pudendal block with 1 per cent procaine. No analgesic, amnesic or sedative drugs were allowed to avoid any further stress on the respiratory center.

The majority of the patients delivered spontaneously. In order to shorten second stage, outlet forceps were applied after complete dilatation if delivery was not immediate. No effect was noted on the strength of uterine contractions. It was felt that labors were definitely less painful and shorter than in normal pregnant women.

Some have suggested that the tumor of pregnancy in the last trimester imposes a burden on the patient's respirations and recommend cesarean section irrespective of prematurity.

Cobb, citing Plass and Oberst's work on respiratory physiology, points out the fallacy of this view. He states:

> "During normal pregnancy there is a progressive increase in vital capacity, respiratory rate, volume of tidal air and minute volume of respired air. The diminished height of the pleural cavities is compensated by increased width. Normally, there is no embarrassment of respiration caused by, or related to pregnancy. Respiratory embarrassment in the pregnant woman with poliomyelitis stems directly from muscular paralysis resulting from the disease, and not the pregnancy. There are no acceptable data or other evidence to suggest that proper ventilation of patients in respirators is affected by a pregnant uterus near term."

Our Minneapolis General Hospital experience is in accord with Cobb's statement.

One may ask: What damage has been done to the infant by oxygen deprivation? How sound mentally and physically will it be? We have one infant delivered vaginally of a mother (Case 4) who appeared terminal. She had marked cyanosis, a high fever and pulse, and suffered many convulsive seizures, but survived. Follow-up on this infant shows it to be normal as of this date.

Report of Cases

Case 1.—A twenty-three-old woman, gravida 1, para 0, was admitted in September, 1952, in the thirty-fifth week of gestation with generalized muscle aching, malaise, headache, chills, fever, nausea, frequent gagging, and sweating. The temperature was 102.8°F per rectum. Examination of cerebrospinal fluid disclosed 65 cells, of which 29 per cent were lymphocytes and 36 per cent were polymorphonuclear leukocytes per cubic millimeter. Physical examination revealed a pulse of 116 per minute, and respiratory rate of 28 per minute. The patient's voice was nasal in quality and there was pooling of secretions. The heart was enlarged, and a loud pulmonic second sound with grade 3 systolic apical murmur and rumbling diastolic murmur over the mitral were heard. A diagnosis of mitral stenosis and insufficiency, compensated, and bulbar spinal poliomyelitis was made. The lower intercostal muscles appeared to be functionless bilaterally. There was bilateral hamstring weakness with weakness bilaterally in the quadriceps and adductors.

The patient's course was one of progressive deterioration, and on the third hospital day, a tracheotomy was done and the patient placed in a respirator. She seemed to improve slightly shortly thereafter. The patient was still unable to handle the secretions, and the bronchoscope was used repeatedly. A gastric tube was passed to relieve gastric dilation. The temperature rose to 106°F and pulse to 240 per minute. Digitalis was administered, and pulse rate dropped to 108. The patient became mentally confused, had seizures and cyanosis, and died four days after admission. A postmortem cesarean section was done, and a stillborn, female infant, weighing 3050 grams, was delivered. Permission for necropsy was not granted on either patient or infant.

Case 2.—A twenty-eight-year-old woman, gravida 11, para 1, was admitted in September, 1952, in the thirty-fifth week of gestation. The symptoms of backache and headache had occurred six days prior to admission. She had been admitted to one of the private hospitals because of nausea, vomiting, and urinary retention. At that time weakness in the lower extremities had been noted and examination of the cerebrospinal fluid revealed 100 cells, 98 per cent of which were lymphocytes and 2 per cent polymorphonuclear leukocytes. The evening prior to admission the respiratory rate was 40 per minute and patient was using accessory muscles for respiration. She was then transferred to Minneapolis General Hospital because of inadequate facilities. Physical examination on admission revealed a respiratory rate of 40 per minute with completely inactive intercostal muscles, bilateral foot drop, and generalized weakness of all muscles of upper and lower extremities. The patient was placed in a respirator upon arrival. She appeared to adjust promptly with marked improvement in color. She appeared to be doing fairly well the first two days of her hospital stay; however, on the third day, her pulse rose to 290 per minute. Administration of digitalis resulted in a drop in pulse to 160 per minute. The temperature rose to 103°F, and the following day the patient became disoriented and finally semicomatose. On her sixth and last hospital day, it was felt a tracheotomy was indicated; however, an attempt to obtain a chest x-ray was made. During this procedure, the patient became cyanotic and was immediately put back into the respirator. The patient died one hour later. A postmortem cesarean section was done, and a living male infant weighing 3000 grams was delivered. The heart beat was heard for approximately five minutes, but the infant made no attempt at spontaneous respiration. The infant was covered with a fine vesicular rash, and the maternal surface of the placenta was covered with many small fresh hemorrhages. Permission for necropsy on the infant was not granted. Postmortem examination of the patient disclosed acute bulbar spinal poliomyelitis.

Case 3.—A twenty-eight-year-old woman, gravida III, para II, was admitted in October, 1952, in the thirtieth week of gestation. Prodromal symptoms had been noted five days prior to admission. On admission, the lower intercostal muscles were not functioning; there was weakness of lower extremities in both abductors and adductors. The cerebrospinal fluid contained 650 cells of which 92 per cent were polymorphonuclear leukocytes and 8 per cent lymphocytes. Respiratory effort increased during the next few days. A rocking bed was tried with little help. On the fourth hospital day, a tracheotomy was done and the patient was placed in a respirator. The patient remained stable for the next two days; however, on the twelfth hospital day, she became unresponsive and cyanotic. During the next two days, the patient required repeated bronchoscopy. Her condition was such during the last forty-eight hours that the house staff on obstetrics was repeatedly alerted for the possibility of postmortem section. The pulse increased and blood pressure dropped steadily. The patient expired on her fourteenth hospital day. A postmortem cesarean section was done, and a stillborn male infant weighing 2050 grams was delivered. Necropsy was not permitted on the infant; however, necropsy on the patient confirmed the diagnosis of acute bulbar spinal poliomyelitis.

Case 4.—A twenty-six-year-old woman, gravida II, para I, was admitted August, 1952, in the thirty-second week of gestation. On admission, back and hamstring stiffness was noted. The cerebrospinal fluid contained 180 cells, of which 26 per cent were polymorphonuclear leukocytes and 74 per cent were lymphocytes. On the third hospital day, palatine paralysis was noted together with marked increase in respiratory effort. Tracheotomy was done, and the patient was placed in a respirator. The patient noted mild abdominal cramps on the third hospital day. Examination revealed the head at Station -3 with no cervical dilation. The patient now had severe involvement of the lower extremities. On the seventeenth hospital day, the patient had seizures of the grand mal type about every twenty minutes and became nonresponsive. Uterine contractions were noted every two minutes, of twenty seconds' duration. The cervix was 4 cm. dilated, head at Station 0. The contractions increased in duration and force. Membranes were ruptured artificially six and one-half hours after onset of labor. The fetal heart rate was 126 per minute in the right lower quadrant, the cervix 8 cm. dilated, and head at station plus 1. The patient was removed from the respirator. An endotracheal tube was inserted into the tracheotomy tube, and the patient's respirations assisted by manual compression of the bag. A living premature male infant was delivered as an occiput posterior presentation after rotation from a right occiput transverse position. The infant weighed 2239 grams and breathed and cried spontaneously. Bleeding during the third stage was approximately 150 cc. The patient was removed from the respirator and placed on a rocking bed three days after delivery.

TABLE V. FETAL SALVAGE

	Abortion	Term L & W	Neonatal Death	Stillborn	Preme L & W	Living	Dead
First Trimester	3	0	2	1	0	0	6
Second Trimester	2	4	0	0	0	4	2
Third Trimester	0	7	1	3	1	8	4
Total	5	11	3	4	1	12	12

The patient was discharged later on, on the one hundred and twenty-seventh hospital day, with complete paralysis of both lower extremities.

Fetal Outcome

The outcome of the pregnancies (Table V) is of interest in that the fetal loss in this study was 50 per cent. Hunter and Millikan had a fetal loss of 32 per cent; Bowers and Danforth reported 33.3 per cent.

There were eleven (45.6 per cent) living term infants delivered. These were term size, the largest weighing 3798 grams and the smallest, a twin, weighing 2367 grams. Bowers and Danforth reported that the majority of the term infants in their series were smaller and developed more slowly than the average. Follow-up studies in the present series revealed normal growth and development. There is only one other infant living and well to date; this was a thirty-four-week premature infant weighing 2239 grams.

Of the remaining twelve pregnancies (Table VI), five ended in abortion and two with stillborn premature infants. Three postmortem hysterotomies were done; two term infants, whose birth weights were 3000 grams and 3050 grams, and a premature infant weighing 2050 grams were delivered. One term infant lived for five minutes. The other two infants were stillborn.

One cesarean section was done at thirty-one weeks because of essential hypertension with superimposed severe pre-eclampsia. The infant weighed 850 grams and died after eight hours. Necropsy disclosed atresia of the ileum and pneumonia. This infant and a term infant with talipes calcaneus and congenital dislocation of the hips represent the only congenital defects found in this group. This supports the view that there is no increase in congenital abnormalities in infants born to women with acute poliomyelitis.

TABLE VI. FETAL LOSS TWENTY-FOUR PREGNANCIES (TWENTY-FIVE INFANTS) COMPLICATED BY POLIOMYELITIS

Cause of Fetal Loss	Number
Abortions	5
Pre-eclampsia with essential hypertension (neonatal 31 wks) (Necropsy—atresia of ileum)	1
Bulbar Spinal Poliomyelitis (Post mortem cesarean section—2 stillborn; 1 lived 5 minutes)	3
Spinal Paralytic Poliomyelitis	
Stillborn 28 and 30 wks' gestation	2
Premature rupture of membranes 31 wks' gestation	1
Total	12 (50%)

The last infant in this group was delivered at thirty-one weeks after premature rupture of the membranes. It weighed 1512 grams and died fourteen hours later with atelectasis.

This represents a fetal salvage of only 50 per cent, far lower than previously reported.

There have been 10 subsequent pregnancies with nine living term infants in the twenty-one remaining patients. One patient, in the severe group, aborted at two months but is at present in the third month of gestation. Two other patients were in the severe group, four patients in the mild group, and three patients in the nonparalytic group. All labors were relatively short, the longest being eleven hours and the shortest twenty minutes, with the average being 5.7 hours. One repeat cesarean section was done for a generally contracted pelvis.

Thirteen women reported no change in menstrual pattern; the remainder showed no marked variation.

Discussion

Twenty-four additional cases of poliomyelitis complicating pregnancy are presented.

The early use of tracheotomy in respirator cases, together with judicious use of bronchoscopy through the tracheotomy tube, was perhaps the most important advance in the care of the patient in this series.

Early in the epidemic, it was felt that the use of an endotracheal tube, placed into the tracheotomy and thus artificial respiration carried out, might aid in the maintenance of the patient outside the respirator during delivery. This was done and found to be very satisfactory.

Postmortem hysterotomy is not the answer to fetal salvage. Time must await the evolution of some other manner of delivery.

In this series, the severity of the residual and mortality was greater in the last trimester than in the first two. This agrees with the earlier reports. This once again points out the varied behavior of this disease.

The increase in attack rate in the pregnant woman, as against the nonpregnant, points out the fact that some other factor, perhaps hormonal, plays an important role in the susceptibility to poliomyelitis.

In a personal communication, Dr. Gaylord Anderson states that the attack rate of bulbar poliomyelitis during epidemics is in a ratio of 1:10 in persons without tonsillectomy, as compared to 1:3 in those who have had tonsillectomy.

Perhaps because of relaxed perineal muscles and high pain threshold, the labor and delivery of patients with poliomyelitis is somewhat easier.

Conclusions

1. Incidence of poliomyelitis in adults is apparently increasing.

2. The exposure rate is higher in the nonpregnant woman, but the attack rate is apparently higher in the pregnant woman.

3. Mortality rate is not any greater in the pregnant woman with poliomyelitis than in the nonpregnant.

4. In this study, the mortality rate and severity of residual was greatest during the third trimester, thus showing the varied picture this disease can present.

5. The abortion rate was no higher than expected for the general population.

6. Labors were shorter and less painful in patients who have had poliomyelitis.

7. The labor was conducted according to accepted standards.

8. Labor and delivery of subsequent pregnancies appears to be easier than in the normal pregnant woman.

9. Prompt tracheotomy, endotracheal suction, bronchoscopy, and positive pressure administration of oxygen greatly facilitated delivery of the respirator cases.

10. Analgesic, amnesic, and sedative drugs are interdicted.

11. Pudendal block with procaine was adequate anesthesia for most cases.

12. Postmortem hysterotomy was unsuccessful to effect fetal salvage.

13. Growth and development in surviving infants were normal.

14. Fertility is apparently not affected by poliomyelitis.

15. There is no change in the menstrual pattern in patients who have had poliomyelitis.

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Enigmas in Lymphoma

Reticulum Cell Sarcoma and Mycosis Fungoides

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PERTINENT to this title is Alice's remark to the Mad Hatter, "I think you might do something better with the time than wasting it in making riddles that have no answers." There exists no serious conviction that another analysis of the obscurities of malignant lymphoma will convert a single adherent from any of the existing viewpoints. Nonetheless, a critical search into the fogged realm of meaning as it pertains to "reticulum cell sarcoma" and "mycosis fungoides" may provide an opportunity for reappraisal of the impasse to which we have been led.

TABLE 1. NOMENCLATURE NOTED IN LITERATURE

Reticulum Cell

- Reticular cell
- Reticulocyte
- Reticulum
- Syncytial reticulum
- Reticuloendothelial cell
- Common lymphoid stem cell
- Lymphoidocyte
- Primitive mesenchyme
- Mesenchymal cell
- Primitive blood cell
- Stem cell
- Hemohistioblast
- Hemocytoblast
- Hemoblast

Reticulum Cell Sarcoma

- Reticular cell sarcoma
- Reticulum cell lymphosarcoma
- Reticular celled lymphosarcoma
- Large cell type lymphosarcoma
- Large round cell sarcoma
- Reticulocytoma
- Reticuloma
- Reticulosarcoma
  - (Diffuse syncytial, trabecular, syncytial, dictosyncytial, polymorphic)
- Reticuloendothelial sarcoma
- Reticuloendothelioblastoma
- Retothelsarcoma
- Endothelial sarcoma
- Hodgkin's sarcoma
- Stem cell lymphoma

It is a curious characteristic of man that he presumes a universality of understanding of words and names among his fellows. Indeed, this quality is often carried to the point of presumption that a difference in interpretation reflects an impaired intelligence. It is hazardous to exploit discord, but this study represents an effort to dilute embarrassment by sharing it more widely.

Reticulum Cell Sarcoma

Insular intolerance with respect to the "reticulum cell" is much in point. There are three common forms of response to its usage: (1) a silent assumption that the term is meaningful, (2) the requirement of acceptance of rigid criteria for identification or (3) acrimonious debate. It is proposed to outline the recorded facts and abstractions that have found their way into our literature and beliefs. Certain definitions of terms would seem essential.

Dictionaries mirror current opinion, although they rarely control it. It is of interest, in passing, to observe the definitions provided in these media for the term "reticulum." This is variously described as an interstitial tissue composed of anastomosing branched cells; as an intricate network of extracellular fibrils; and, as a fibrillar network within the cytoplasm of cells. The terms "reticulum" and "reticulin" are offered as identical, related or dissimilar entities. The "reticulum cell" is related to the

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"reticular cell" which in turn is identified with reticular tissue, said to form the framework of lymph nodes, spleen and bone marrow. The variations of synonyms devised have been legion. (Table I).

TABLE II. RETICULUM CELL SARCOMA

Histologic Characteristics as Noted in Literature*

Size of Reticulum Cell

Large	9
Small	8
12-15 μ	3
15-22 μ	3
22-50 μ	4

Giant Cells

Frequent	5
Rare	4
Absent	3
Resemble Reed cells	5
Do not resemble Reed cells	4

Configuration of Reticulum Cell

Syncytial	20
Pseudopodal	10
Ill defined	7
Polygonal	4
Stellate	3
Round or oval	6
Fusiform	3
Varied combinations	14

Cytoplasm of Reticulum Cell

Abundant	14
Scant	13
Clear	6
Granular	5
Basophilic	6
Eosinophilic	14
Neutrophilic or amphophilic	8
Phagocytic	9
Nonphagocytic	5

Nuclear Characteristics

Large	6	Round or oval	14	Vesicular	27
Small	5	Reniform	9	Hyperchromatic	5
Varied	4	Lobulated, irregular	9	Pale or even staining	7
2 - 7 μ	3	Mitosis, frequent	10	Membrane distinct	10
7 - 10 μ	3	Mitosis, rare	3	Membrane thick	6
10 - 12 μ	2	Mitosis, absent	3	Membrane thin	3
				Membrane folded	2

Nucleolus

Prominent	20
Not prominent	9
Absent	4

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*The numbers indicate the number of references agreeing with each category.

However, these are dry, didactic matters and etymology has a small place in biologic progress. It is well to proceed to the definitive contributions of cytologists.

In this dynamic era, the moving force in pathology is the recognition of functional characteristics. It appears that there are six widely held views of the nature of the reticulum cell. These are:

1. A phagocytic cell and/or its precursor.
2. A primitive element with totipotential blood-forming qualities.
3. A primitive element capable of forming blood cells or mesenchymal stroma.
4. A fibril or stroma-producing element related to the fibroblast.
5. An endotheliumlike cell lining vascular spaces.
6. A cell combining two or more of the above qualities.

One should rightfully hesitate to poll any enlightened group as to its consensus in this situation. It is obvious that a variance of this order is prophetic of the confused state which may be anticipated. A note of frustration is encountered whenever efforts are made to establish conformity of criteria in the diagnosis of reticulum cell sarcoma. It is patently impossible for all of these functions to represent the potentialities of any one cell or system of cells, unless it be the mesodermal anlage, in which case it would be necessary to add certain other capacities. Such an effort would be purposeless with available knowledge, for as Haining and co-workers[13] have said, "—the ancestry of cells, like the ancestry of human beings, is often easy and pleasing to claim, but hard to verify."

Whatever the opinion of the nature of the reticulum cell, there is some uniformity of thought that reticulum cell sarcoma is the product of the neoplastic overgrowth of this element. Table II, tabulating the cytologic criteria which have the dignity and authority of publication in reputable medical journals, reflects the existing discord. The number of citations listed in relation to each observation enumerated is varied; the authors often omit note of salient characteristics, presumably with the blithe implication that the reticulum cell is well known to all. The lack of uniformity of opinion may be appreciated by reference to Figure 1, which represents an effort to reconstruct the form of the reticulum cell from the descriptions encountered in the literature. The wide disparity in cytoplasmic and nuclear features manifested may be attributed in some part to variations in histologic techniques employed but this may, by no means, be taken as the total explanation. One can only conclude that the appellation "reticulum cell sarcoma" is loosely used and, as Gorer[12] has indicated, is usually applied to tumors comprised of "any large, incompletely mature cell not otherwise assignable."

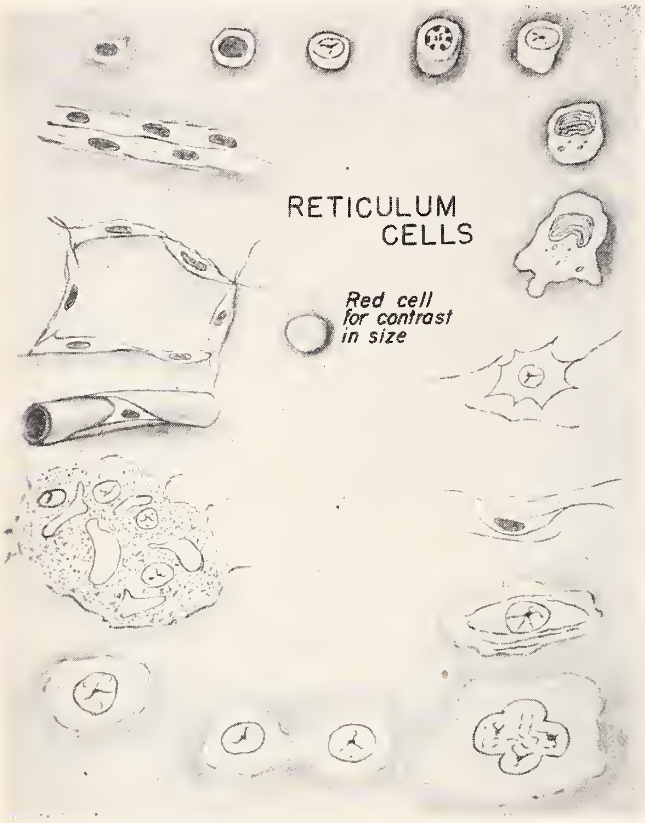


Fig. 1. Sketches representing an effort to reconstruct the appearance of the "reticulum cell" from the various descriptions available in the literature.

Moreover, the issues do not cease at the confines of the reticulum cell itself. There is little consistency of viewpoint in the relationship of the cell to the "reticulum" or whatever one may wish to name this argyrophilic fibrillar substance. Table III summarizes some of the observations which have been encountered.

TABLE III. RETICULUM IN RETICULUM CELL SARCOMA AS NOTED IN LITERATURE*

Reticulum noted	32
Reticulum not noted	22
About single cells	8
About vessels	4
Within cells	6
Cells apposed to	8
Cells connected by	3
Sheathed by syncytium	3

*The numbers indicate the number of references agreeing with each category.

Insistence upon the presence of the fibrils as a pathognomonic requirement is countered by the contentions that they represent residua of pre-existing stroma or the result of non-specific tissue reaction to neoplastic insult. Even among those who recognize a relationship to the tumor cell, there is no harmony as to location or distribution.

Obviously, there is no incontrovertible solution. To debate the nature or derivation of a cell which has so many different intellectual implications and which, in its tumor phase, has never been pontifically designated must lead to defeat. Perhaps a momentary pause for reflection would be judicious. It may serve a purpose to proceed to the other horn of the self-imposed dilemma.

Mycosis Fungoides

As one peruses the proceedings of dermatologic societies, it appears that the concept of mycosis fungoides has attained at least a superficial level of accord. All but a few dissidents appear to concur in the proposition that mycosis exists as a clinical entity. Described almost a century and a half ago,[1] there is general acceptance of the syndrome of a chronic cutaneous disorder proceeding through three characteristic phases. It is conceded that certain forms of disseminated, pruritic dermatitis may continue persistently or intermittently for long periods of years, ultimately exhibiting an infiltrating or plaque-forming phase and terminating in what has been designated a tumor or fungoid state. The inevitability of these transitions has been challenged, but the over-all characteristics have broad acceptance. There are those who recognize a *forme fruste* classified as the "d'emblee variety" in which the earlier two phases are omitted[2] but others consider this lesion to be unrelated to mycosis fungoides.[4]

TABLE IV. CONDITIONS DESCRIBED IN LITERATURE DURING PREMYCOTIC STATES

Dermatitis, allergic
Dermatitis, contact
Dermatitis, exfoliative
Dermatitis, herpetiform
Dermatitis, neuro
Dermatitis, seborrheic
Dermatitis, venenata
Eczema
Erythema multiforme
Erythroderma
Iododerma
Lichen planus
Parapsoriasis
Pemphigus
Pityriasis rubra pilaris
Poikiloderma atrophicans vasculare
Pompholyx
Prurigo mitis
Psoriasis
Ringworm
Rupioid syphilis
Urticaria

TABLE V. CUTANEOUS LESION OF MYCOSIS FUNGOIDES NOTED IN LITERATURE

(Fungoid Stage—129 Cases)

Hodgkin's	12
Hodgkin's-like	14
Hodgkin's sarcoma	3
Reticulum cell sarcoma	14
Lymphosarcoma	18
Stem cell lymphoma	2
Lymphoblastic lymphoma	5
Lymphocytic lymphoma	7
Lymphatic leukemia	14
Myeloid leukemia	2
Monocytic leukemia	1
Plasmacytoma	3
Reticuloendotheliosis	3
Lymphoma	1
Nonlymphomatous	28
Unknown	2

Despite the whispers of conflict in these general matters, an air of concord exists. Unfortunately, as an analysis of hypotheses proceeds beyond this point, dissonance intrudes itself. Fraser[8] has listed nine notions relating to the pathogenesis of mycosis; these may be modified and condensed in mildly controversial modern parlance as follows:

1. A granulomatous inflammatory disorder of the skin which (a) remains of this order throughout its course, (b) is transformed into a lymphomalike lesion or (c) is transformed into frankly evident lymphoma. In relation to the two latter eventualities, there is dispute as to whether these occur occasionally, usually or invariably.

2. A malignant lymphoma of the skin which (a) remains restricted to the skin or (b) is distributed in skin, lymph nodes and viscera.

3. A cutaneous manifestation which is secondary to an underlying systemic lymphoma.

Nor are the differences limited to general questions of pathogenesis. They also appear in regard to the specificity of the microscopic lesion during the premycotic state, the inevitability of the fatal chain of events once the early stages of the ailment have been initiated, and, as has been remarked, the identity of the d'emblee phenomenon with mycosis fungoides.

There are few who now hold to the original hypothesis of Alibert that mycosis fungoides is an infectious disorder. More generally accepted, currently, is the belief that the prolonged inflamatory process ultimately becomes transformed into malignant lymphoma or a lymphomalike lesion of the skin. Rigidity of requirements by some authorities would eliminate those cases from the category of mycosis in which the lymphomatous lesions do not supervene, but there are others who would discard the cases in which it does become manifest. Moreover, there are those who maintain that the clinical features are sufficiently characteristic that mycosis may be said to exist whatever the nature of the histologic changes.

On the other hand, the claim that meticulous study of biopsy material procured during the early premycotic state will provide cytologic evidence presaging the florid condition to come is belied by the frequency with which seemingly banal inflammatory lesions have antedated the fungoid stage. Table IV lists the many conditions which have been recorded during the premycotic phase. Obviously, since these represent the observations of many competent clinicians and pathologists, the thesis of inevitability of the tumor state must be a myth. It is remarked by Sutton and Sutton[25] that the earlier lesions "are as capricious in their course as they are variable in their aspect." In fact, Highman[14] has stated, "Mycosis is too often predicted in the presence of plain psoriasis or parapsoriasis and many a Delphian dermatologist must swallow his oracular words, as the doomed patient continues to live happily with his eruption, never to get mycosis." One may, of course, extend refutation of this theme to the ridiculous by requiring that no individual with a suspect chronic dermatitis be given a clean bill with respect to mycosis fungoides unless he has lived his full allotment of years and then has had a complete necropsy examination, the findings of which have had the concurrence of an unimpeachable jury of dermatologists, hematologists, pathologists and logicians.

TABLE VI. EXTENT OF INVOLVEMENT IN MYCOSIS FUNGOIDES
(Eighty-eight Necropsies Noted in Literature)

Author	*Cases*	*Skin only*	*Skin and viscera*
Allen[2]	21	18	3
Berman[3]	18	2	16
Cawley et al[5]	10	2	8
Forbus[6]	1	1	0
Fraser[7-10]	5	1	4
Gates[11]	15	1	14
Keim[15,16]	8	0	8
Lane and Greewood[17]	1	0	1
Miller[18]	1	0	1
Oliver[19]	1	0	1
Ormsby and Finnerud[20]	1	1	0
Pardee and Zeit[21]	1	0	1
Post and Lincoln[22]	2	1	1
Sigel[23]	1	1	0
Stratton[24]	1	0	1
Wertheim and Smith[26]	1	0	1
Total	88	28	60

Whatever the prophetic quality of the premycotic lesion, there is little evidence to contradict the proposition that in its infiltrative and fungoid stages the lesion commonly assumes a structure closely akin to that of malignant lymphoma. As indicated in Table V this, however, is by no means always the case. In the collection of 129 cases listed, in thirty (perhaps thirty-three) instances the florid lesion attained was not identified as lymphoma. It would appear, therefore, that a significant number of patients with mycosis fungoides in its terminal nodular phase, albeit a minority, fail to show evidence of neoplastic metamorphosis. If one were to add to these cases those equivocally classified as "Hodgkin's-like" and as "reticuloendotheliosis" the nonneoplastic group might be considered even more impressive.

The matter of distribution of lesions in the ultimate infiltrative and nodular phases, whether or not the alterations are neoplastic, requires critical probing. Berman[3] has indicated that an evaluation of the recorded cases which have reached necropsy reveals that almost all of these have shown lymph-node and visceral dissemination. This contention has found many adherents and is frequently quoted in the standard texts of pathology. The inability of earlier writers to recognize the frequency of dissemination has been ascribed to a failure of thorough and critical study. On the other hand, Allen[2] has observed visceral lymphoma in comparatively few of his cases. Table VI lists eighty-eight cases of mycosis fungoides encountered in a survey of the literature. In each instance necropsy examination was carried out. Twenty-eight (32 per cent) exhibited no evidence of extracutaneous distribution of lesions. These results are somewhat weighted by three series in which visceral involvement occurred in thirty-eight of forty-three cases and one in which this occurred in only three of twenty-one cases. Since these represent selections from the literature and from collected cases at consultation centers, an element of overlap of single case reports also published may be conceded.

A fortunate circumstance has permitted the direct examination of case records, and postmortem protocols and tissue from forty-one patients with mycosis. A composite of this personal experience with that garnered from the reports of others is shown in Table VII. Although there is only a slight change manifested in the over-all incidence of visceral lesions, the personal series exhibits a bare majority of cases without this phenomenon. It must be clear, however, that mycosis, in its advanced stages, need not present itself as a systemic disorder, at least in relation to the appearance of visceral lymphomatous lesions.

A closer analysis of the forty-one cases comprising the personal study brings to light the fact that the absence of cutaneous lymphoma constitutes no deterrent to the development of disease of this character in lymph nodes or organs (Table VIII). This may be taken casually to support the opinion that mycosis fungoides is merely a cutaneous reflection of visceral lymphoma. On the other hand, the number of instances in which the reverse was the case, coupled with the well-documented observations of patients whose cutaneous lesions antedated the appearance of neoplasm, wherever located, by two to four decades, makes this contention a very tenuous one.

TABLE VII. EXTENT OF INVOLVEMENT IN MYCOSIS FUNGOIDES NOTED AT NECROPSY

	Literature	*Personal**	*Combined*
Skin only	28 (32%)	22 (54%)	50 (39%)
Skin and viscera	60 (68%)	19 (46%)	79 (61%)
Total	88	41	129

*Includes 21 cases from the files of the Armed Forces Institute of Pathology. The remainder were gathered from a number of institutions, including the Massachusetts General Hospital and the Cincinnati General Hospital.

TABLE VIII. RELATIONSHIP BETWEEN CUTANEOUS NEOPLASIA AND VISCERAL LYMPHOMA IN MYCOSIS FUNGOIDES AT NECROPSY

(Forty-one Cases—Personal)*

Cutaneous Lesions: Nonneoplastic		17
Viscera uninvolved	13	
Visceral lymphoma	4	
Cutaneous Lesions: Neoplastic		24
Viscera uninvolved	9	
Visceral lymphoma	15	

*The author wishes to express appreciation to the Director, Armed Forces Institute of Pathology, Washington, D. C., for the opportunity to examine and include herewith data from twenty-one cases in the files of the Institute.

Comment

Despite the tenor of this presentation, there is a desire to be neither specious nor facetious. Semantic diversity in medicine of the order disclosed, unfortunately, is not peculiar to hemopoietic disorders. Moreover, merely spotlighting the issues will not serve as a substitute for solution. Having created a muddy roil, one cannot just smugly step aside, although there is little anticipation of an effect upon custom. Efforts at simplification of complex situations have many disadvantages, not the least of which are their failure to satisfy any but their initial proponents.

If the term "reticulum cell" is to be used, it must have considerably clearer definition than now exists. In essence, it cannot continue to mean all things to all men. If it is taken to indicate a primitive totipotential blood-forming element, the terms "primitive blood cell," "hemocytoblast" or "stem cell" are clearly more definitive and have had traditional usage. Its morphologic characteristics have been widely delineated and need not concern us here. The consideration of the cell as having relationship to primitive mesenchyme with capacity to form any or all mesenchymal elements falls into the realm of uncompleted embryologic research. If the function of phagocytosis constitutes its fundamental qualities, whether or not the cell is mature, the term "histiocyte" cuts across the welter of phraseology which has plagued this subject for well over half a century and, further, has the acceptance of the Committee for Clarification of the Nomenclature of Cells and Diseases of the Blood and Blood-Forming Organs (Fourth Report). This cell, too, has well and widely recognized morphologic features. The belief in a relationship to ordinary vascular endothelium has few adherents; it is generally regarded that the littoral sinus cell is closely akin to the histiocyte.

Since the argyrophilic fibrillar net comprising the framework of lymph nodes, spleen and other body structures has been shown to have the characteristics of collagen, the element instrumental in its production must be the fibroblast, whether or not this in turn may be derived by metamorphosis of still another cell. Needless to state, the fibroblast has well-recognized morphologic characteristics. The term "reticulum" should, in the interests of clarity, be reserved for descriptive purposes only and should not designate a concept, a system, a specific element or a tissue potentiality. Those devoted to the latter viewpoints should recognize the misconceptions which have grown out of their acceptance.

It is proposed, therefore, that there is no such entity as a reticulum cell and that reticulum cell sarcoma, as recorded in the literature, consists of a group of conditions related only in that they represent primary neoplasms of hemopoietic tissue. Uniform understanding may be expected only if lesions of this type are classified in terms of recognizable qualities (functional and morphologic) of their component cells. In those instances in which specific cellular identification is not possible, it is proposed that one use a recognizable generic term indicating neoplasm and tissue of

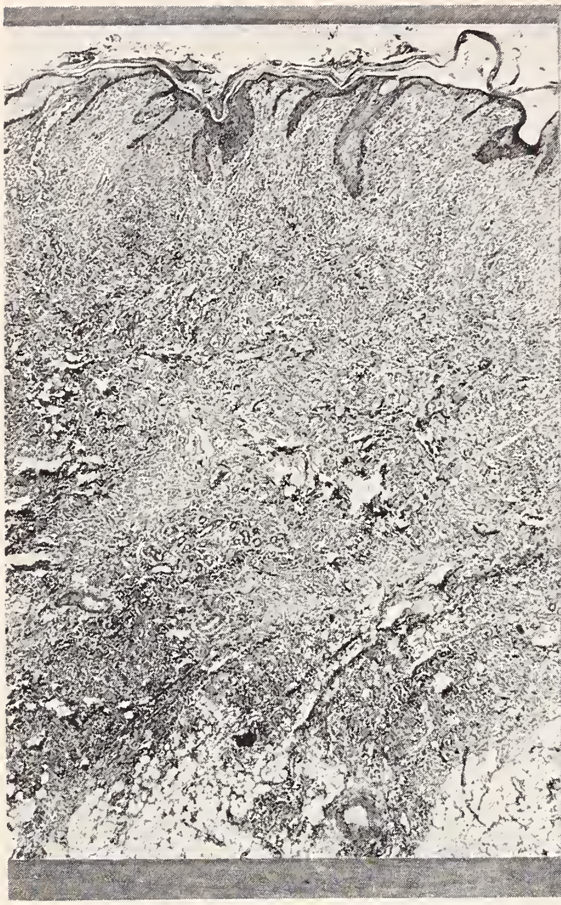


Fig. 2. Low-power photomicrograph of the skin in mycosis fungoides in the nodular phase. There is a dense cellular infiltrate proceeding in irregular fashion into the subcutaneous fat.

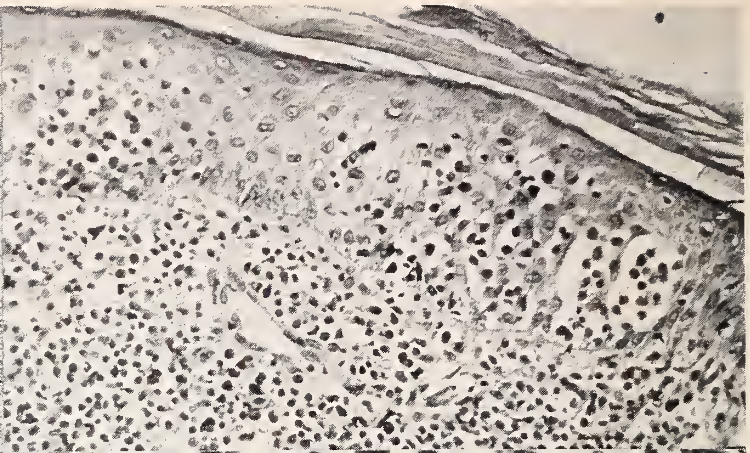


Fig. 3. *(above)* Medium-power view of the cutaneous lesion in classic mycosis fungoides during the plaque stage. The heavy lymphoid infiltrate of the corium is accompanied by intra-epidermal invasion (Darier-Pautrier abscesses).

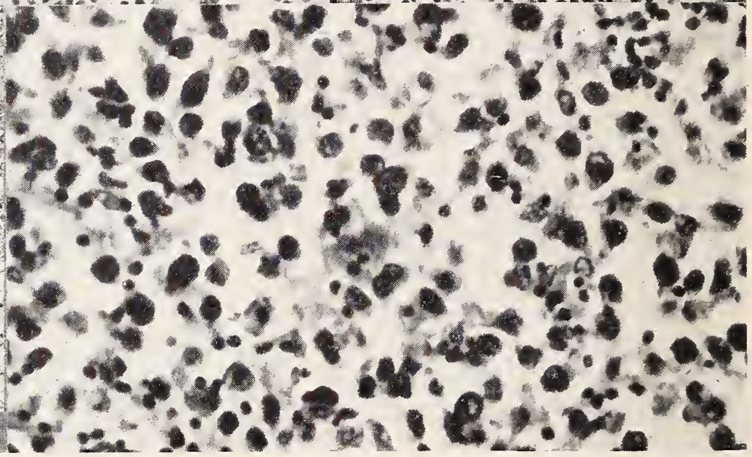


Fig. 4. *(below)* The fungoid stage of mycosis fungoides. A high-power view of the corium demonstrating the pleomorphic cellular character of the lesion.

origin, such as malignant lymphoma or lymphoblastoma.

The characteristic train of events of the clinical syndrome of mycosis fungoides and the long, relatively satisfactory usage of the term dictate its retention. It is ncessary, however, to attempt to set in proper perspective certain aspects of the disorder which seem to have had varying interpretations. The three characteristic phases as described above are rather universally appreciated. On the other hand, there is a sizable body of evidence to suggest that the process does not necessarily proceed inexorably through each stage to a fatal conclusion. It would seem that any one of a considerable number of forms of chronic dermatitis may, but do not always, lead to the series of alterations representing mycosis fungoides.

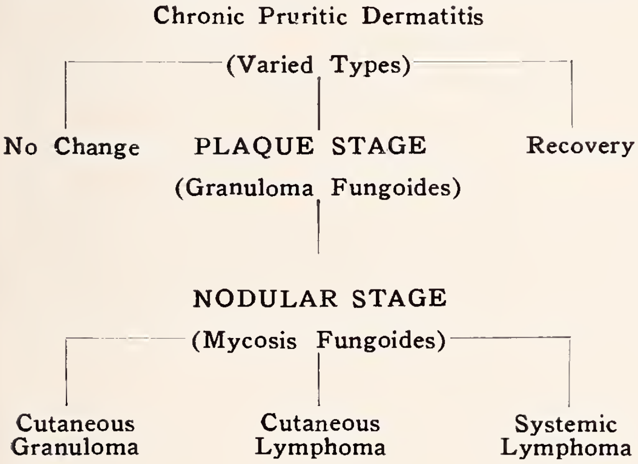
Thus, the diagnosis rests on hindsight and not upon prophetic foresight. When, however, the cutaneous lesion assumes, as it may, the features of the second (infiltrative and plaque) stage, the procession of events becomes more nearly inevitable. In contradistinction to the alterations in the initial process, the lesion at this stage appears to be pathognomonic. Characteristic are a peculiar pleomorphism with relatively aggressive mitotic proliferation of histiocytes, an intermixture of other leukocytes, the presence of bizarre giant cells (usually symplastic reactive elements, occasionally simulating Sternberg cells), deeper extension into the corium and intra-epidermal invasion in the form of "Darier-Pautrier abscesses" (Figs. 2, 3 and 4).

The great majority of cases in which this phase has been attained may be expected to progress to the stage of exuberant nodule formation. In a few, these fungoid masses retain the pleomorphic pattern of the second stage and do not develop lesions meeting the cytologic requirements of neoplasm. A few assume an appearance retaining some of the inflammatory polymorphism subtly altered to simulate Hodgkin's disease. Most cases at this point, however, exhibit widespread, but not necessarily universal, frank conversion into one or another of the forms of malignant lymphoma. There is no uniformity in the type of lymphoma produced.

Of the group in which mycosis has advanced to this point, a sizable number succumb without specific alterations of lymph nodes, blood or viscera. On the other hand, over half of the patients at necropsy reveal extracutaneous distribution of the lymphomatous lesions, which may even be accompanied by leukemia.

Schema

PATHOGENESIS OF MYCOSIS FUNGOIDES



The accompanying schema represents an effort to demonstrate this pathogenetic concept in graphic form. This schema is open to the usual charges of over-simplification and presumption but data advanced in the body of this paper seem to warrant such a contrivance. There is certainly no evidence to justify the contentions that mycosis fungoides is neoplastic or even a specific process at its outset. Moreover, the sequence of transitions, although seemingly immutable at the fatal conclusion of the disease, does not seem to be so in the initial phase. It is probable that a goodly number of patients considered to have a premycotic lesion fail to show progression or may even exhibit remission of the dermatitis. Such instances, naturally, would uncommonly find their way to publication. Finally, among those persons who eventually do exhibit the final fungoid lesions, these are not invariably lymphomatous, nor when they are, is there always visceral lymphoma. The possibility that lymphomatous transformation represents the effect of long-continued irritation of varied nature upon the skin and lymphoid tissue represents an interesting, albeit tenuous hypothesis.

Summary

1. A survey of the pertinent literature indicates a serious incompatibility of opinion with regard to reticulum cell sarcoma and mycosis fungoides.

2. There appears to be no satisfactory means of bringing into harmony the expressed views on either the form or the function of the reticulum cell.

3. It is proposed that the term be avoided and that more definitive appellations be employed in designating the several cells which have apparently heretofore been placed in this category.

4. The diversity of viewpoints in the pathogenesis of mycosis fungoides seem resolvable if the recorded observations are properly apportioned.

5. By utilization of data derived from the literature and from a sizable personal experience, a seemingly valid pathogenetic thesis is evolved.

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(Continued on Page 705)

Will Analgesia Evolve from a Century of Anesthesia?

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MAY I assert, first, that I am not advocating use of a particular drug but expounding an idea. I am, furthermore, continuing to entertain the hope that a new, safer, more pleasant era in the art and science of pain-relief may be in the offing—perhaps not far in the offing.

For thirty years, I have sought to produce a general condition, wherein the patient would retain some consciousness but at the same time his sensibility to pain would be so reduced that he would tolerate some operative pain. He might even remember something of the operative procedure but would not have been hurt enough to complain—then or later. To designate that state, in my own mind, I have used a term which has varying significance to various people. I have called it "analgesia." For the purposes of this presentation, therefore, when I use the word will you please think of the state I have described? If an etymologist will suggest a new and precise term, such as Oliver Wendell Holmes offered to Morton on November 21, 1846,[1] I, and doubtless others, will welcome his contribution. Because of the pronounced forgetfulness of recent events which attends use of the new drug, Victor Cohn has suggested the term "controlled amnesia."

Chronologic Periods in the Modern History of Generalized Pain-relief

Leaving out of consideration here local and regional measures for combating pain, three periods in the science and art of pain-relief can be distinguished. The boundaries are not absolute for sometimes, in one period, an experience was had which foreshadowed a later period. The expected results, however, were prevailingly those now to be described:

The first period was that in which various agents were used to produce general anesthesia. The patient was unconscious and tactile sensibility was abolished. In this period of anesthesia, even late in the period, the state of relaxation sometimes was insufficient to allow the surgeon to operate as gently and safely as his personal standards demanded. Then, in the attempt to gain satisfactory relaxation by deeper anesthesia, occasionally a patient was lost.

That type of tragedy led to the second of the three periods. Really it was a subdivision of the first and third periods, and it may be thought of as the era when relaxants were used. The purpose generally was to bring about satisfactory relaxation of the patient who was unconscious from the effect of general anesthetic agents. This was the main purpose of balanced anesthesia.

To be sure, over the years during which I used balanced anesthesia, sometimes I produced analgesia. Almost always, however, there was something undesirable about the result. If the main agent was nitrous oxide, the patient was likely to become cyanotic or to move. If the preponderant agent was something else, administration of a relatively small dose of it would be accompanied by some unsatisfactory phenomenon which could be banished by increasing the dose. Then the patient became quiet and satisfactorily oxygenated but—he was under anesthesia.

The third period, that of analgesia, really was entered ages ago. Anesthesiologists, therefore, have long been in a position to investigate the analgesic effects of small doses of anesthetic agents combined with narcotics or other agents.

Abridgment of paper read at the meeting of the Minnesota State Medical Association, Duluth, Minnesota, June 7 to 9, 1954, and, with modifications and additions, at the meeting of the American Medical Association, San Francisco, California, June 21 to 25, 1954; published in full, under another title, in JAMA, 157:1399-1403 (Apr. 16) 1955.

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The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

TABLE I. EXPERIENCE WITH MRD-125; JULY 30, 1953 TO MAY 31, 1954, INCLUSIVE: 112 PATIENTS (27 MALES; 85 FEMALES)

Category		MRD-125										Totals	
		Alone	With premedication						And curare. No nitrous oxide or tube	Without premedication. But with nitrous oxide and oxygen			
				And nitrous oxide and oxygen									
					And tube	And anectine‡ and tube	And curare				And tube		
								And tube					
Age, years	10 to 20		5	15	3	1*				1		25	
	21 to 40	2	13*†	29	2	6	1	2		1	3	59	112
	41 to 60	2	4	8	7				1			22	
	61 and more	1	1	3	1							6	
Type or region of operation	Dental	4	21*	53	11	7*		2		2	3	103	
	Eye			1	2		1					4	
	Dilatation and curettage			1								1	112
	Radium to uterus		1									1	
	Anal papilla								1			1	
	Bilateral chem. splanchnic block	1										1	
	Caldwell operation		1†									1	

*In 1 case, pentothal given after supply of MRD-125 exhausted.
†In 1 case, premedication followed by topical and regional anesthesia, supplemented by MRD-125.
‡Anectine is succinylcholine chloride.

An Enlightening Experience

On July 30, 1953, I injected MRD-125* into a vein of a volunteer subject. The substance is 5-ethyl-6-phenyl-meta-thiazane-2,4-dione. In order to determine what effect the drug was having, I caused the conscious subject's arm to be pricked with a pin but he did not move. That effect I had not elicited so soon with any other general anesthetic agent. I thought, if this drug will anesthetize the skin like that, it ought to be effective in the distribution of the trigeminal nerve, which innervates both skin and teeth.

Consequently, I administered MRD-125 intravenously to two patients who were to undergo extraction of teeth.[2-6] When they were fully analgesic, they still would swallow and open and close their eyes on command. This was too much for one physician-spectator even though he had considerable knowledge of the potentialities of the drug. "I must leave the room," he said. "I cannot stay and witness this torture."

But there was no torture, no pain. The drug had more analgesic qualities, in smaller dose, than any I was used to. Recovery, furthermore, was particularly prompt.

My associates and I now have impressions gained in 112 cases (Table I) in which MRD-125 has given evidence of its analgesic effect, especially when preliminary medication was given. In 103 of them, the operation was dental extraction. Results were substantially those obtained in the two cases at which the spectator unnecessarily took fright. One of these 103 cases was particularly interesting. The patient was the subject of a demonstration, on April 15, 1954, before a group of physicians who were taking refresher courses at the University of Kansas Medical Center. She was seventy-three years of age and had a fractured hip. She needed some teeth extracted while in the hospital and, for this purpose, she was moved from her room to the operating room. Movement caused pain to her and she complained of it even after the usual preliminary medication had been given. The pain ceased, however, after she received a dose of MRD-125 that was too small to allow dental extraction or to cause her to stop talking.

Nine cases of the 112 were not in the dental category. I shall not comment on all of the nine. However, in one of them an operation was performed on the ocular muscles. Results were not better than those obtained in such cases with pentothal and curare. In another case, dilatation and curettage were performed.

MRD-125, besides being an analgesic drug, is an anesthetic drug, and more must be said of its use as such. When MRD-125 is used for general anesthesia, certain desirable and certain undesirable effects are obtained.

Favorable effects are several. There seems to be little depression of respiration. Blood pressure, pulse rate and the electrocardiogram remain substantially unaffected. The electro-encephalogram, which as long as MRD-125 is used for analgesia resembles that obtained when pentothal is used, changes, when the MRD-125 has caused general anesthesia, to one somewhat like that caused by cyclopropane. Bleeding seems to be lessened. Dr. Louie T. Austin has noticed this in oral surgery. Dr. T. H. Seldon[7] has some experimental evidence on it. With the aid of a Clark window, he observed the capillaries in the rabbit's ear. His opinion is that when MRD-125 was administered, the capillaries, as when nitrous oxide and oxygen are used, were smaller than when such drugs as ether, cyclopropane, ethylene or pentothal were given. In a few cases in which MRD-125 has been used, the skin temperature of the great toes, and of the right calf, has been determined with thermistors; the general rise in temperature was slight unless premedication had been given. Let me emphasize that the previous sentence began with the words "in a few cases." What may turn up later, I don't know.

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*The Wm. S. Merrell Company, Cincinnati, Ohio. After this paper was read, the manufacturer gave the preparation the name "Dolitrone."

Other pertinent, but as yet limited, data were obtained in a series of cases in which Dr. T. M. Terpinas[8] and I were producing anesthesia for dental extraction. After Dr. Terpinas had observed 100 patients, and his apparatus was still standing, I asked that he observe twenty-five more patients but that he substitute MRD-125 for pentothal. This he did and compared the last twenty-five of his 100 patients who had received pentothal with the twenty-five who had received MRD-125. He was able to show that the arterial oxygen saturation when MRD-125 was used always was equal to, or was much more than, that when pentothal was used. This may be because respiration is depressed more by pentothal than by MRD-125. However, both groups of patients received adequate oxygen. The twenty-five patients who received MRD-125 under the observation of Dr. Terpinas are included in the 112 cases of the table.

Still further favorable effects have been that relaxation has been much better with MRD-125 than with nitrous oxide alone; this is true either of analgesic or anesthetic doses. Also, with the patient anesthetized, introduction through the nose or mouth of an endotracheal tube into the larynx and trachea has been relatively easy. Laryngeal spasm usually has been absent.

Unfavorable effects of MRD-125, when used as an anesthetic agent, sometimes are seen. Thrombophlebitis occurs but in almost negligible frequency. Brief nausea has followed operation in some cases.

As yet, to put MRD-125 into solution, the solvent must be more alkaline than it should be. The alkalinity is by no means of unheard of degree, for the pH of a 2.5 per cent solution of MRD-125 is 11.4 whereas that of a 2.5 per cent solution of pentothal is 10.4. Progress, however, is being made to decrease alkalinity of the solution.

The question will be asked whether tolerance or sensitivity can be expected to develop when MRD-125 is used. I have given it twice to only one patient. No signs of either tolerance or sensitivity were evident.

Of course, I don't know all there is to know about MRD-125. I have an experience of only 112 cases, plus the experience which has accumulated since this preliminary report was put together. Thus far, more than any other product, it has seemed, when combined with other agents, to bestow a humbly hoped for, not necessarily a final, blessing on thirty years of effort to produce safe, smooth analgesia by the balanced use of various agents.

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Foibles and Pitfalls in Obstetric Hemorrhage

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DESPITE the progressive reduction in maternal mortality, far too many women still die unnecessarily. Obstetric hemorrhage, the major cause of deaths associated with pregnancy and childbirth, is particularly hazardous and lethal. Over the country, it is responsible for more than a third of all maternal deaths. It contributes importantly to many others. The yearly constancy in the deaths due to obstetric hemorrhage is extremely distressing, since both knowledge and facilities now are available to avert a large percentage of these fatalities.

For dramatic effect, newspapers often exaggerate in their headlines and captions. In the public press, hemorrhage might be headlined with words like *suicide, homicide, manslaughter*. For us, however, these terms are false and misleading. Some women might be said to have unwittingly committed suicide through failure to obtain medical care or by disregarding the counsel of their physicians. Such malevolence does not exist professionally. Likewise, homicide and manslaughter—nasty and disagreeable words—are alien to medical practice.

Like Bunyan's Pilgrim, we suffer as physicians on our chosen journey through life. We walk through the Valley of the Shadow of Death. We are beset by professional foibles and pitfalls; they waylay even the most conscientious and experienced practitioners. As we have gained understanding and competence, we have decreased our experience with catastrophe. Present maternal mortality rates are so low and serious complications so infrequent that no physician is able to learn adequately from his own cases or from the experience of his hospital.

Critical reviews of maternal deaths have become essential in determining what are the current problems in obstetric practice. Their evaluation is necessary to discover our professional shortcomings. Those maternal deaths which have occurred in Detroit since 1948 and in Michigan since 1950 reveal some of the foibles and pitfalls in obstetric hemorrhage (Table I).

TABLE I. MATERNAL MORTALITY

TOTAL MATERNAL DEATHS	447	
Probably Preventable	312	(69.7%)
Maternal Deaths Due to Hemorrhage	187	(41.9%)
Factors for Preventability	168	(89.8%)
Patient	18	(9.8%)
Physician	150	(80.0%)

(Detroit 1948, 1949, 1953; Michigan 1950, 1951, 1952)

These deaths involved 447 women. Studied dispassionately, not in an effort to find items for censure, but sincerely to discover whether they might have been avoided had the best possible medical facilities and skills been available at the time, 312 women (almost 70 per cent) may be considered to have died unnecessarily. Of the total deaths, 187 (slightly more than 40 per cent) were due directly to hemorrhage. All of these have been studied personally. In almost 10 per cent, the patient was considered responsible. In 80 per cent, there were factors which pointed to physician responsibility. It is evident that the seriousness of obstetric hemorrhage should never be minimized by those who care for the expectant mother.

The factors in these fatal cases are listed in Table II. They tell us nothing new. As the problems involved are reflected upon, three foibles become outstanding. These I shall call Diagnosis by Presumption, Management by Procrastination, and Treatment by Confusion.

Diagnosis by Presumption

Here professional myopia is unmistakable. The physician with restricted vision relies upon probability. He gambles with uncertainty. Uncritically, he honors the traditional reluctance to examine the patient who is bleeding, especially if pregnancy is probable. Such short-

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TABLE II. FACTORS IN FATAL OBSTETRIC HEMORRHAGE

Ruptured Uterus	42	(22.5%)
Postpartum Uterine Atony	41	(22.1%)
Ruptured Ectopic Pregnancy	36	(18.2%)
Premature Placental Separation	22	(11.8%)
Placenta Previa	17	(9.2%)
Laceration Cervix/Vagina	8	(4.4%)
Retained Secundines	6	(3.4%)
Abortion	5	(2.8%)
Other	10	(5.6%)

Placenta Accreta	3
Postoperative	3
Uterine Inversion	1
Ruptured Ovarian Vein	1
Afibrinogenemia	1
Undetermined	1

sightedness usually is associated with improper habits of examination. Diagnoses based upon the patient's recital of symptoms, not supported by physical findings, are accepted and the incident minimized. Tranquil optimism of this sort is the lull before the storm.

Ectopic pregnancy is illustrative. Characteristically, the patient has a delayed or abnormal menstrual period. She may have pelvic pain. Too often, she thinks little of these and seeks no medical care. More than a third of the women dying from a ruptured ectopic pregnancy had not been seen by a physician until they were dead or moribund. Patients must be impressed that they should see a physician promptly, if they are two weeks beyond their expected menstrual period. There must be no delay if there also is pelvic pain, with or without spotting. The notion of waiting until pregnancy has advanced to three months before seeing a physician must be corrected vigorously by all of us if we are to treat our patients properly.

A careful history, including the nature of at least the three previous menstrual periods, is important in arousing suspicions of an abnormal pregnancy. But this is not enough. Every patient must be carefully and thoroughly examined, always with the intent of *proving* the presence of an ectopic pregnancy. Proper habits of examination are essential. Casual pelvic examination, especially when done in bed and restricted to the rectal route, generally misses the tender adnexal mass of a tubal pregnancy.

One must always remember that despite what is learned from the history and examination, additional points towards diagnosis may develop. Recently, a presumptive diagnosis of incomplete abortion was made. The patient was sent to the hospital for a curettage. The usual casual pelvic examination was done, this time under anesthesia, and the uterus curetted. During her reaction from the anesthetic, the patient complained of severe pelvic pain. Hypotension and an elevated pulse rate were found. An alert colleague suspected a ruptured ectopic pregnancy, ordered blood for transfusion, and recalled the physician. By the time he arrived, the diagnosis was obvious. Adequate, prompt blood transfusion and immediate operation averted a fatality.

Bleeding in any pregnant patient may herald a catastrophe. Diagnosis of the cause demands alertness. It demands a meticulous review of preceding events. It demands a cautious, yet thorough, pelvic examination. It demands a constant watch for additional diagnostic clues. Whenever a situation permits the suspicion of an ectopic pregnancy, and pelvic examination, even under anesthesia, is unsatisfactory or equivocal, aspiration of the cul-de-sac should be done. Colpotomy with visualization of the pelvic structures often clarifies obscure cases.

A word regarding treatment of ectopic pregnancy. In a third of the preventable cases, no operation was done. The diagnosis was missed. Pelvic examination either was not done or was inadequate. In a fourth of the professionally preventable cases, the patient was moribund when operated upon. In every instance, the diagnosis was apparent many hours before. Management of ectopic pregnancy is simple. It involves but three steps: early diagnosis, adequate replacement of the blood lost, and operation.

Management by Procrastination

This might be called the "ostrich" approach. Management based upon uncritical presumptions is haphazard and illogical. The physician, whose optimism leads him to elect a program of "wait and see," usually neglects to consider the dire hazards inherent in obstetric abnormalities. Such lack of foresight permits complications to increase to the degree that disaster becomes inevitable. The fallacies in procrastination are many. We hear them often. Out of context, they are recognized easily. "It's only spotting, means nothing, and will stop." "She's a multipara and won't have any trouble." "I don't think hospitalization is necessary now." Delusions like these undermine professional capability and lead to tragedy.

Uterine bleeding in the last trimester never is meaningless. Painless bleeding is an early symptom of placenta previa. The first bleeding rarely is fatal. The alert physician will have sufficient time to manage this complication successfully. Of the seventeen deaths attributed to placenta previa, two or more episodes of bleeding before the fatal hemorrhage occurred were found in more than 75 per cent. Yet delay in diagnosis was almost universal. It is significant that nine of the seventeen women died undelivered. A third of the total received no blood whatsoever. Several of the patients objected to being sent into the hospital. They felt well, the bleeding was scant, they wished to stay at home. Their physicians overlooked the potential seriousness of the situation and did not insist that they go. In every case, the patient was a multipara.

Placenta previa requires active management. If the bleeding first appears before thirty-seven weeks, expectant management (directed procrastination, if you like) in the interests of fetal survival occasionally may be elected. Nevertheless, hospitalization is important for the proper planning of adequate treatment. On admission, complete blood study and cross-matching of whole blood must be done immediately. There should be no rectal examination. Such often provokes severe bleeding; it never gives satisfactory information.

Generally, the initial bleeding subsides with bed rest and mild sedation. At this point, choice of management should be made. Roentgen study by soft tissue technique for placental site is exceptionally useful. With experience, diagnostic accuracy is about 95 per cent. It also demonstrates the frequently associated malpresentation. Of the cases of placenta previa occurring in our hospital, breech or transverse lie was the position in about one-fifth and an abnormally displaced vertex in almost 15 per cent. Roentgen study, in my opinion, is essential if there is to be any thought of delaying intervention.

As soon as blood becomes available, demonstrated blood loss must be replaced. Expectant treatment cannot be a primary choice, if labor is present, if the fetus is close to term, or if the bleeding continues in any significant degree. Vaginal examination under these circumstances becomes mandatory. It is the only reliable means of accurate clinical diagnosis. This requires the so-called "double set-up," a prepared operating room with all necessary assistants at hand, the instruments laid out and properly cross-matched blood in the room. After intravenous fluids are started and the bladder emptied, a planned, gentle vaginal examination is done. Such will determine the necessity and method of delivery. A decision made after adequate preparation and in an orderly manner as has been described allows the physician, not the emergency, to direct therapy.

Treatment by Confusion

This represents professional hysteria. Based upon expediency, it is always thoughtless, often selfish. The procedures resorted to are inappropriate and badly timed. Complications become compounded until chaos threatens. Desperation then replaces judgment and training.

Rupture of the uterus was the largest single cause of death from hemorrhage. In some 20 per cent of these, oxytocics were used either to induce labor or to overcome dystocia. While induction of labor for convenience may have a defense, I believe that responsibility for any subsequent difficulty rests squarely upon the physician. Oxytocics should never be used until the obstetrical problem is crystal-clear and no contraindications are *proven* to exist. There were forty-two deaths due to uterine rupture. In a third, version and extraction was used for delivery. In half of these, the cervix had to be incised or dilated manually before it could be carried out. In every instance, version and extraction appeared unnecessary. The situations which provoked its use either could have been avoided or answered by less hazardous means, had they been fully evaluated when the patient entered the hospital. It is significant that only eight of the women in this group were delivered spontaneously or by truly *low* forceps. The multiparous uterus has a notorious liability to rupture. Only 15 per cent of the uterine ruptures involved primigravidae. In all of them, dystocia was long in evidence. Rupture followed version and extraction or unsuccessful attempts at forceps delivery. In all of the deaths where rupture was associated with a major obstetrical procedure, preparation for the undertaking was inadequate. Half of the women were given no blood at all or were given small amounts too late to be of help.

These illustrations of the foibles of presumption, procrastination, and confusion admittedly are unpleasant. In a way, they are but preliminary sketches. With added detail, they reveal the grimness of the pitfalls which surround obstetric hemorrhage. There are three major pitfalls—the acceptance that most hemorrhage is unavoidable, the assumption that there is no hemorrhage unless there is shock, and the belief that time is a healer.

Hemorrhage Avoidable

It is unfortunate that certain of the complications involving bleeding during pregnancy have been called "unavoidable." This promotes professional resignation. It must be remembered that *manageable bleeding* always precedes hemorrhage. Some of the complications, such as ectopic pregnancy, placenta previa, and premature placental separation, are the result of pathologic conditions which we cannot alter. Nevertheless, all can be managed with success.

Resignation in the face of hemorrhage is disastrous. It results from failure to diagnose the cause of bleeding early, from delay in appropriate treatment, and from inadequate support of the patient during the bleeding episode. We must have a proper attitude towards hemorrhage. We must regard bleeding as a warning sign of potentially grave trouble. It often leads to hemorrhage. Hemorrhage rapidly may become extremely difficult to combat. Any bleeding during pregnancy must be respected.

Shock Following Massive Blood Loss

In hemorrhage, it has been demonstrated repeatedly that if the systolic blood pressure falls below 100, a loss of at least 30 per cent of the blood volume must have taken place. This means that in moderate shock, with a systolic pressure of 80, there has been blood loss in the neighborhood of 1,500 cc. If we are to diagnose hemorrhage so as to circumvent fatalities, we must be more accurate in our determinations of blood loss. Usually, we are only half right. I suspect that our observations often are biased and inaccurate, because there lurks in us the feeling that we personally are responsible for hemorrhage. Certainly, we tend to minimize undesirable events. This is especially so when personal experience with fatal complications is small.

Because there is reliable treatment for obstetric hemorrhage, it must be considered first whenever shock is encountered. Beware of concealed hemorrhage. If no bleeding is evident externally, think about premature placental separation, ruptured uterus, and ruptured tube. Until blood loss as the cause for shock can be demonstrated *not to coexist,* never think of explaining it as due to drug reaction or trauma. Remember, too, that blood transfusion, specific treatment for hemorrhage, is beneficial in many other causes of shock. While working out the diagnosis, at least order cross-matching of blood.

Reliance solely upon fluids and vasopressors is foolhardy. Evidence is accumulating that excessive vasoconstriction may not only produce shock but may seriously delay renal recovery as well as perpetuate circulatory inadequacy. Moreover, vasopressors may hide the persistence of a significant blood deficit. Fluids and vasopressors should be used only as emergency means of support for the patient in shock until specific therapy can be provided.

The Physician, Not Time, the Healer

When resignation creeps into the management of obstetric emergencies, professional rout is certain. The malevolence of disaster quickly can pervert our judgment and produce a strong feeling of personal inadequacy. We react too often with false heroics. We mumble about "irreversible shock" and resort without conviction to uterine packs, hysterectomy, and a frantic procession of drugs. Some of these have occasional merit. But their use must be preceded by complete understanding of what is being combatted and what momentary advantage is expected. "Irreversible shock" really means therapeutic failure. The term has no place in clinical practice, because it is an open invitation to neglect in treatment.

In the face of apparently overwhelming reverses, we must maintain a high degree of professional courage. The adversity of our trials can be overcome by faith in ourselves and the proper use of our available resources. If we have battled catastrophe and seem to have won, we still must remain on guard and defensive. Other phases of the battle may try our resiliency and reserves. Bleeding may increase or recur. The blood may not seem to clot. Hypotension may persist, despite vasopressors. Oliguria or anuria may develop.

Now, in a positive way, I should like to summarize how the foibles and pitfalls in obstetric hemorrhage may be avoided. There are two aspects, prophylactic and active management.

Prophylaxis

There are four injunctions in prophylaxis.

1. *Be alert.*—At the first visit, inquire specifically into bleeding associated with previous pregnancies, with tooth extractions or other surgery, and any which occurred without reasonable cause. Obtain a complete blood count and determine blood type, Rh factor as well as serology. Remember, the emergency may occur before the next visit. Give supplemental iron in adequate amounts throughout pregnancy. If there is a significant anemia, treat it appropriately.

2. *Be suspicious.*—Evaluate any abnormal menstruation or other bleeding and pain. An ectopic pregnancy may rupture any time. With abnormal fetal position, placenta previa may be present. When hypertension is present and bleeding and uterine pain develop in the third trimester, premature placental separation is likely. The multipara is dangerous. Uterine rupture is a constant threat to her, particularly if there has been uterine surgery, overdistention from gestational products, or trauma from obstetric intervention.

3. *Anticipate.*—With a suggestive history or other cause for suspicion, be prepared to combat hemorrhage. Anticipate it in patients with prolonged labor. About 15 per cent of all inertia labors have postpartum hemorrhage. Retained placenta is far more common here. In prolonged labor, version and extraction should be outlawed as a method of delivery.

4. *Be prepared.*—If bleeding is suspected to occur, or if it has occurred, however small in amount, prior to or during labor, be certain that the patient has an adequate blood volume before delivery. Have at least 1,000 cc. of compatible blood in the delivery room. Maintain access to the circulation by a free flowing of fluid through a large gauge needle.

Active Management

There are five cardinal principles of active management.

1. *Replace all blood loss of* 500 *cc.*—Double the estimated loss. If, then, this is 500 cc. or more, replace it volume for volume. Never include in the calculation of replacement the fluids or plasma expanders which have been given.

2. *Support the patient.*—Volume-for-volume replacement of blood loss is essential. In emergency, plasma expanders such as P V P, Dextran, or 6 per cent gelatin may be used. Human plasma is least desirable. Maintain pulmonary ventilation, by bag respiration if necessary. Supply oxygen in high concentration. Support the circulation, using vasopressors to hold the systolic pressure at 80, until the source of bleeding can be controlled and blood volume adequately restored.

3. *Observe carefully after delivery.*—Find the cause of pre-partum or intra-partum bleeding and treat appropriately. Carefully explore the uterus and examine the cervix and vagina for lacerations following any difficult low forceps, all midforceps, breech, or version and extraction deliveries. Give an oxytocic at least after delivery of the examined placenta. Be constantly certain of the state and size of the uterus. If excessive bleeding occurs, establish the cause, remembering that uterine atony is diagnosed only by negative findings. For atony, add Pitocin (1.0 cc. per 1,000 cc.) to the intravenous fluids being given. If defibrination is suspected, observe blood samples for clotting and dissolution while replacing blood loss by whole blood transfusion. When indicated, give fibrinogen in two gram amounts to a total necessary to restore coagulation.

4. *Reappraise constantly.*—Every patient given intravenous fluids, intravenous oxytocic drip, or blood transfusion at delivery should be watched by a competent observer during such treatment, and for at least an hour after its completion. Subsequently, the patient should have regular and frequent checks of pulse, blood pressure, lochia, and urinary output for at least twenty-four or thirty-six hours.

5. *Be certain the bleeding is controlled.*—In the treatment of obstetric hemorrhage, the uterine pack appears as lethal as version and extraction. While it may be defended, critical reevaluation shows that it has only temporary value in dire emergencies. As generally employed, it hinders hemostasis. The usual uterine

pack can absorb 1,000 cc. of blood before additional loss can be seen at the vulva. It falsely engenders a sense of security. By delaying proper treatment it permits deterioration into severe shock which too often is found too late. There should be no hesitancy in doing a laparotomy in the face of obstetric hemorrhage when thorough appraisal of the situation indicates that such is necessary to control the source of hemorrhage. Remember, operation is done to staunch the bleeding. In this group of cases, in three instances operation was done but the bleeding was not stopped. In the extremely critical patient ligation of the uterine arteries may be preferable to hysterectomy. Hysterectomy should not be done if there is defibrination.

Shortly after I had completed what I thought would be the final draft of this paper, there appeared a report of the Maternal Welfare Committee of the Massachusetts Medical Society based upon its current study of maternal deaths. Two sentences of this report stated the conclusion I wished to make with such brevity and clarity that I discarded mine to quote from it.

> "Careful analysis of past deaths reveals that simple adherence to well established and freely accepted principles as taught in every medical school, combined with a certain minimum of equipment and facilities would have avoided the majority of them. Today, 'minimum facilities' must include immediately available blood for transfusion."†

All of us, I know, are dedicated to the principle that no mother should die a "preventable death." If we can realize our dedication, there will be no personal overtones in these lines from Thomas Hood,

"One more Unfortunate,
Weary of breath,
Rashly importunate
Gone to her death!
Take her up tenderly,
Lift her with care;
Fashioned so slenderly,
Young and so fair!"

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†New England J. Med., 252:739, 1955.

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CEREBRAL PALSY: RECENT RESEARCH

Previous abortions and miscarriages may potentiate cerebral palsy in subsequent children, according to studies conducted by Drs. Eric Denhoff and Raymond H. Holden of the Meeting Street School for Cerebral Palsy in Providence, Rhode Island. Observations on the histories of 100 cerebral palsied children compared to those of 504 consecutive ward births led the authors to state that "it must be assumed that the mothers of these (cerebral palsied) children have some constitutional factor or endocrine dysfunction which contributes to the aberrant development of their offspring."

It has generally been held that trauma before or during birth contributes to brain damage which then results in mental and physical defects. In this study, however, "although all the cases of the 'suspicious' group suffered some trauma at birth . . . nevertheless, those whose mothers had not previously aborted seemed to develop normally. On the other hand, the infants who were subjected to birth trauma *and* who were born of mothers with a history of previous abortions or miscarriages were later found to be more developmentally handicapped."

Because of these observations, the investigators suggest that a "maternal 'constitutional' factor (heredity, poor nutrition, physiologic dysfunction) may in some way lower the threshold of resistance . . . in the newborn so that he is more susceptible to brain damage than an infant born to a mother without this handicap. . . . In other words, this study seems to show conclusively that birth trauma *alone* is not necessarily responsible for later evidence of brain damage."—E. Denhoff and R. H. Holden: Etiology of cerebral palsy: an experimental approach, *American Journal of Obstetrics and Gynecology,* 70:274 (Aug.) 1955.

The Heritage of Ethics in Medicine

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WE LIVE in an age when, in many quarters, almost everything that is human seems to be regarded as something reprehensible. Man, by his mastery of forces that are not human, has managed to survive the most devastating war in all history, and he can even claim spectacular victories over his environment. He has made new conquests of the elements, and by subjugating these to his will, he has learned to kill with amazing facility.

Yet, many wise observers are afraid that in surrendering his security and his welfare to the machine and to new engines of destruction, man has lost his freedom of mind and soul, and has become the unhappy prisoner of fear and suspicion. He has become the victim of his knowledge today, even as he was once the slave of his ignorance. Having made the choice to place his reliance upon mighty forces which cannot think, feel or see, man is now tortured by his inability to live side by side with other men in an atmosphere of peace and mutual confidence and respect. If, as some have said dolefully, he has reached a point from which he dares not turn back but knows not how to go forward, it is not because he has learned too much about his environment, but because he has learned too little about himself. That vast array of accumulated knowledge about forces which can be harnessed to serve man has not been matched by mastery of man himself or by felicity in his relations with other men.

It is a curious thing that modern man, in his frantic search for more and more means of dependence on new instruments for security and perhaps even preservation, has not been wise enough to go back to his predecessors to see if by some chance they had managed to learn something in a bygone age which would be of value to him in the present. Even in the Middle Ages, enlightened men were willing to do that much. They went back to the glories of Greece long since dead, and when they applied what they found, they were able to create an age that is still an unparaleled wonder of human endeavor: the Renaissance. It was a rebirth of art, letters, concepts of freedom and the government of men, and of systems of ethics.

We do not hear too much about ethical systems today. Perhaps we are afraid to give much thought to ethical problems when we know that the head of a government who signs a solemn covenant of peace and international good faith may, in the next stroke of his pen, unloose the deadliest weapons of death and misery thus far devised by the ingenuity of man.

Yet there are systems of ethics, however feebly they may seem currently to be embraced and defended. And among the oldest of such systems—far older than the Renaissance of the Middle Ages—is the system of ethics of the physician. This particular system has had no Renaissance because it has needed none—it was evolved in a timeless age of antiquity greater than that of Greece itself, and it has endured to our own day with practical modifications which have served the needs of changing environments, but which have not perverted the ancient integrity of its objectives.

In the United States, in particular, members of the medical profession have been more than conscientious in shouldering the responsibilities implied in such a system of ethics. Our progress in public health, our improvements in medicine and the teaching of medicine, our concern that the best possible medical care shall reach all the people—these are tangible accomplishments which most reasonable Americans are able to perceive and evaluate for themselves.

In other respects, the usual American citizen's concept of the contribution of the medical profession to the great social questions of the day may not be so happy. He may have been told by politicians, for instance, that the American Medical Association only last year opposed a proposal

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for Federal re-insurance in the field of medical care, and he may have been assured that the American Medical Association also went on record against two provisions of the Social Security Act. Someone is almost certain to have told him that the American Medical Association differed with the Federal Government on official policy in the medical care of veterans with disabilities not related to their service in the armed forces.

Politicians of necessity have loud voices, and they know how to get into newspapers. Most physicians are quiet fellows, and if they are jealous of their good name, they try to keep out of the public press. So it is not surprising that the American public may not realize that the American Medical Association actually gave active support to no fewer than eleven of fifteen major medical bills enacted into law by the Eighty-third Congress. Of these fifteen major medical bills, the American Medical Association opposed only two. On two more it took no stand.

Four bills of major importance to every American were passed by the Eighty-third Congress, and the American Medical Association supported every one of them. These are the Hill-Burton Act, to stimuate and support the construction of hospitals, establishment of the U. S. Department of Health and Welfare, approval of a charter for the National Fund for Medical Education and extension of the so-called doctor draft law to provide adequate medical care in our armed forces. When you read in the public prints some highly colored attack upon the medical profession or the organization which represents it, remember also that the American Medical Association chose to take the part of the American people where it really counts—in the legislative halls where our laws are made, and not in sensational newspaper headlines or in flamboyant magazine articles by hucksters who have something to sell and are not always on speaking terms with the truth.

Are there, then, abuses in American medicine? There undoubtedly are, as there must have been even in Heaven, since we are told that because of his sins the Archangel Lucifer was cast headlong from that most perfect of all realms. You will note, however, that Lucifer was punished and driven out into iniquity by his celestial peers, without external assistance. Now, members of the medical profession do not claim to be angels, and they do not even—as a rule—resort to supernatural powers, but they do have effective means for stamping out abuses in their midst and punishing offenders, and they have had these means and used them quietly and efficiently over the centuries, long before anyone ever heard of a muck-raking magazine or a crusading newspaperman. Let us not forget that the army with the loudest trumpets does not always serve a just cause.

Virtually every one of the sensational popular accounts of medical abuses which go unpunished is false, without any foundation in fact. The abuses themselves would never be tolerated by the medical profession, which would be the first to take action against the offender, long before any question of law or even propriety arose. Since the ancient precepts of ethical conduct apply equally to physicians in the mass as well as the individual practitioner, the willful violator of these principles soon finds himself, in effect, banished to a solitary island where he has neither hope nor company, and where the deathly silence is broken only by the tolling of a bell which he knows only too well tolls for him alone. The medical profession many centuries ago learned how to correct any incipient infractions of its code, and it has Devil's Islands of its own which are always ominously visible on the horizon to the physician who might be tempted to swerve from the honorable course.

For more than 100 years the American Medical Association, as physicians far back into antiquity have always done, has utilized its *Principles of Medical Ethics* to serve in a manner prescribed by the preamble to those published principles:

> "These principles are intended to serve the physician as a guide to ethical conduct as he strives to accomplish his prime purpose of serving the common good and improving the health of mankind. They provide a sound basis for solution of many of the problems which arise in his relationship with patients, with other physicians, and with the public. They are not immutable laws to govern the physician. The ethical practitioner needs no such laws; rather they are standards by which he may determine the propriety of his own conduct. Undoubtedly interpretation of these principles by an appropriate authority will be required at times. As a rule, however, the physician who is capable, honest, decent, courteous, vigilant, and an observer of the Golden Rule, and who conducts his affairs in the light of his own conscientious interpretation of these principles, will find no difficulty in the discharge of his professional obligations."

That exemplifies current thinking on ethical problems in medicine. But we can go back much further than that. The pattern for all subsequent development of medical ethics was established by the oath of Hippocrates. This oath, named for the father of Greek medicine, is a marvel of compact expression. In some 300 words it outlines many of the basic principles on which have been built most ethical codes during the past 2,200 years. Hippocrates lived in the fourth century before Christ, but the pattern of principles which bears his name remains the prime feature of all systems of medical ethics. From the standpoint of integrity, no one has ever improved on these principles. From the standpoint of practical application, work never ceases on the problem of adjusting these principles to the requirements of the contemporary scene.

Every medical student learns, first of all, to abide by the Hippocratic oath, and the influence of this oath on the ethical beliefs of physicians of today is shown in the first section of the first chapter of the *Principles of Medical Ethics* of the American Medical Association. Here attention is called to the fact that a physician should be "an upright man, instructed in the art of healing." Furthermore, it is laid down that "he should also be modest, sober, patient, prompt to do his whole duty without anxiety; pious without going so far as superstition, conducting himself with propriety in his profession and in all the actions of his life."

The National Medical Convention, which was a forerunner of the American Medical Association, at its first meeting in New York, held in 1846, appointed a committee to prepare a code of ethics. The proposed code was offered at a meeting which was held in Philadelphia in 1847.

The present *Principles of Medical Ethics* have evolved, with revisions, additions and changes of wording from this early code. At the present time and since June of 1953, the *Principles* have been undergoing another complete revision. There will be future revisions, because physicians are as alert as anyone else to the changing needs of their time, but the fundamental integrity of the individual has not been revised, and I hope it never will be.

Through the *Principles of Medical Ethics,* the American Medical Association endeavors to engender and assure proper behavior on the part of its members. A casual review of these principles by an impartial observer cannot do otherwise than convince him that no one is guided by a higher ethical code than that of the physician.

Some portions of the principles are devoted to the duties of physicians in their relationships with their patients. Others deal with the duties of physicians in regard to other members of the medical profession, and still others pertain to the duties of physicians in their relationship with the public. An attempt is made to establish principles of a broad general nature, instead of providing clear-cut laws. It is a mistake to regard these principles as either inflexible laws or confining rules.

"The prime object of the medical profession is to render service to humanity; reward or financial gain is a subordinate consideration." Good medical service cannot be expected unless society appreciates the value of such service sufficiently to reward properly those who can and will give good service. But it is equally true and even inevitable that the medical profession will lose its right to survival and will perish if it places financial reward above humanitarian considerations. Whenever the medical profession, as has been the case in some countries under compulsory sickness insurance (better known as "socialized medicine"), has been forced to make financial reward a primary consideration, the quality of medical service and with it the health of those served has suffered a decline. Any attempt to make the conquest of disease subordinate to the income of the physician, or to produce and distribute such services according to competitive standards, backed by methods of salesmanship, violates the fundamental principles of medical ethics and is degenerating to the individual as a physician.

The *Principles of Medical Ethics* are clear enough. But to ensure that they will be interpreted correctly and applied equitably, an appropriate deliberative body is necessary. The value of this arrangement is attested by courts of law, which perform a similar function in forensic matters, and by clergymen, who interpret theological principles for their congregations. The judicial body of the American Medical Association is the Judicial Council, and its duties are similar to those of the United States Supreme Court.

Let us examine some of the provisions of the *Principles.*

Advertising

"Solicitation of patients, directly or indirectly, by a physician, by groups of physicians or by institutions or organizations is unethical. This principle protects the public from the advertiser and salesman of medical care by establishing an easily discernible and generally recognized distinction between him and the ethical physician. Among unethical practices are included the not always obvious devices of furnishing or inspiring newspaper or magazine comments concerning cases in which the physician or group or institution has been, or is, concerned. Self-laudations defy the traditions and lower the moral standard of the medical profession; they are an infraction of good taste and are disapproved."

We know that this principle is sound because we know that the advertising of medical service has never failed to break down standards and to lower the quality of that service, anywhere, in any age. Advertising and solicitation are so inseparably a part of modern economic life, however, that it requires continuous vigilance to guard against their intrusion into the field of medicine. Physicians generally recognize that in an age of scientific wonders there is a constantly increasing body of news and information concerning disease which is helpful to public health and medical progress. Yet it is also true that exploitation of such news for profit may harm the public. Everyone knows how sensational publicity of "cures" for cancer, tuberculosis and other diseases has resulted in unnecessary suffering and false hopes of relief. This, to any reasonable man, is cruel and reprehensible. Sometimes, publications by physicians in medical journals actually are only preliminary announcements of incomplete scientific investigations, and they are frankly stated to be such by their authors. They are intended only to inform the profession of the nature of work in progress, and they do not present final results or conclusions. Often, publications which have appeared elsewhere than in medical journals have been flagrant fakes.

Radio and television broadcasting has brought new advertising problems. Quacks, patent medicines and all kinds of "cures" thrust their advertising into millions of homes. There is no way to measure the injury to the public health that has resulted from selling such "medical" services without appropriate evaluation and supervision. The American Medical Association attempts to combat this situation by educational medical publicity designed to assist the public in caring for its health and to provide information concerning appropriate methods of obtaining medical service. It has always done this, but without fanfare or crashing cymbals. No physician has ever held back from the public a remedy that has been tested adequately by reputable scientists and found to be efficacious against disease.

Payment for Professional Service

"The ethical physician, engaged in the practice of medicine, limits the sources of his income received from professional activities to services rendered the patient. Remuneration received for such services should be in the form and amount specifically announced to the patient at the time the service is rendered or in the form of a subsequent statement.

"Unethical methods of inducement to refer patients are devices employed in a system of patronage and reward. They are practiced only by unethical physicians and often utilize deception and coercion. They may consist of the division of a fee collected by one physician ostensibly for services rendered by him and divided with the referring physician or physicians or of receiving the entire fee in alternate cases.

"When patients are referred by one physician to another, it is unethical for either physician to offer or to receive any inducement other than the quality of professional services. Included among unethical inducements are split fees, rebates, kickbacks, discounts, loans, favors, gifts, and emoluments with or without the knowledge of the patient. Fee splitting violates the patient's trust that his physician will not exploit his dependence upon him and invites physicians to place the desire for profit above the opportunity to render appropriate medical service.

"Billing procedures which tend to induce physicians to split fees are unethical. Combined billing by physicians may jeopardize the doctor-patient relationship by limiting the opportunity for understanding of the financial arrangement between the patient and each physician. It may provide opportunity for excessive fees and may interfere with free choice of consultants, which is contrary to the highest standards of medical care."

The ethical antagonism to "fee splitting" which is forbidden in this section is based on the fundamental principle that whatever weakens the confidential relationship between patient and physician is likely to make diagnosis more difficult, treatment less effective and recovery less prompt. No patient would choose to be the unwitting victim of secret negotiations when his health or even his life is in the balance. Frank and open relationships with the patient are destroyed when the specialist secretly rebates a portion of his fee to the referring physician. The American Medical Association has always maintained steadfast opposition to such practices.

Limits of Gratuitous Service

"Poverty of a patient, and the obligation of physicians to attend one another and the dependent members of the families of one another, should command the gratuitous services of a physician."

The professional obligation of physicians to give gratuitous services when the poverty of the patient allows no other course is in direct contrast with the principles of modern commodity economics. Food, clothing and shelter vie with medical care in almost equal importance to health, yet no obligation is assumed by the producers and purveyors of these commodities to recognize poverty in fixing prices or as a reason for gratuitous service. Nevertheless, the age-old acceptance by the medical profession of this duty of gratuitous service to the poor has been of great economic importance. The physician never receives any remuneration for a considerable part of his services to the sick. Very few of his fellow-citizens, however, take the time to consider this notable contribution on his part.

The Physician's Responsibility

"The avowed objective of the profession of medicine is the common good of mankind. Physicians faithful to the ancient tenets of this profession are ever cognizant of the fact that they are trustees of medical knowledge and skill, and that they must dispense the benefits of their special attainments in medicine to all who need them. Physicians dedicate their lives to the alleviation of suffering, to the enhancement and prolongation of life and to the destinies of humanity. They share whatever they have learned and whatever they may discover with their colleagues in every part of the globe. They recognize instinctively that the need for improvement of medical knowledge and skills is never at an end, and while they strive toward satisfaction of this need they are zealous in making available to physicians of good character who possess the desire and the ability to learn the aggregate of progress in medical education, research and discoveries as they may exist at the time. They do not remain content to limit their activities to the care of the infirm, since they recognize also their useful rank among the vast concourse of citizens upon whose shoulders the destiny of our nation rests. At the same time they will resist attempts to debase their services by diverting them to ignoble purposes. In their relationships with patients, with colleagues, and with the public, they maintain under God, as they have down the ages, the most inflexible standards of personal honor."

This shows clearly the high principles which physicians utilize in the conduct of their professional activities.

Moles, Malignancies and Birthmarks

Birthmarks before puberty are rarely cancerous but after this age, any skin lesion which changes its size and characteristics may be malignant, according to Dr. John Charles Long, Jr., director of the surgical services at the Plainview Hospital and Clinic Foundation in Plainview, Texas.

The most common type of birthmark is the pigmented mole or *nevus,* which may be brown, blue, or black. "The brown nevus usually does not undergo cancerous change after puberty. They can be left alone unless they become irritated, or if they appear unsightly. Blue or black nevi may be malignant lesions. For this reason, they are often called 'black cancer' and generally, they do not grow hair. The nevi which grow about the external genitalia or on the feet are considered dangerous. To remove these moles during childhood eliminates the danger of cancer developing."

Biopsy of suspected birthmarks involves surgical removal of the lesion including a small margin of normal tissue which avoids opening the birthmark during the operative procedure. Microscopic study of the biopsy will indicate whether or not cancer is present.

If cancer is present, surgery is usually indicated as many lesions are resistant to x-ray therapy while others are not cured by "burning-off" or cauterization procedures. Non-cancerous lesions may be treated by many methods such as sandpapering techniques, tatooing with pigment matching adjacent skin areas, carbon dioxide snow, but rarely by x-ray therapy which carries the danger "that a cancer lesion may develop following radiation therapy."—J. C. Long, Jr.: Are birthmarks malignant? *American Journal of Nursing,* 55:955 (Aug.) 1955.

Three Beginnings, Founded in the Faith

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THE three beginnings were evenly spaced across the past 100 years. They were modest beginnings, but they were rich in promise, because these beginnings were "founded in the faith": a faith in future achievement.

The first of these three beginnings was the University of Minnesota. A century ago the University was in its infancy, aged four. The popular term "lusty infant" was decidedly inappropriate. Bane eclipsed blessing in the early years after the enrollment of the first twenty students in the autumn of 1851.

The *St. Anthony Express,* a newspaper oratorically staunch in support of the newcomer, was constrained to be frank. It not only urged those who would "see this infant institution have a rapid and healthy growth. . . (to) rally around it. . ." but it also pronounced dismally that the school, "as yet, has no funds to sustain it."[1]

True, there was a building of three stories—including the basement—costing $2,500. True also, there was income from tuition, graded according to the level of advancement of the courses, and ranging per student, per quarter, from $4.00 to $6.00. And finally, with rather more conviction than architecture or finances provided, it was reiterated that the scene of these operations was one of charming natural beauty. The building lay "only a few rods from the Falls. . . the romantic beauty of whose banks surpasses that of the Hudson."[2]

Whatever promise or consolation was provided by these modest assets, the institution was beset with afflictions, some of which may be enumerated:

The donor of the land upon which the school building stood repented his folly in yielding to the temptations to support higher education and reclaimed the property into the realm of profitable business real estate.

The professor—and the only one in the institution at the time—resigned, because he found he had no fixed salary, but was expected to survive on students' fees, supplemented with income from lectures to the public. A chancellor of the time complained bitterly of the insufficiency of stamps and stationery available to him.

Much of the land which had been voted by the Congress of the United States to the institution for its support and endowment could not be acquired, because the hostile settlers hoped they themselves might some day possess this government land.

A drought prevented floating to market the timber which had been cut on such lands as the school did secure.

Public subscriptions for the support of the institution suffered dreadful delay and decimation in the payment.

Bonds in the sum of $40,000 issued against the University's land holdings upon authorization of the state legislature interested but few takers.

A country-wide financial panic (1857) spread ruin and despair, and "Never since Minnesota was discovered has money been so scarce. . ."[2]

Through such dark clouds of gloom and foreboding, there arose the first building on the present campus, called "Old Main." But then, as now, physical structures alone could not maintain the University. Few were the students who entered Old Main, until the complete collapse of the ill-fated enterprise with the outbreak of the Civil War. Of Old Main, the *St. Paul Pioneer and Democrat* then said: "The whole building is going to ruin. . . all the doors being open and the snow drifted in. . . it melts on warm days through the floors. . . . That's the way the money goes."[3]

This was the beginning which did not die be-

Convocation address, University of Minnesota, Minneapolis, July 14, 1955.

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cause it was a beginning founded in the faith, founded in a resolve which was sometimes brash, seeking to "put Harvard in the shade,"[4] founded in a determination not always characterized by academic restraint, as when it was declared that, "God evidently intended Minnesota as the masterpiece of all his works, the axle on which the Union should turn, the sun of our Federal system, between which and New England there could be no other comparison than between Hyperion and a Satyr."[5]

Although sometimes brash and sometimes unrestrained, this faith in future achievement was strong and enduring. It possessed and inspired John Sargent Pillsbury, "the father of the university," who, more than any other man, was responsible for restoring the defunct institution, after the Civil War, to its long rich life. Commencing as a creditor of the University, to the extent of $5.50, "for locks, nails, and iron," Pillsbury guided the fortunes of the University in its early adolescence, in his varied capacities as hardware merchant, organizer of volunteer regiments for the Civil War and Indian uprisings, businessman, city councillor, regent of the University, state senator, international financier and governor of Minnesota.

It was Pillsbury's conviction that "There is no factor that can do more for the state and the nation than the University with its wide open doors ever welcoming all to enter."[6] It was this faith which has since inspired the great men of Minnesota: Folwell, Northrop, Vincent, Burton, Coffman, Ford, Coffey and President Morrill and the many others of today. These had no desire to "put Harvard in the shade," but they have led the University into the bright sun of scholarly pre-eminence.

The second of the three beginnings was in Rochester, Minnesota, a half-century ago. Here there was being evolved a project of medical education and research in a unique setting. As humble, indeed, were these beginnings as were those of the University fifty years earlier. A small but excellent library was begun and a laboratory of experimental medicine was opened. The laboratory occupied a portion of the barn of Dr. Louis B. Wilson, who later became the first director of the Mayo Foundation. The library and the laboratory, symbolic of medical education and research, were at the disposal of a small group of physicians who were the associates of the Doctors Mayo and temporary students at the intern or assistant level.

The origins of this group may be traced back to the time when the University found itself in a state of suspended animation. It was during the Civil War that Dr. W. W. Mayo took residence in Rochester and it was in that period that the two Mayo brothers were born.

During the summer of the year the elder brother returned home from medical school (1883), a tornado struck the city, an event of unusual importance for medicine and higher education in Rochester. Caring for the tornado casualties was severely hampered for want of a hospital. The Sisters of Saint Francis, who functioned as nurses in makeshift emergency quarters, determined that Rochester needed and should have a hospital.

Nature's role in the development of the University in Minneapolis had been gentle, confined to providing what an early enthusiast called "one of the most beautiful sites in the Union for the establishment of a university."[1] Nature's role in the Rochester beginnings was grim and harsh. Dean Theodore Blegen recounts that his father, a resident of Rochester at the time, referred to the tornado as "one of the most terrible storms in the history of the world."[7] Ill though this wind was, a great good emerged as St. Mary's Hospital, probably the largest of its kind in the world today. St. Mary's Hospital is staffed by the faculty of the Mayo Foundation, and is a major teaching hospital on the Rochester campus of the University.

By the turn of the century—fifty years ago—the embryonic beginnings of the Mayo Foundation for Medical Education and Research were taking form. The Mayo brothers had already decided upon a plan "to put aside from our earnings any sums in excess of. . . a reasonable return for the work we accomplished. . . . Each member of the staff received a salary which was sufficient to permit wholehearted attention to his work. There was no profit sharing—accumulations over and above the amount necessary for the purposes I have outlined were conservatively invested. . . ."[8]

This was a beginning, an idea of two doctors to save money, but it was a beginning founded in the faith, in the conviction that "The people's money, of which we have been the moral custodians . . . (should be) returned to the people"[8] through medical education and research.

Later, Dr. W. J. Mayo was able to say that the fund accumulated "had grown far beyond our expectations." He reiterated the principle that this money "had come from the sick, and . . . ought to return to the sick in the form of advanced medical education, which would develop better trained physicians, and to research to reduce the amount of sickness."[8]

The brothers Mayo were certain that the ends they desired could best be achieved through the University of Minnesota. Instruction of young physicians and provision of facilities for research at Rochester should be part of the University.

Parallel with these Rochester beginnings, men of vision at the University campus charted a new course in graduate medical education. President George Edgar Vincent bemoaned the fact that success in medical practice as a specialist depended almost entirely upon native ability rather than systematic training, since virtually no such systematic training existed anywhere. President Vincent and graduate-school dean Guy Stanton Ford saw in the collaborative enterprise in Minneapolis and Rochester the possibility of " a graduate school which will stand absolutely alone in the sphere of medical education in America today."[9]

President Vincent's dream of systematic training of specialists in medicine, made reality by the efforts of Dean Ford and Mayo Foundation director, Louis B. Wilson, in Minneapolis and Rochester, anticipated a major development in the training and identification of specialists in this country. There now exists a group of experts in each special field of medicine from anesthesiology to urology, who publicly certify to the training and competence of physicians desiring to function as specialists.

Prior to the employment of this concept, any medical-school graduate could declare, for example, "I am a pediatrician." Neither his colleagues nor the public could readily verify or refute his claim. Today the public and the medical world at large can readily verify such a claim, as a consequence of general acceptance of President Vincent's principle that, to be a medical specialist, systematic training must supplement native ability.

Now these beginnings have come to full fruition; they were the humble beginnings of a laboratory in a barn, a handful of physicians studying and practicing surgery, a resolve that medical earnings are a public trust, a determination to establish high standards of graduate medical education.

The faith in which these beginnings were founded has come to great and good substance in the medical departments of the graduate school of the University of Minnesota. This school, on the Minneapolis and Rochester campuses, is by far the largest of its kind in the world. As to its quality, we may safely assert that it is of a high order.

The third beginning is 1955, a century since the University's beginning, a half-century since the early beginnings of the Mayo Foundation. The third beginning is now—this commencement.

You have inherited knowledge and learning, which will melt away as did the snow through the floors in the ruins of Old Main, unless the course you set follows the pattern of a beginning founded in the faith, a faith in future achievement, in your own future achievement.

Benjamin Franklin was wont to ask the question: "Do you love truth for truth's sake, and will you endeavor impartially to find and receive it for yourself, and communicate it to others?"[10]

In the course of your life at the University, you have learned to love truth for truth's sake, as indeed you must also have learned to love beauty for its own sake, in your participation in the total rich life of the University. Let there be the further determination, with Franklin, to continue impartially to find truth and receive it. Few of you will search out truth as scientists in research laboratories or as scholars in University libraries. But all of you must continue to seek the homelier truths which guide men and women of good will to be good citizens, good neighbors, and perhaps most difficult and most important, good parents.

To Franklin's inquiry, "Will you endeavor to communicate. . . (the truth) to others?" you may most effectively answer by an abiding loyalty to the University, as citizens who speak out in its behalf and as alumni who give of your means to further the University's work. You may be unaware of the extent to which you have already contributed to the vitality of the University. As a questioning student you have stimulated your instructors to more incisive thinking. As an interested student you have provided inspiration for wider learning and better teaching.

Your continued support of the University is not in payment for value received, but an extension of the mutually stimulating student-teacher relationship to that of a citizen-University alliance. In so far as you identify yourself with the University in the future, the scientific discoveries in her research laboratories and the voices in the classroom will be yours in part. You will be continuing to find truth and communicate it to others.

I would wish for all of you who graduate tonight the courage and the strength which sustained those who, a century ago, sought a state University on lands still belonging to the Indians, and those who, a half-century ago, started a laboratory of experimental medicine in a barn.

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COBALT AND THE THYROID

Due to the previous appearance in the JAMA of a communication suggesting that a cobalt salt could have produced a reversible hypothyroidism, particularly in infants with sickle cell anemia,[1] the editor has in fairness published four papers embracing animal and human studies,[2] and indicating the nontoxicity of cobalt with reference to the thyroid. Klinck,[3] of the Armed Forces Institute of Pathology, reviews the findings of ten cases of thyroid hyperplasia in young children. Five of these had received cobaltous chloride and ferrous sulfate. The microscopic picture in both groups was quite similar. The author stresses the importance of using caution in ascribing the changes noted to any one etiologic factor, inasmuch as the histologic picture of hyperplasia may result from many causes. On the basis of his observations, he concludes that goitrogenic activity of cobalt cannot be established. Further studies are indicated. Holly,[4] of the Department of Obstetrics and Gynecology, University of Nebraska College of Medicine, conducted a controlled investigation on 227 pregnant women, plus their offspring, and in addition studied the effect of cobalt in animals. There was no evidence of toxicity in the seventy-eight women who received cobalt alone or with iron, and all children resulting from these pregnancies were normal. No thyroid enlargement was encountered. In the animal investigation, no demonstrable histologic change was noted. The author's findings substantiate the absence of any goitrogenic effect of cobalt. The significant effect of cobalt plus iron in pregnancy is the maintenance of normal hematologic values in nearly 100 per cent of the patients. Jaimet and Thode,[5] of Hamilton College, McMaster University, Hamilton, Ontario, conducted extensive thyroid function studies on seventeen children receiving Roncovite in liquid form. No thyroid enlargement was seen in any case, nor did clinical hypothyroidism develop. The authors conclude that cobalt in amounts up to 6 mg. per kilogram daily for ten weeks does not affect any phase of thyroid function as measured by the radioactive iodine tests, and has no goitrogenic action. Scott and Reilly,[6] of the Department of Radiology, University of California School of Medicine, and Veterans Administration Hospital, Fort Miley, investigated the effect of cobalt administration on various functions of the thyroid in rats. The usual hematopoietic action was noted, and the incidental observation was made that growth of a transmissible fibrosarcoma was inhibited. There was no inhibition of I^{131} uptake, thyroxin synthesis, hormone release or circulating hormone levels. The authors conclude that the addition of cobaltous chloride to the drinking water of rats in 60 mg. per kilogram daily dosage had no appreciable effect on iodine metabolism. Similar results were obtained with an iron-cobalt mixture.

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Inversion of the Uterus Immediately Following Delivery

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THE condition of inversion of the uterus occurring immediately postpartum, in which the fundus uteri inverts through the relaxed cervix and presents itself in the vagina or at the introitus, manifests itself by hemorrhage and by shock out of all proportion to the amount of visible hemorrhage.

The incidence is reported variously from one case in 760 deliveries to one in 16,000 deliveries. In the past ten years at Swedish Hospital, the following case of inversion is the only one noted in over 25,000 deliveries.

In 1940, Das reviewed the literature and found 297 cases, with a mortality of 13.2 per cent. Since 1940, the following reviews and reports have been found:

	Cases	*Deaths*
Bell, Wilson and Wilson	80	14
Quigley	14	4
Loizeaux and Mastraioinni	4	0
	—	—
	98	18 (18%)

The early recognition and treatment of the condition leads to better results as follows:

	Cases	*Deaths*
Diagnosed and treated within one hour	56	7 (12½%)
Diagnosed and treated after one hour	42	11 (25 %)

Case Report

Mrs. P. E., aged twenty-three, gravida I, Para O, delivered spontaneously after nine hours of labor, a living female infant, persistent occiput posterior. The placenta was delivered spontaneously without traction on the cord, and only slight pressure on the fundus, after twelve minutes third stage, at 1:55 p.m., January 14, 1955. Episiotomy was repaired under local anesthetic, but bleeding persisted and increased (Dr. Duryea).

The following sequence of events ensued.

2:20 p.m.	1000 cc. 5 per cent glucose in water with 2 ampules pitocin added, started I.V. (Dr. Donald Larson)
2:40	Blood pressure 50/0
2:48	500 cc. blood started with pitocin 0.5 cc. added. Patient redraped, examined by Dr. Hauge—inverted uterus found.
3:05	Second 500 cc. blood started. Blood pressure 60/0, estimated blood loss to date—750 cc.
3:25	Blood pressure 116/? Cyclopropane started. Fundus replaced by Dr. Hauge and held with fist in fundus.
3:35-4:15	Blood pressure gradually stabilized at 110/60 while blood and intravenous glucose with pitocin added were run in rapidly—fundus held by Dr. Hauge—patient under cyclopropane.
4:15	Pitocin 0.5 cc. intravenous. Cervix still fully dilated, fundus beginning to contract (Dr. McKenzie) about 500 cc. clot removed.
4:22	Pitocin 0.5 cc. intravenous—Fundus contracting better. Cervix fully dilated.
4:34	Ergotrate grs. 1/320 intravenous.
4:40	Anterior lip of cervix forming again but cervix still 6-8 cm. dilated, fundus firm. Fist removed from inside fundus.
4:50	Cervix admits one finger only—fundus firm. No bleeding. Blood pressure 112/70. Pulse 84.
5:15	Patient awake and talking.
2:48-5:30	Patient received 3000 cc. blood and 1000 cc. 5 per cent glucose, 4 cc. pitocin and grs. 1/320 ergotrate intravenously.

The patient made an excellent recovery. Temperature reached 103 degrees on the second and third days postpartum, but was normal thereafter. She was unable to void for several days but excreted about 1000 cc. urine daily. Hemoglobin 12 grams on January 22, 1955, eighth day postpartum. Mother and baby were discharged in good condition on eighth postpartum day.

Comment

The early recognition and treatment of the condition, with the availability of large amounts of blood for the treatment of the profound shock, undoubtedly saved this patient's life.

1. By using the fist as a tampon and molding the atonic uterus on the fist, the bleeding was fairly well controlled, and repeated inversions were prevented. As long as the cervix remained completely dilated and flaccid, this procedure appeared to us to be more feasible than uterine packing. As soon as the cervix reformed (and

(Continued on Page 711)

From the Obstetrical Department of Swedish Hospital, Minneapolis, Minnesota.

Death in Utero

An Unusual Case

JAMES WARREN McGILL, M.D.
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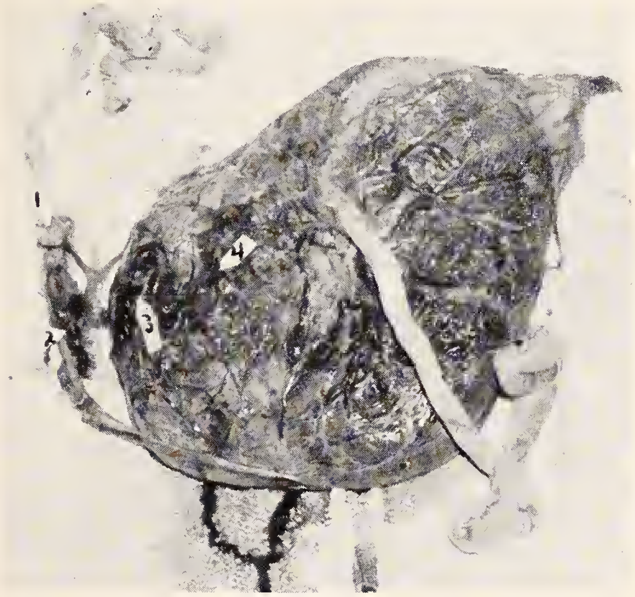

Fig. 1.

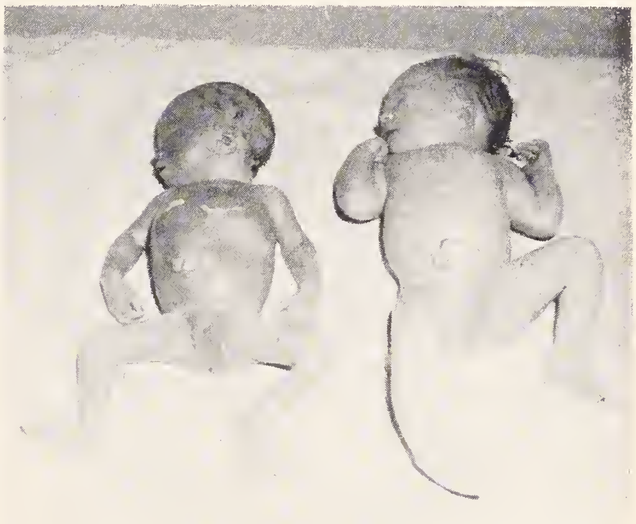

Fig. 2.

The patient, a white woman, aged forty, para ii, gravida iii, entered the hospital at 10:30 p.m. on August 2, 1954, in labor and at term. Labor had begun at 10:00 p.m. Her membrane ruptured spontaneously at 10:45 p.m., and at 11:10 p.m. a seven-pound girl was born normally; the cord was tied and cut. Pressure on the fundus caused the breech of the second twin to engage.

The membrane was ruptured artificially, and the frank breech was born; delivery was completed by gentle traction in the groins. The delivery of the fetus had been easy. The head was very cyanotic in appearance, and the skin of the abdomen was macerated. The cord was tied and cut and the placenta separated spontaneously and presented itself in the vulva, and was easily expressed.

It was noted that there was but one placenta and that the attachment of the cord of the second twin to the placenta was almost thread-like. Closer examination revealed that there was almost complete tearing of the cord at the site of its placental attachment.

As noted in the first illustration, the arrow numbered 2 indicates the torn end of the cord with a firm thrombus protruding from one of the cord vessels. The arrow numbered 3 is the site of the attachment of the cord to the placenta. A long, firm thrombus extends from a vessel there.

The area between arrows 1 and 2 indicates a firm thrombus that extends from the torn end of the cord for about 1½ inches up the cord. The area between arrows 3 and 4 indicates a similar firm thrombus extending from the site from which the cord was torn, along the cord vessels on the surface of the placenta.

That the tear or rupture of the cord was not recent is indicated by the fact that the amniotic fluid was not bloody. Also, the condition of the skin of the fetus indicated that death had occurred several days previously. The mother was questioned as to whether she had sustained any falls, blows to the abdomen or other injury, but said she had not. Neither did she notice anything unusual at the time that the fetus would have died.

The second illustration shows the twins. The live one weighed seven pounds; the second one, five pounds, four ounces.

No explanation is offered for the occurrence; it is merely presented as an obstetrical curiosity.

Presented before the Minnesota Gynecological and Obstetrical Society, Duluth, Minnesota, April 30, 1955.

Current Concepts

PROPHYLAXIS OF RHEUMATIC FEVER

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There seems to be an apparent drop in the incidence of rheumatic fever. Whether this is real or is a cyclic affair, no one can tell as yet. If it is real, we shall have to search for an explanation of this phenomenon, and perhaps the use of antibiotics in the treatment of streptococcal infections may be the answer. It has been well established that rheumatic fever follows infections due to group A beta hemolytic streptococcus and one could reason that if there were fewer streptococcal infections, there would be fewer cases of rheumatic fever.

One factor in this picture would be the prompt, adequate therapy of streptococcal infections with penicillin. The general practitioner has been criticized because of what has been claimed a promiscuous use of penicillin, but if this were true it would result in one unmixed blessing and that is the early interference with streptococcal infections so that rheumatic fever does not result. In the case of the child with rheumatic fever, it has been stressed[1] that he be protected against his environment by the daily intramuscular administration of 400,000 units of penicillin, until such time as throat cultures are proven to be free from the streptococcus, or at least for ten days, if laboratory facilities are lacking. Then, the daily prophylactic use of 200,000 units of a stable penicillin product is recommended to further protect him during his hospital stay from doctors, nurses, visitors and patients who might be carriers.

This rather simple procedure itself has been reflected in our experience by a happy finding of the fact that the period of hospitalization is shortened in these days over what it was ten years ago. In 1942, the average hospital stay for rheumatic fever children in our group at the Children's Hospital in Saint Paul was ninety-five days. In 1952, it was twenty-five days. This, I am sure, is a reflection of the change from the polycyclic type of recurrent, flaming disease to a monocyclic form which comes to an end and does not flare up. One reason for this in our interpretation is the above-mentioned protection of the child while he is in the hospital. It is recommended by the Council on Rheumatic Fever[1] that children who have had rheumatic fever be given prophylactic, daily doses of a stable penicillin compound orally. The usual thought on this is 200,000 units daily. Many criticisms have been aimed at this program and the usual points made are as follows:

1. The child will become sensitive to penicillin.
2. The drug will no longer be effective against the streptococcus after such prolonged use.
3. Strains of streptococci will be produced which will be penicillin resistant.
4. The cost of the drug is prohibitive for continuous use.

These are very obvious criticisms, but they would have to be examined individually to determine their truth. With respect to the problem of sensitizing the child to penicillin by prolonged use of this drug, it has been our experience that if people are sensitive to penicillin, it is discovered very early in the use of the drug, and the drug must be discontinued. We have had no patients who have developed sensitivity over a period of years. The problem of lessened effectiveness of penicillin does not bear out in fact, and it has become obvious that the thousandth tablet is as effective as the first. The problem of penicillin-resistant forms of streptococci developing in the future is quite tenable. However, fortunately for us, this has not happened yet. Whenever it does, some other antibiotic will have to be placed in use.

The matter of the cost of the drug is a very real and very serious objection to its use. The cost for an average family with one child in this category is approximately $80 a year for continuous prophylaxis. When two or more cases in the family require prophylaxis, it is obvious that this is a real financial burden. This problem is real, but so far there seems to be no obvious solution. This matter of family expense has to be accepted the same as payments on the car, refrigerator, et cetera.

It has been suggested that sulfadiazine could be used as well and would be much cheaper, but I am sure that with the frequent visits to the physician necessary when the child is on sulfadiazine prophylaxis that the factor of economy is counter-balanced. Sulfadiazine also has the factor of being bacteriostatic rather than bactericidal and there is a very real opportunity for the child taking the drug to become a carrier and consequently a public health menace. A statistical analysis of a series using sulfadiazine and penicillin have also shown a definite preference for penicillin. The physician can practically reassure the parents that if they will cooperate in this matter, the child will not have a recurrence of rheumatic fever if a product is used which is stable in the presence of the gastric juices. Otherwise, the crystalline penicillin should be given at least an hour before breakfast so as to be sure and have a minimal amount of gastric acidity present. Whatever break-throughs have occurred have been credited to the fact that the penicillin was taken but it was taken at meal time and with a coexistent high level of streptococcal exposure. The protection level has not been high enough to withhold an invasion of the organism.

The question is frequently asked as to how long penicillin prophylaxis should be used. I would feel that this can only be answered individually and depends on the anticipated exposure to streptococci in the community. During childhood, the intimacy of contact is great and, therefore, the child is in a more hazardous position than an adult. It is our feeling that penicillin prophylaxis should be taken continuously throughout each year for the duration of the school career. After an individual gets a job or a girl becomes a housewife, the intimacy with streptococcus throats will be lessened, and the drug could be discontinued. Various positions, such as those in medicine and nursing, place the individual constantly in touch with exposure, and I believe these two groups should accept the problem of prophylaxis as a lifetime nuisance. When a girl becomes married and is a housewife or works at a clerical position, I would feel that prophylaxis could be discontinued until such time as her own children are school age, at which time they again will place a complete cycle of exposure on the mother. This is not quite so true of the father, whose contact with the children when they are sick is very minimal.

Summary

In summary I would feel that the present rather optimistic picture in the epidemiology of rheumatic fever is due, in large part, to the availability of a good bactericidal agent. It is unfortunate, perhaps, that we have to place so much confidence in one drug. However, until the whole mystery of rheumatic fever is better understood and some other agent or means of combating the disease is developed, the prophylactic use of penicillin as described above is our best therapeutic weapon.

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UNIVERSITY RECEIVES GRANT

A $59,650 Kellogg Foundation grant has been awarded to the University of Minnesota for further development of its course in hospital administration, James A. Hamilton, course director, has announced. This year's part of the grant—which is to be used over a three-year period—amounts to $21,450. The money will go into research work in hospital administration, integration of the on-campus and off-campus teaching program and preparation of textbook material.

Laboratory Aids

Sponsored by
The Minnesota Society
of Clinical Pathologists
George G. Stilwell, Editor

CYTOLOGIC STUDIES IN THE DIAGNOSIS OF CANCER OF THE LUNG

JOHN R. McDONALD, M.D.
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The cytologic examination of bronchial secretions and sputum has become a valuable method for the diagnosis of various types of carcinoma of the lung. It is a highly specialized procedure that, unfortunately, is not done in every laboratory. This procedure is of particular value in the diagnosis of primary bronchogenic carcinoma, although it sometimes yields positive results in alveolar cell tumors and metastatic malignant lesions of the lung.

Cytologic examination of sputum and bronchial secretions is one of four methods that can be employed to make the diagnosis of carcinoma of the lung. The other three are thoracic roentgenography, bronchoscopy and exploratory thoracotomy. Each of these four methods has a definite field of usefulness.

The rationale for the examination of secretions and body fluids in the diagnosis of carcinoma depends on the exfoliation of identifiable cells from malignant tumors that have access to a free surface. Malignant cells can be recognized in clumps, in clusters or singly by the fact that they have larger nuclei than do normal cells, irregularity in the size of the cells and nuclei, an altered ratio between nucleus and cytoplasm, hyperchromatism and large nucleoli.

The general principle of recognition of malignant cells in secretions was first recorded by Papanicolaou,[1] in 1928. The first study done on the recognition of malignant cells in smears of sputum was that of Dudgeon and Wrigley,[2] in 1935; they found cancer cells in 68 per cent of 58 cases of proved bronchogenic carcinoma. Barrett[3] and Gowar[4] later reported positive cytologic results in sputum in 68 and 64 per cent, respectively, of cases of bronchogenic carcinoma. Wandall,[5] in 1944, found cancer cells in the sputum in 84 of 100 cases of proved bronchogenic carcinoma. A study of bronchial secretions was made by Herbut and Clerf,[6] in 1946; they reported positive results in 82.4 per cent of 57 cases of proved bronchogenic carcinoma. They considered that examination of sputum was too time-consuming to be utilized. Papanicolaou,[7] in 1946, reported positive results from examination of sputum in 88 per cent of 25 cases of proved bronchogenic carcinoma. Woolner and I[8] examined sputum as well as bronchial secretions and washings and considered that all three types of material should be employed; hematoxylin and eosin were used for staining in this study.

Examination of sputum requires more time than does examination of bronchial washings because of the greater dilution of malignant cells in sputum than in bronchial secretions by other cells, such as phagocytes and leukocytes. However, sputum has the added advantage of being more readily obtainable than are bronchial washings. It is best to examine more than one specimen of sputum, three probably being the optimal number. Patients are instructed to produce sputum from deep in the bronchial tree and three or four good hawkings of sputum are all that is needed. The time of day at which the specimen is collected is not particularly important. The patient should spit into a glass sputum bottle containing approximately 30 cc. of 70 per cent ethyl alcohol.

Two methods of handling this material have been employed. The commoner method is to prepare smears, stain them with either Papanicolaou stain or hematoxylin and eosin and examine the preparations microscopically. Less commonly, the material is put in paraffin blocks and sectioned. Bronchial washings are handled somewhat differently. The solution of sodium chloride used to

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From the Section of Surgical Pathology, Mayo Clinic.
This is the thirty-second in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

wash the bronchus containing the suspected lesion is centrifuged and the centrifugate is smeared or placed in paraffin and dealt with as is sputum.

Because of the time required for the scanning of cytologic preparations, it has been found advantageous to employ trained technicians to do this part of the examination. The final diagnosis is made by the pathologist, of course. Since the procedure is so specialized, it takes a great deal of time to train a technician to do the scanning. The Minnesota Division of the American Cancer Society supports the training of technologists to do cytologic examinations of exfoliated cells for pathologists. This organization provides scholarships in exfoliative cytology for medical technologists who are residents of Minnesota, enabling the technologist to study for 3 months in an established center. The technologists are chosen by the Minnesota Society of Clinical Pathologists.

Cytologic studies for exfoliated cells done for suspected pulmonary lesions yield positive results in approximately 70 per cent of cases of bronchogenic carcinoma. Approximately 2 to 3 per cent of these results will be falsely positive. When patients whose sputa contain malignant cells undergo bronchoscopy, the latter procedure produces positive results in 55 per cent of cases. It would appear, therefore, that cytologic examination of sputum and bronchial secretions is a definite aid in the diagnosis of primary carcinoma of the lung. Because of the time-consuming nature of this procedure as compared with the examination of smears from the uterine cervix, it is probable that cytologic studies of sputum never will be used as a routine screening method for the detection of carcinoma of the lung. Rather, it would appear to have its usefulness for patients in whom there is a suspicion of carcinoma in the lung on the basis of a roentgenogram of the thorax or a history of cough, excessive amounts of sputum or hemoptysis.

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Note: A number of the author's references have been deleted in the interest of economy of space.

Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

PSYCHOMETRIC MEASUREMENTS

When the writer's seven-year-old niece was going to Itasca Park, she asked what "source" of the Mississippi meant. When informed, she said, "But I never thought the Mississippi began. I thought it always just *went*." With the marked emphasis upon testing and vocational guidance at secondary school or adult levels and the heavy demands for psychological services to courts and hospitals, the problems at the preschool and elementary levels where adolescent behavior originates are frequently overlooked.

Although children develop at different rates and vary greatly in their levels of maximum development, longitudinal studies show that in the areas of general intellectual growth and in progress toward social and emotional maturity, as well as in specific areas such as mathematical and linguistic achievement, there are *developmental sequences* which hold true. Interference with these natural developmental trends, either through serious education deprivation or through "forced feeding," as when a child is placed in a typical Grade I situation when his mental age is four and a half years, can lead to frustrations and tensions. Irregularities in patterns of development and behavior should be found and treated early.

A school psychologist can cooperate with school administrators, physicians, courts, and parent groups:

(a) by contributing to research and the public information program.

(b) by interpreting to schools, nurses, social workers, visiting teachers, and the courts the psychometric findings and trends.

(c) by helping parents use the developmental trends and mental status of children in educational planning.

(d) by early selection through verbal and performance scales of pupils for placement in state-aid classes for atypical children and in the home-bound teaching program.

(e) by applying specialized remedial techniques to pupils who achieve below their mental age expectancy levels.

(f) by referral to physicians suspected cases of organic, sensory, or physiological disorders and serious, emotionally disturbed cases.

It is apparent that the school psychologist serves in an important role in our complex educational system.

HELEN MIRIAM BISHOP, PH.D.
Public School Psychologist
Saint Paul, Minnesota

VENEREAL DISEASE STILL A PROBLEM

While the advent of the sulfa compounds and penicillin produced a profound reduction in syphilis and gonorrhea over the country and indeed the world, they are still not the complete solution. Cases still have to be found before they enter the picture. The year 1946, as the first post-war year, saw a general increase in morbidity of both diseases. In Minnesota, 400 more cases of syphilis were reported than in 1945. On the other hand, gonorrhea showed a decrease of more than 400 cases. Since that time, syphilis has continued to decrease steadily and very largely in the early category. Gonorrhea showed consistent decreases until 1953, when a 13 per cent increase was noted in reported cases and a further increase was noted in 1954. Infectious syphilis is unlikely to become a problem in Minnesota when we note that in 1953 only forty-nine early or potentially infectious cases were reported and only forty-three in 1954.

Conversely, every old case of syphilis must be suspected as cardio-vascular or neuro-syphilis, and every one should be given the most searching investigation and examination. On the one hand, disability with possible hospital expense faces the heart patient, and on the other, final admittance to a state hospital if clinical symptoms are found. Cardiovascular disease may be arrested by penicillin, and the earlier it is discovered, the greater the value of the treatment. Asymptomatic neuro-syphilis can be diagnosed only by examination of the spinal fluid, and there are still too many physicians failing to do this because the history is negative and there are no clinical symptoms, sometimes not even completely positive blood findings. A study of 135 cases of late and late latent syphilis reported in 1954 showed spinal examinations done in only fifty cases. The ratio should be much higher.

Positive serologic findings in blood and spinal fluid are still the most important single symptom of syphilis. Experience here with our multiple and repeated tests indicates that repeated completely positive findings are rarely false or so-called non-specific. One should be slow to decide against treatment in such a situation. On the other hand, it does not seem like "good medicine" to quickly decide that it is easier to give penicillin than to make prolonged and complete studies first, thus leaving the diagnosis forever in question.

One thing, of which Minnesota should be especially proud, is that in spite of no premarital testing law it is, I believe, the only state in the country to have a record showing that in four out of the past five years no live baby has been born with syphilis.

Intensive studies have been made to try to explain the increase in gonorrhea. The recognized increase in juvenile delinquency seems not related, as less than 10 per cent of cases occur in those under twenty. On the other hand, 638 cases out of 806 (in 1954) occurred in the twenty to forty group, and 339 among married, widowed, divorced and separated individuals. One might suspect a decline in moral attitude and a decreasing fear of infection due to the rapid and almost certain success of penicillin. The difficulty in securing prompt and adequate information on contacts is also a large factor. As a sample, a study of the month of January, 1955 indicated that in forty-four cases out of 104 reported, no contact information was given.

It is evident that although syphilis and gonorrhea are no longer a major health problem in Minnesota, a complete "fade-out" is still in the distance.

H. G. Irvine, M.D.

THE DEMOCRATIC WAY

Our democratic form of government is admittedly less efficient, more cumbersome and more costly than a well-run dictatorship. Yet few of us would be willing to settle for a dictator—even one who was all-wise and all-good, as well as all-powerful. We believe in freedom of choice—in government by the people.

It seems to us in the Tuberculosis Association that an effort to combine all charities in one gigantic fund drive is, in effect, an attempt to set up a charity dictatorship.

We all approve the Community Chest idea, as a voluntary banding together of welfare groups that find it beneficial to join in their fund-raising. But we question the wisdom of any effort to coerce *all* voluntary groups into a single fund-raising organization. As James E. Perkins, M.D., managing director of the National Tuberculosis Association, has pointed out: "He who controls the purse-strings also controls the program." Is it possible to select one group—in any state or community—that will be well enough informed on all health and welfare programs to determine each association's fair share of funds raised?

As for the matter of free choice, the average American has one or more "pet" charities. A father whose son has been crippled by poliomyelitis is eager to support the March of Dimes. A family that has seen one or more members suffer and die from tuberculosis will have a particular interest in eradicating that disease. Each individual wants the privilege of selecting the cause or causes in which he is vitally concerned. He finds little or no emotional appeal in a blanket request for support.

These are our general objections to an all-encompassing, fund-raising organization. As a Christmas Seal association, our three specific reasons for not joining Community Chests are:

First, the Christmas Seal, now half a century old, is recognized as the international symbol of the fight against tuberculosis. For hundreds of thousands of Americans, the use of Christmas Seals has become a holiday season custom.

Second, The National Tuberculosis Association depends for its support, not on large contributions from a few, but on a little bit from the many. Funds are raised, not by personal solicitation, but almost entirely through the mail. An individual receives Christmas Seals and a letter of appeal in his own home and decides for himself whether or not he wishes to support the program. There is no pressure from a solicitor or from an employer. Any contribution is purely voluntary.

Third, the Christmas Seal sale is more than a fund-raising campaign. It provides our best opportunity to reach all people in every community with facts about tuberculosis. The campaign period is a time for most effective all-out education through newspapers, radio, television, in schools, in churches, and at organization meetings.

The doctor is the first line of defense against tuberculosis, but he cannot do the job alone. Health education is a major weapon. Tuberculosis is a public health problem, and an informed and alert public is essential to defeat this age-old disease.

E. A. Meyerding, M.D.
Minnesota Tuberculosis and Health Association

THE CONSULTING PSYCHIATRIST "SPEAKS"

In 1954, out of a total of 1,758 referrals made to Family Service of Saint Paul, Minnesota, a Community Chest agency, only thirty-three (1.8 per cent) were instigated by physicians. How might this significantly low number be explained? Why this serious and perhaps appalling lack of communication between two supposedly closely allied professional groups?

There is undoubtedly a variety of reasons. I should like to comment on the two that I consider most important and most worthy of consideration.

The first revolves around the simple but pregnant fact that doctors by the very nature of their orientation tend to refer their patients to other doctors. There is a definite but natural hesitancy on the part of many physicians to share the confidences of their patients with others outside the medical sphere. In order to maintain and uphold medical standards, we have adopted a strict and rigid code of medical ethics which acts as a strong deterrent and prevents us from more active participation with our fellow workers in the field of human welfare. In our partial isolation, we may be unaware that social agencies no longer play the part solely of "relief workers," doling out food, shelter, clothing and money to the indigent. The social service worker of today is a person trained in the art of dealing with his fellow man on both a psychological and sociological level, a person striving for complete professional status and entitled to it. In this connection, interested and responsible individuals around the country are now beginning to deal with this issue by presenting bills to their state legislatures, requesting formal licensure as professional workers.

This brings me to the second reason for the apparent chasm existing between the physician and the Family Service agency. The doctor may be unacquainted with what a social agency has to offer. The question is often asked of me, "How can the medical profession best utilize a Family Service agency?" What Family Service has to offer will be answered in another editorial. Suffice it to say that in addition to financial consulting and legal advice, a whole host of problems centering around marital difficulties, family relationships and child behavior come under its jurisdiction. Intertwined social and psychological perplexities that can be handled on a casework level ought to provide occasions for worthwhile referrals from the physician to the social agency.

How may a closer relationship be effected between practicing physicians and social agencies? This can be accomplished only if these two groups learn to understand, to trust, and to respect each other. It must be thoroughly and completely comprehended that our goals are identical, the health and welfare of the individual. When we are able to recognize our mutual responsibilities, and this time hopefully is not far off, this realization will allow appropriate and mature communication between physician and social agency, redounding to the benefit of all.

Philip M. Margolis, M.D.
Consulting Psychiatrist
Family Service of Saint Paul

THE FUNCTION OF THE HILL FAMILY FOUNDATION

The Hill Family Foundation was established in 1934 by the late Louis W. Hill and was sustantially enlarged following his death in 1948.

Mr. Hill believed that the purpose of a foundation, as set forth in its charter, should be broadly stated to permit adaptation to the chang-

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This is the second in a series of editorials on Family Service.

ing conditions inherent in perpetual existence. Therefore, the objectives of this Foundation are stated as being "for educational, scientific and benevolent purposes which contribute to the public welfare in such a manner as shall, to its Board of Directors, seem best."

The present policy of the Foundation became effective in 1950. Its program is not limited to any one field, but rather to the support of research and experimental projects which will have a significant effect within their fields and for which there is not general support.

The Foundation's objectives are: to pioneer and assist pioneer organizations in the fields of science, welfare and education; to assist in the development of the efficiency and correlation of existing programs which have public acceptance and support; to aid in the development of new bodies of knowledge and to encourage the utilization or correlation of existing bodies of knowledge; and to support projects directed toward the development of improved liaison between research and practice in the broad field of human welfare.

The Foundation is a grant-making organization, contributing only to projects related to established, non-profit organizations and agencies. It considers its geographic area to be the Upper Midwest, and priority in consideration is given to projects and programs of special value to or originating in this area. Contributions are not made to general operating budgets, annual financial campaigns, capital fund campaigns, or to religious programs of any particular denomination or sect. To maintain flexibility, the Foundation prefers to make grants for periods of less than five years.

In the field of health and the medical sciences, the Foundation is chiefly interested in basic or fundamental research. A goodly portion of its grants in this field are for projects and activities conducted under the direction of The Medical School, University of Minnesota. However, grants also are made for meritorious projects sponsored by other health and medical organizations which can demonstrate that they have the qualified personnel for direction and supervision of the proposed research or experimental undertakings.

A. A. Heckman

COLLEGE HEALTH SERVICES

Almost 300 years ago, John Locke, philosopher and physician, in an essay on "Some Thoughts Concerning Education," said: "A sound mind in a sound body is a short but full description of a happy state in this world. He that has these has little more to wish for, and he that wants either of them will be but little the better for anything else." This simple statement, though 300 years old, expresses very well the philosophy underlying the health programs in American universities and colleges.

The total number of young people in colleges and universities in America today is over two million and growing rapidly. The late Dr. T. A. Storey, more than thirty years ago, said: "Among all the subtle forces that mold public opinion and direct its attitude, the most powerful in its possibilities resides in that small group of men and women made up of these citizens who have received a college education and have learned how to use it. These one-time students and graduates constitute a significant major portion of the better educated parents and the men and women who become political leaders or leaders of public thought and action along other lines." The college graduate, as a member of a special group in which society has invested heavily, should become a leader not only in his own specialized field, but also in those areas in which family and community health may be conserved.

The main purposes of college health services are educational and preventive because they are a part of educational institutions. Students must learn what to do in event of acute illness when away from parents or family physician, where to seek competent medical care, and how to discriminate in matters of diet, activity, rest and other principles of healthful living. They must find sound sources of medical information and must learn how to use this counsel in the solution of their own physical and emotional problems. The college physician has a unique opportunity to teach students the importance of good medical care, where to obtain it, as well as advise on personal health problems of the student.

Tuberculosis case-finding programs in colleges are one example of the preventive and educational aspects of college health services. Twenty years before the idea of community mass x-ray surveys became popular, colleges and universities throughout the country were using this method of tuberculosis case-finding. Under the leadership of Dr. J. A. Myers, the program of routine tuberculin-testing and chest x-ray was developed in the colleges. Not only were early cases of tuberculosis discovered by this method, but students learned the meaning and importance of the tuberculin test and the need for periodic chest x-rays of those reacting to tuberculin.

The health examination required in most colleges is another educational tool. The student who has a periodic medical examination as an undergraduate will learn the value of such periodic examinations and continue them in his later years.

Another function of the college health service is similar to that of an industrial health service. By provision for the care of minor illnesses, emotional and physical, as well as the teaching of good health habits and disease prevention, much academic loss of time can be prevented.

A balanced health program as provided in the majority of colleges and universities today, including periodic health examinations, tuberculosis case-finding programs, health care and education in preventive medicine, should do much to prepare the college student to exercise the leadership needed to assure the best medical care for his family and community in the future.

Ruth E. Boynton, M.D.
Students' Health Service
University of Minnesota

POLIOMYELITIS VACCINATION IN MINNESOTA

Vaccination with Salk vaccine of persons in the priority groups in Minnesota under the Poliomyelitis Assistance Act of 1955 will be largely through private physicians in their own offices. This pattern of administration of vaccine follows the pattern that has been long established in this state for the administration of immunizing agents.

For many years the protection of susceptible populations against such communicable diseases as small pox, diphtheria, or whooping cough has been carried out almost entirely through physicians in their own offices. Both your society and the state health department are of the opinion that immunization against poliomyelitis can and should be handled in the same manner as are biologics for other immunizations.

Acceptance of the Minnesota plan by the medical profession and others is recognition of the fact that physicians in this state recognize and accept a basic responsibility for the practice of both preventive and curative medicine.

The plan reaffirms the confidence the public has in the high standard of medical practice in Minnesota. The public accepts the concept that every physician will do all within his power to make certain that within his own community the vaccine will be available only to eligible persons in the priority groups.

Both your society and the state health department believe that the plan is the best possible procedure for this state that could be devised within the provisions of the federal act.

The state plan was designed to provide the greatest possible amount of vaccine for priority groups through public funds with the least expenditure of tax monies. Although the Poliomyelitis Assistance Act provides that up to 20 per cent of allocated funds may be used for administration of the vaccine, all funds available to Minnesota will be spent for the purchase of vaccine. Administrative costs will be paid out of funds made available to the state health department under federal grants for public health purposes.

In the meantime, it is essential that all physicians observe the restrictions outlined in the state plan. The co-operation of physicians in other aspects of the poliomyelitis control program this year is ample evidence that the present program will achieve a successful conclusion. The National Foundation for Infantile Paralysis program for the immunization of first and second grade children has been carried out with real credit to the various community organizations. The medical profession is to be complimented on its splendid co-operation in this program.

The profession also has established an outstanding record for its active support of the poliomyelitis surveillance program carried on this year by the state health department. The reporting of cases, and the submission of information, stool and blood specimens from patients has contributed considerable data that will further efforts to control this disease.

Minnesota has been fortunate that the present poliomyelitis season has been a relatively light one. It is also encouraging to report the apparent protection given to susceptible persons by a single dose of Salk vaccine.

Both your society and the state health department are looking forward to the time when sufficient vaccine is available to provide at least one immunizing dose of Salk vaccine to all persons in all priority groups. At this time, with the vaccine in short supply, all of us have a basic obligation to use what vaccine as is available for those persons in the first priority group, namely, from birth to nine years of age and pregnant women.

You can be assured that every effort will be made to keep both the medical profession and the general public fully informed of all current developments that pertain to poliomyelitis and the vaccine program.

R. N. Barr, M.D.
Minnesota Department of Health

INVERSION OF THE UTERUS

(Continued from Page 700)

this happened shortly after the first use of ergotrate), the fundus was firm and there was no evidence of reinversion.

2. From the published reports, it appears that acute puerperal inversion occurs within thirty to sixty minutes postpartum. We urge, then, that the fundus should be watched for the first hour after delivery, preferably with the patient in the delivery or recovery room.

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EX-CONVICT MASQUERADING AS DOCTOR OF MEDICINE SENTENCED FOR BIGAMY

Re: State of Minnesota vs. Charles A. Allen.

On August 23, 1955, the above-named defendant, thirty-six, 1405 Yale Place, Minneapolis, was sentenced by the Hon. Paul S. Carroll, Judge of the District Court of Hennepin County, to a term of not to exceed five years at hard labor in the State Prison at Stillwater for the crime of bigamy and having three prior felony convictions. Allen previously, on August 18, had entered a plea of guilty to the bigamy charge before Judge Carroll, and immediately before being sentenced he also pleaded guilty to an information charging him with the prior convictions. This information charged that Allen had been convicted of the crime of forgery on August 14, 1949, in Portland, Oregon, and sentenced to a term of from twelve to fifteen months; that on January 31, 1952, he was convicted of forgery in the first degree at Montesano, Washington, receiving a sentence of not to exceed twenty years, and that on August 24, 1953, he was convicted at Mohall, North Dakota, for obtaining money under false pretenses and given a two-year sentence.

Allen, who was born at Cheyenne, Wyoming, in 1919, although working as an orderly at a Minneapolis rest home, represented himself to the operator of that home and her daughter as being a doctor of medicine and having at one time been licensed to practice in Colorado. The Minnesota State Board of Medical Examiners was requested to determine whether Allen actually was a doctor of medicine or whether he was licensed to practice in any state. Extensive inquiry by the Board failed to disclose that Allen had ever held a doctor of medicine degree or a medical license. The defendant subsequently married the daughter of the operator of the rest home, despite the fact that he had previously been married in Vancouver, Washington, on January 7, 1949, and had not obtained a divorce. Allen was arrested on August 10, 1955, by Minneapolis police, and on the following day his wife signed a criminal complaint against him which resulted in the conviction for bigamy.

PROLONGED PREGNANCIES

There is no particular danger to the baby when pregnancy extends beyond the customary nine months, according to Dr. S. Kolonja of Klagenfurt who reported a series of 8,000 deliveries to a meeting of the Austrian Society for Gynecology and Obstetrics. "Waiting for spontaneous delivery is advisable. When no measures were taken to induce delivery, no fetal death that could be ascribed to the prolongation of pregnancy occurred, but fetal mortality and the need for operative intervention was multiplied several times when delivery was induced by means of drugs." However, it is necessary to differentiate between true prolongation of pregnancy and a belated delivery due to other factors.—Foreign Letters: *Journal of the American Medical Association,* 158:1456 (Aug. 20) 1955.

President's Letter

COUNTY MEDICAL SOCIETY ACTIVITIES

Following the summer lull, all county medical society officers and committeemen are, or should be, busily engaged in outlining a program of activities for the county medical society for the ensuing year. County society programs should be educational in nature, but the educational features should not be limited to scientific subjects alone, but should have at least equal voice with economic and social problems which face the profession and the public as far as medicine is concerned.

It would be of great value if the president of each county society could assign a subject to a panel of members for discussion at some future date which could bring out all the various facets of the numerous problems which face medicine, not only in scientific, but in the social and economic world. Your state officers have repeatedly emphasized the importance of a good indoctrination program for new members, and a re-indoctrination program for older members who seem to have lost touch with medical organizations.

Subjects of immediate and consuming importance are numerous, and many of our society members are willfully ill-informed or misinformed concerning immediate economic problems facing medicine. Subjects which lend themselves well to panel discussion may be some of the following: union health and welfare plans; lay-sponsored medical care plans; social security for physicians; the Bricker Amendment; the role of employer and employee in industrial or occupational medicine; problems of civilian defense, and Minnesota's part in the overall program; and hospital and physician relationships. The above are only a few of the important subjects for panel discussions that would be highly informative.

We often hear members say that they will not go to a county medical meeting if politics are going to be discussed. Pure science seems to be their forte. Far be it from me to belittle the scientific part of the program of any county medical society, but scientific medicine may well suffer untold damage because a physician has been unwilling to acquaint himself with the economic and social pressures which will inevitably affect the practice of scientific medicine in the future.

Every county society, no matter how small, should have an excellent grievance committee to handle local-level problems and complaints against member-physicians. A well-functioning committee, known to the community, can do a great deal to aid good medical-public relations, and can be an effective link between the profession and the people it serves. Many small grievances, considered and discussed thoroughly and sincerely on a county basis, can be halted or solved, thus preventing misunderstandings from becoming larger and more difficult to resolve satisfactorily.

Likewise, a good, active public relations committee can be a decided asset to a county medical society. This committee, working in conjunction with all other county society committees, can be the center of many activities, coordinating them for a smooth-functioning society, whose work fosters improved public relations.

I have little patience with the doctor who will tell you that he has no time to spend for county medical society activities, particularly if they concern the economic welfare of the profession and the people whom he serves. It is the duty of every doctor, not only to acquaint himself with the scientific advances of medicine, but also to be well informed regarding the economic problems of his profession. As a recent publication on medical ethics so aptly puts it, "Is your halo on straight? Are you seriously and earnestly attempting to inform yourself on both the economic and scientific problems of medicine? If not, let's straighten your halo."

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

SECRETARY INDICATES CHANGE OF HEART

Secretary of the Department of Health, Education and Welfare, Marion B. Folsom, has recently indicated that after a thorough study of the Administration's reinsurance proposal, called a mainstay of the Administration's health proposals, a change of heart may take place. According to recent newspaper reports, Mr. Folsom is not so convinced as his predecessor Mrs. Hobby was, that the plan is a good one for this country.

A recent newspaper story states that Mr. Folsom is "taking a new look at the whole business of health reinsurance."

He is quoted as stating:

> "We haven't yet formulated a health program so far as this department is concerned. I am studying the whole health reinsurance idea, part of which was new to me. I have to make a decision soon so as to make recommendations to the President for his next State of the Union speech."

Folsom commented that there evidently had been little luck in getting Congress interested in this plan (reinsurance).

He continued:

> "The people who want compulsory federal health insurance say this reinsurance won't do the job, that it won't reach the lower-income people and that it is inadequate. And the doctors shy away and don't want the federal government to do anything."

Mr. Folsom also gave his personal philosophy on the subject of continuing and catastrophic illness. He said he favored the utmost use of voluntary insurance plans. Also, he stated he was against federal subsidies on the ground that one subsidy just led to another.

Continuance of Income

Further, he outlined the most important thing to the wage earner is a sickness benefit plan that will assure continuance of income. The second most important protection should be a $100 deductible voluntary insurance plan that would cover three-fourths of catastrophic illness costs up to such sizable sums as $5,000 to $10,000. There is already a wide coverage of Blue Cross for hospital costs and Blue Shield for surgical costs, he pointed out.

He continued, noting that these plans furnish protection for all but long-range and catastrophic illness. But they carry nothing for nursing costs, nor for ordinary care in chronic cases after Blue Cross benefits run out.

Mr. Folsom believes an additional policy is needed that does not cover the first $100, or perhaps $200, but that picks up from that point on to pay three-fourths of all additional costs to a high minimum — up to $5,000 or $10,000. The employer might pay part of the costs, perhaps on a 50-50 basis.

Wall Street Journal Comments

Noting that Mr. Folsom is taking a new look at reinsurance, the *Wall Street Journal* comments editorially:

> "Indeed, Mr. Folsom doesn't think the plan would work either. He thinks the net effect would only be another vast Government subsidy and one subsidy only leads to more and more. His attitude is certain to be twisted into something different by those who want more socialism in the Government, and doubtless he will be called a heartless fellow. This will be unfair, for Mr. Folsom recognizes that there are health problems and lags in the present forms of health insurance. But he recognizes also that there are wrong ways to go about trying to cure all the ills and cure them all at once."

Bureaucracy Won't Help

The *Journal* goes on to say that the surface in the health field, as noted by the Secretary, has hardly been scratched by voluntary insurance in spite of its added services and growth in recent years. It states:

> "But what most of the companies have accomplished has been done on a sound actuarial basis and Federal bureaucracy has neither helped nor, as is often the result, hindered the growth. Already two-thirds of all employed people in the nation are covered by some sort of sick benefit plan."
>
> "We would certainly be very much happier about it," the *Journal* comments, "if everyone had assurance that a sudden illness or a chronic ailment would not prove

too much of a drain on their other responsibilities and we hope that the time will not be long before the voluntary associations can cover most such catastrophies."

Government Can Help Some

The *Journal* feels that the Government can help in this effort. One way would be to reduce the taxes which drain the ailing as well as the healthy. Another suggested way would be to allow more reasonable tax deductions for expenditures having to do with health care.

In conclusion, the *Journal* states:

> "But there is one way not to do it. To put a Government bureaucracy into the business of insuring against sickness will not measurably aid the people's health. It would only be to make more dangerous the virus of state control."

GOVERNMENT INEFFICIENCY SEEN IN GREAT BRITAIN

Having little to do with medicine or medical economics, but nevertheless exemplifying government duplicity of effort and inefficiency in too much government in private affairs, a recent newspaper story asks the question "How many men does it take to install one gas stove?"

The story, by Seymour Topping and datelined London, is headlined "Not the Heat — It's Stupidity; Takes 23 Men to Install Stove." Mr. Topping quotes the *London Daily Express* in its campaign for more efficiency in Britain, and the paper's story about a man and his stove. The story states:

> "According to W. B. Wilkinson, who runs a London hardware store, this is what happened when he asked the local government-run gas board for a new stove. An inspector called, looked at the old stove and agreed to the request. Two men arrived to deliver the new cooker. Two days later, two men arrived to fit it. Later, another two men called and took the old stove away.
>
> "Seven men so far.
>
> "Meanwhile, the men fitting the new cooker decided that a new meter was needed. A man arrived with a new meter. Two more men called to connect it. Another man also called to take away the old meter.
>
> "It was then decided that the pipes needed blowing out. Three men drove up in a truck and set to work with a compressor unit. After a morning's work they decided the stoppage was not within the building, but must be under the sidewalk or road.
>
> "Fourteen men so far.
>
> "At this point an inspector arrived with a colleague. They surveyed the situation. Then, one morning, a truck carrying six men, automatic picks, compressor and other tools, drew up. They dug up the road and pavement.
>
> "For two days—no gas at all.
>
> "A new section of pipe was installed and for nearly a year everything was peaceful. Then Wilkinson reports, the other day a man dropped in. He announced he would be there the following Thursday to paint the pipes.
>
> "'What pipes?'
>
> "'The new gas pipes, of course,' was the reply.
>
> "And he was No. 23."

The *London Daily Express* editorialized: "Can anyone beat that for a record of over-employment?" And there was no comment from the gas board.

DOCTORS, UNION FUND SIGN UP

A recent article in *Business Week* notes a new development on relations between unions and medicine. Commenting that the unions and the AMA have fought each other on broad questions of public policy such as a national health program and on local practical issues such as group medical coverage of union workers, the story tells of what may well be the beginning of the end of some of the antagonism:

> "Now, in California—where the local units of AMA and the labor organizations have been as deeply antagonistic as anywhere else—a new trail in cooperation is being blazed by the working relationship established between a conservative county medical association and a left-wing union."

The whole thing began about a year ago when the Welfare Fund of the International Longshoremen's & Warehousemen's Union and the Pacific Maritime Association launched a dental care program. Now, the story goes on, under a novel plan with the help of the San Joaquin Medical Society, the fund is providing medical and surgical coverage for the 550 longshoremen in the port of Stockton, California, and their families.

"The Stockton health contract accords a degree of protection not commonly available in group coverage," the magazine states. "But what makes it unique is that it bridges the chasm between closed-panel and open-panel health insurance."

Explaining, the article says:

> "In California, that's an accomplishment. The California Medical Association has watched with disfavor the mushrooming growth of Henry J. Kaiser's closed-panel health plan, which now covers 500,000 on the coast. In the closed-panel restriction on choice of a

physician, the CMA sees an assemblyline approach to medical care and a rupture of the normal relation between doctor and patient."

As the Kaiser Foundation Health Plan has spread, the magazine notes, the CMA has improved its own open-panel plan, the California Physician's Service, by making full benefits available to people of higher income. In most counties, full coverage under this plan is available to people up to the $6,000 income level.

Free Choice

Workers under the new Stockton contract get the economic benefits usually associated with closed-panel service, while preserving the free choice feature inherent to the open-panel system, the magazine states. It continues:

"Members of the San Joaquin County Medical Society last year created the San Joaquin Foundation for Medical Care as the vehicle through which they could participate in the welfare fund's preventive medicine experiment. Now the foundation has assumed the additional role of receiving insurance payments from the welfare fund and paying doctors for service to the insured."

Practical Working Plan

Any member of the county medical society may become a member of the foundation and thus qualify to treat those covered by the contract. So far, 125 of the 154 members of the society have qualified and the number is growing. For emergencies arising outside the county, provision is made for reimbursing physicians who are not members.

For each worker covered, the welfare fund pays the foundation $7.31 a month. Doctors bill the foundation for services rendered.

For health insurance purposes, the doctors have three schedules for fees, based on average income of a group.

Most of the welfare fund comes from the employer contributions of 10c per man-hour. The rest is made up from the 1 per cent payroll tax on workers for disability insurance; the state takes 1 per cent of the first $3,000, the fund gets what is left. The fund also gives money to the Kaiser Foundation for a complete health package in the larger port cities and an insurance company gets some funds for medical insurance in smaller ports.

Results Tabulated

According to the magazine, the results of this preventive medicine experiment are still being tabulated. Full-scale medical examination with all the frills was made available to any longshoreman or warehouseman who wanted it. Of 900 eligible, 688 took the tests. The object was to spot incipient ills before they developed into costly medical, surgical and hospital cases.

PHYSICAL MEDICINE RESIDENCY FOR CAREER DOCTORS

A residency training program in physical medicine and rehabilitation has been started by the Veterans Administration for its *full-time career physicians.* The pilot plan will operate in Veterans Administration hospitals in Boston, Massachusetts; Bronx, New York; Hines, Illinois; Houston, Texas; and Los Angeles, California. Under the program a career Veterans Administration physician may take the training without any reduction in salary. He will be obligated to serve Veterans Administration for a year for one year's training; one and one-half years for two years' training and two years for three years' training.

Veterans Administration explained that the new program is separate from residencies in physical medicine and rehabilitation already in effect in fifteen Veterans Administration hospitals. The latter generally are for younger physicians recently out of medical school who do not contemplate a career in Veterans Administration medicine. Regular residency pay is provided in these instances.

Veterans Administration Medical Director William S. Middleton, in a separate announcement, reminded area medical directors and heads of Veteran Administration medical facilities that patients with chronic illnesses unable to care for themselves won't be discharged until outside arrangements are made for their care. This will be followed, Dr. Middleton indicated, even though hospitalization of an increasing load of veterans with chronic illnesses imposes "serious professional and administrative problems" on the Veterans Administration.—*AMA Washington Letter,* July 29, 1955.

While there is profound disagreement concerning the importance of acquired immunity, no one questions the importance of natural resistance in tuberculosis. It is clear that each individual possesses innate characteristics which determine the manner and intensity of his tissue response to the presence of tubercle bacilli. Clinical observations reveal that physical and mental fatigue, metabolic disorders like diabetes and starvation, and many other non-specific physiological disturbances often undermine resistance, while healthful conditions of living increase it. All this was well known fifty years ago; among the problems of which he urged study, Trudeau listed "above all, the mechanism of natural immunity."—Rene J. Dubos, Ph.D., Nat. Tuberc. A. Tr., May, 1954.

Public Health

POLIOMYELITIS VACCINATION IN MINNESOTA

Studies carried on this year by the state health department through its poliomyelitis surveillance program indicate that, even on the basis of a single dose of Salk vaccine, the procedure seems to give real protection.

As of September 19, 1955, there had been twenty-one cases of polio among the 112,115 first and second grade children who had received the Salk vaccine, with only two paralytic cases. In 33,259 nonvaccinated classmates there had been twenty cases, nine paralytic. This represents a case rate of 18.7 per 100,000 for vaccinates, and 60.1 per 100,000 for nonvaccinates. The case rate for paralytic polio among vaccinates was 1.8 per 100,000 and 27.1 among nonvaccinates.

These rates indicate there are three and a half times as many cases among the nonvaccinated as there are among those who received one dose of Salk vaccine. On the basis of these rates, there are fifteen times the number of paralytic cases among the nonvaccinated as among the vaccinated.

As of September 19, there have been 409 cases of polio this year, with five deaths, compared with 419 cases and eight deaths for the same period of 1954.

STATE PLAN FOR DISTRIBUTION OF SALK VACCINE

All Minnesota counties will receive Salk vaccine for poliomyelitis on the basis of percentage of population in the priority groups. Vaccination will largely be through private physicians in their own offices. The Minnesota plan for distributing the Salk polio vaccine made available under the Poliomyelitis Assistance Act was announced by Dr. A. J. Chesley, executive officer of the state health department, following approval by the state board of health and its poliomyelitis technical advisory committee. Approval of the plan by the United States surgeon general has been received.

All vaccine allocated to Minnesota at present will be purchased and distributed through the state health department with funds made available through the $30,000,000 federal Poliomyelitis Assistance Act. Minnesota's allotment under this act is $593,448. The entire amount will be used for the purchase of vaccine.

There will be no distribution of vaccine at this time through the usual commercial channels. When production reaches a high enough level to justify such a procedure, and/or when funds available to the state health department are exhausted, vaccine will then be released by the state through commercial channels.

Minnesota will have funds available through the Poliomyelitis Assistance Act to purchase about 760,830 doses of Salk vaccine between now and February 15, 1956. It is estimated that the cost to the state and federal government will be 78 cents per dose. To date, the allocation to Minnesota by the United States Public Health Service in Washington totals 172,000 cubic centimeters (each representing one dose) of released vaccine. The vaccine is expected to become available around October 15.

Even when the state receives its full quota of 760,830 doses, the amount is sufficient only to give a single dose of vaccine to approximately 70 per cent of the eligible population or two doses to 35 per cent of the population. Approximately 1,111,005 persons in Minnesota are currently eligible to receive vaccine, based on the number of persons under 20 years of age and the number of pregnant women.

Only persons under 20 years of age and expectant mothers are eligible to be vaccinated with vaccine purchased under the Poliomyelitis Assistance Act of 1955. The Act provided that the surgeon general could establish categories of persons to be accorded priority. The establishment of the 5-9 age group as the first priority group was on the advice of the national poliomyelitis advisory committee.

The Minnesota plan proposes to expand the first priority group to include the 0-4 age group and all pregnant women.

This expanded priority group would receive its first dose of vaccine as rapidly as it becomes available. A second dose, preferably after an interval of about four weeks, would be given after the demand has been met for the first dose. Under the plan, persons in the 10-19 age group fall into the second priority group.

Salk vaccine will be made available to all physicians on request without cost. Physicians' fees for administering the Salk vaccine will not include any charge for the vaccine. This has long been the practice for biologics for other immunizations traditionally supplied by the state health department, Dr. Chesley pointed out.

Requests will be processed and filled on the basis of percentage of eligible persons in counties and areas. Public clinics in Minneapolis, St. Paul, Duluth, and Rochester-Olmsted County, if expanded to include poliomyelitis vaccination, will receive, upon request, a proportionate share of the vaccine allocated to the county or area. The fed-era law prohibits the application of a means test in such public clinics.

To assure equitable distribution among areas, physicians, and clinics, the state health department will keep a current record of the county area allotment, requests against such allotments, and shipments made. Vaccine will be supplied only on the basis of reports furnished by physicians and clinics. Additional vaccine will be withheld if reports are delinquent. The report will include name, age, sex, date and site of inoculation, manufacturer and lot number.

PARALYTIC POLIOMYELITIS CASES REPORTED IN MINNESOTA 1949-1953
Rate per 100,000 Population by Individual Year of Age

Rank	Age Group	Total Cases 1949-1953	Yearly Average	Average Rate
1	5 years	251	50.2	92.1
2	7 years	223	44.6	84.9*
3	6 years	234	46.8	84.2*
4	3 years	259	51.8	83.1
5	1 year	282	56.4	80.6
6	4 years	238	47.6	76.4
7	8 years	196	39.2	74.6*
8	2 years	258	51.6	73.8
9	9 years	167	33.4	63.6

POLIOMYELITIS CASES (ALL TYPES) REPORTED IN MINNESOTA 1950-1954
Rate per 100,000 Population by Individual Year of Age

Rank	Age Group	Total Cases 1950-1954	Yearly Average	Average Rate
1	5 years	490	98.0	179.7
2	6 years	463	92.6	166.6*
3	7 years	407	81.4	155.0*
4	4 years	452	90.4	145.0
5	3 years	439	87.8	140.9
6	10 years	299	59.8	131.8
7	8 years	332	66.4	126.4*
8	9 years	312	62.4	118.8
9	12 years	253	50.6	111.6
10	2 years	364	72.8	104.1
11	11 years	234	46.8	103.2
12	14 years	202	40.4	95.4
13	13 years	215	43.0	94.8
14	1 year	331	66.2	94.7
15	15 years	171	34.2	81.6

*Age groups largely covered by vaccination program in first and second grades of all schools, 1955.

Dr. Chesley pointed out that the federal government set up the voluntary control program to make certain that every state would get its fair share of the vaccine. The shortage of vaccine will continue for some period of time. Minnesota, with 2.17 per cent of the estimated eligibles for vaccination, will receive that proportion of each batch of vaccine produced and released.

The Minnesota plan insures that each area in the state will receive an equitable distribution in the same manner.

The state health department has set up a quota for each county based on the estimated number of persons in each priority group. As vaccine shipments are received, each county and area will receive its proportionate share. Hennepin County, with approximately 20 per cent of the state's total population in the 0-9 age group, would receive that proportion of each vaccine shipment. On the same basis, Ramsey County would be eligible for about 11 per cent of the vaccine, and St. Louis County would be entitled to an estimated 6 per cent. Most counties would receive approximately one per cent or less of the vaccine.

Hennepin County, with 117,228 eligible nonvaccinated children in the 0-9 age group, representing about 20 per cent of all children in the state in this age group, is the largest. It will receive vaccine in that same proportion. This means that of the 172,000 doses to be released to the state, Hennepin County would get 20 per cent or 34,400 cubic centimeters. This will provide the first dose of vaccine for slightly more than one-third of the total eligibles. With a total of 645,099 estimated nonvaccinated children in the 0-9 age group in the entire state, the same proportion of one out of three and one-half holds true on a statewide basis.

When vaccine through commercial channels is made available in the state, manufacturers will supply the state health department with invoices of vaccine shipped into the state and pharmacists and supply houses will supply weekly sales reports. These reports will be checked with the county file and allotment data. Should any county or area get more than its proportionate share of the vaccine at any time, the situation will be corrected in future shipments. Biologic supply firms will be kept informed as to allotment and needs. Manufacturers, pharmacists, and supply houses have already indicated their willingness to comply with the recommendations of the state board of health.

The Minnesota plan for the distribution of public vaccine provides for the vaccination of Indians in the designated priority age group on the county allocation plan. Vaccine will also be supplied to state institutions for wards of the state on a proportionate basis for immunization of persons in the existing priority group.

Voluntary clinics organized and established by physicians in co-operation with the local health officer may be established, but the plan adopted by the state board recommends that such clinics not be organized until the vaccine supply is considerably greater. With only enough vaccine to give the first dose to about one-third of the children in the priority group, a clinic would not be a sound procedure, Dr. Chesley pointed out. If such clinics are organized, the vaccine will come from the allocation of the area the clinic serves.

There are approximately 223,165 children in the 5-9 age group, according to a formula which excludes the 112,115 children in the first and second grades vaccinated earlier this year in the National Foundation for Infantile Paralysis program. Vaccine to give these children their second shot was distributed by the state health department on September 12 for use in school clinics arranged by local communities. This program is completely separate from the Poliomyelitis Assistance Act program.

There are approximately 37,625 pregnant women in the state and an estimated 384,309 children in the 0-4 age group to swell the total to 645,099 persons in the first priority group recommended in the Minnesota plan. The second priority group includes 465,906 persons in the 10-19 age group, to make the 1,111,005 persons in Minnesota currently eligible to receive the vaccine.

The recommendation to enlarge the priority group to include the 0-4 year olds was based on the paralytic poliomyelitis cases reported in Minnesota for 1949 through 1953 and poliomyelitis cases (all types) reported in Minnesota for 1950-1954. Both the attack rate and the paralytic polio rate for all ages, 0-9, were so close that there could be little difference drawn in any consecutive ages such as 0-4 and 5-9. It was therefore felt that all these ages should be included in the first priority group.

Letter to Minnesota Physician

Dear Doctor:

You may now order, from the Minnesota Department of Health, poliomyelitis (Salk) vaccine for active immunization against poliomyelitis of the first priority group of individuals in your practice. Details of the State Plan of distribution of the vaccine are given in the enclosed abstract.

The essential points of the plan are:

1. Vaccine supplied by the Department is to be used ONLY FOR FIRST INJECTIONS OF THE FIRST PRIORITY GROUP—children 0-9 years of age inclusive, and pregnant women.
2. Vaccine should be requested only for your own use. Doctors not doing vaccinations should not request vaccine. Pregnant women should receive their injections from the doctor providing prenatal care.
3. Distribution will be based upon the percentage of eligible persons in each county and area.
4. Shipments of vaccine, after the initial distribution, will be made only if proper reports of use are furnished to the Minnesota Department of Health by physicians and clinics.
5. There will be no vaccine available in Minnesota at this time through commercial channels. You will be notified when vaccine can be obtained through commercial sources.

Salk vaccine will be sent to all physicians on request without cost. The initial shipment will be small to insure a fair share for everyone who may request vaccine. Subsequent shipments will be available within the limits of the county allotment. Vaccine will be sent, when available, only if requests for it are received.

The State Board of Health recommends this vaccination procedure as safe and effective. At the same time, the Board urges doctors to report immediately any adverse reaction to the vaccine or the development of suspected poliomyelitis in any vaccinated individual.

Please use the enclosed form for your initial request for vaccine. Do *not* list names, etc., of individuals in this request. Such information will be required on the report form which will accompany the shipment of vaccine, and which will be returned to the Department of Health following use of the vaccine. Requests for subsequent, additional supplies of vaccine should be submitted when this report of use is returned to the Department and may be placed on the back of the form.

Respectfully,
A. J. Chesley, M.D.
Secretary and Executive Officer
Minnesota Department of Health

REQUEST FOR POLIOMYELITIS VACCINE

MINNESOTA DEPARTMENT OF HEALTH
Division of Disease Prevention and Control
University Campus, Minneapolis 14, Minnesota

Date......................

I hereby request poliomyelitis vaccine for the first inoculation of the following number of eligible first priority persons in my practice.

Children 0-9 years of age inclusive........

Pregnant Women........

Total Number........

Signed............................, M.D.

Address..........................

....

..........................County

Do not list names of individuals in this request. Such information will be required on the report form which will accompany the shipment of vaccine, and which will be returned to the Department of Health following use of the vaccine. Requests for subsequent, additional supplies of vaccine should be submitted when this report of use is returned to the Department and may be placed on the back of the form.

History of Medicine in Minnesota

THE PIONEER DOCTORS OF CHIPPEWA COUNTY PRIOR TO THE YEAR 1900

LEON G. SMITH, M.D.
Montevideo, Minnesota

(Concluded)

Dr. Herbert B. Crommett was another doctor who was in Chippewa County but briefly. About the only information about his early life is that he was born in 1873, belonged to the Allopathic School of Medicine and graduated from the University of Minnesota in 1896.

His professional card appeared in the *Leader* and *Commercial* in 1898-99. One news item January 14, 1898 said: "Dr. Herbert B. Crommett of Minneapolis arrived this week to locate here. His office will be over Peterson and Smith's Hardware Store;" on March 25, 1898 he "took Chas. Corse to Minneapolis for an appendectomy;" on July 28, 1899, he "resigned as manager of the baseball club;" and November 24, 1899, he "left for a new field in Wisconsin." He was listed in Amery, Wisconsin, from 1900 to 1923 but by 1925 his name had disappeared from the directory.

Maynard's first doctor seems to have been **Dr. Albert J. Tictin.** He was born November 8, 1864 in Germany. He was a graduate from the college of Physicians and Surgeons in Chicago, in 1890. The Medical Directory listed him from Barnum, Minnesota, the same year, indicating that he first located in that village. He later moved to Alden, Minnesota. It was not until 1897 that he came to Chippewa County, locating in Maynard. There he practiced medicine and managed a drug store.

The *Montevideo Commercial* of June 6, 1899 stated: "Dr. A. J. Tictin, of Maynard, is reported to have married the past week." His name does not appear in the roster of the Camp Release District Medical Society, when it was organized in 1903, but he was still in Maynard then, because a news item of January 22, 1904, stated: "It is reported that Dr. Tictin will move to Clara City."

No further record of his being in Clara City or Maynard appears, and the only additional information we have of him is that at some time he moved to Brooklyn, New York, where he died January 27, 1937.

The last doctor to settle in Chippewa County during the Nineteenth Century, is still, after fifty-eight years of practice, fifty-six of which were spent in this county, actively engaged in his profession.

If an opinion poll could be taken among doctors and laity alike, throughout this four or five county area, and the question asked, "Who is the best loved doctor in western Minnesota?" I feel certain the overwhelming answer would be Dr. M. A. Burns, of Milan.

Dr. M. Alpheus Burns was born in Panora, Iowa, May 13, 1873, the son of William H. and Margaret Barthelow Burns. His parents met when his father, as a Civil War soldier, was fighting in the South and became acquainted with the former Virginia girl, then living in Maryland. After the war, they were married and lived for a time in Washington, D. C., moving to Iowa in 1870.

In 1886 the family moved to St. Paul, where Dr. Burns attended high school at St. Paul Central. He entered the University of Minnesota Medical School, and graduated in 1896.

He practiced medicine at Rosemount until January, 1897, at which time he started his internship at the Minneapolis City (now called General) Hospital.

In January, 1898, he settled in Milan, Minnesota, where he has been practicing continuously ever since, except for occasional interludes for post-graduate work. The first of these was in 1901, when he took a course in surgery at Post-Graduate College, New York City. That was when the knowledge of asepsis was just coming in, and he saw rubber gloves used in surgery and obstetrics for the first time. He bought several pair and brought them home with him, and laughingly tells of how, when he wore them at first on confinement cases, the old midwives were quite disgusted with the young, upstart doctor who was so finicky that he wore gloves to protect his hands.

Though of Scotch-Irish descent, he, very early in his career, picked up a very workable knowledge of the Norwegian language and was able to converse quite well in that tongue.

He bought a Rambler automobile in 1902, which he believes was one of the very first automobiles in Minnesota.

In 1904, he married Wilhelmine McKenzie, a graduate nurse, who was born in Park Hill, Ontario, Canada. There are three children, Floyd, George, and Mary.

A younger brother, F. W. Burns, had graduated from the Illinois University Medical School in 1903, practiced in South St. Paul for two years and moved to Watson, Minnesota, in 1905. In 1910, he moved to Milan and became associated with Dr. Burns there.

They built a hospital which was completed very shortly after the Montevideo Hospital, and its first patient was admitted February 7, 1911. Only a year later, this hospital burned to the ground. There were eighteen patients in the building at the time, and it was a miracle that no lives were lost. These patients were transferred to the Montevideo Hospital, and ever since, Dr. Burns has been upon its staff and has done his work there.

His brother moved away in 1913 and Dr. Burns practiced alone until shortly before World War II, when his older son, Floyd M., finished medical school and internship, and entered practice with him. Again, during the war, he was compelled to work alone. Floyd served for several years in the United States Navy, and took a year of surgical work in a V.A. hospital, after which he returned to Milan to become associated with his father.

Besides his professional work, Dr. Burns has always been very active in community affairs, having been mayor of the village for several years, Health officer for most of his professional life, and a school board member for more than twenty-five years.

He also has been president of the Camp Release District Medical Society, and held many other public offices.

He is one of a small and select group, the Fifty Club of the Minnesota State Medical Association, membership in which is possible only for a doctor who has been in the active practice of medicine for 50 years or more.

Some of his escapes from injury or death have been narrow. One of these incidents bears narrating. It was his custom, when the ice was frozen thick on Lac qui Parle, to save many miles of winter driving by cutting across the lake. Near the time of the spring break-up, he made a call across the lake late one afternoon, on a route where he could see that the ice was safe. On the return trip, however, it was after dark and a storm arose, so that, when he reached the open ice, he could not see his way. He let the horses pick their own course, which they did very well, and he arrived home safely. But, when he came over the same route the next day, he could see the marks of his sled runners from the night before, winding around patches of open water.

As this is being written, in the spring of 1954, Dr. Burns has reached the age of 81. A year ago, he accepted a call to Appleton before daylight one stormy morning, and got stuck in the snow at the viaduct, nearly two miles from Milan. He walked back to town in the blizzard, and routed out a garage man to take him to his destination over another road.

Over the many years he practiced alone, I am sure he did more driving, over a territory of a thirty mile radius, than any other three of his confreres combined. Often he would see patients in his office all day, sit up on a hard kitchen chair in some farm-house all night awaiting a delivery, and then by morning, drive fifteen miles to the Montevideo Hospital to do major surgery, and do it well.

Fortunately, Mrs. Burns is in reasonably good health, and he is as alert and full of vigor as ever. His family lives near him, Mary (Hegdahl) with her husband in St. Paul, George and his family on a farm near Milan, and Dr. Floyd and his family only a few blocks away, with Floyd taking over as much of the burden of work as his father will allow.

A few years ago the Camp Release District Medical Society nominated Dr. Burns as a candidate for the Outstanding General Practitioner Award. I quote one paragraph from that nomination which sums up the feeling of his fellow doctors: "Above and beyond his community activities, the one characteristic which makes Dr. Burns the outstanding general practitioner of our district, and, in our minds, of the entire state, is his strict sense of professional duty. He is of the older generation of physicians, who comes at once when his patients call him, night or day, blizzard or rain, 'come hell or high water.' "

This concludes the chronicle of the doctors of Chippewa County from its beginning to the turn of the century.

There are, however, three other doctors who settled here in the first years of the new century, who spent almost their entire professional lives in this county. One practiced here for over 30 years, the other two, more than 40 years. Each of them was prominent in community affairs; each was an exceptionally good doctor. Their many years of service to the people of this area and the importance of their contribution to the welfare of their communities, make it fitting that a brief biographical sketch of their lives be added to the history of our pioneer doctors. These physicians are Drs. L. R. Lima and L. N. Bergh of Montevideo, and Dr. H. T. Foshager, of Clara City.

Dr. Ludvig Reinhart Lima was born in Stavanger, Norway, April 30, 1877. He received his primary education in Norway, and emigrated to the United States. In 1896 he enrolled in Augsburg College in Minneapolis for his premedical work, graduating in 1899 with a B.A. degree. He obtained his medical education at the College of Medicine and Surgery of Hamline University in St. Paul, graduating in 1903. After a year's internship in the City and County Hospital of St. Paul, he entered the active practice of medicine in Montevideo, and his first professional card appeared in the Montevideo papers November 11, 1904.

Shortly before coming to Montevideo, upon September 25, 1904, he was married to Constance Bergh. Four children were born, Brunhild, Hildegarde, Antoinette and Ludvig R., Jr.

Three years after locating here, he was joined in practice by Mrs. Lima's brother, Dr. L. N. Bergh, and that association continued for many years.

Dr. Lima was a member of the Camp Release District Medical Society, the Minnesota State Medical Association and the American Medical Association during all of his years of practice. He was a surgeon for the Milwaukee Railroad during most of his professional life, and was on the staff of the Montevideo Hospital from its inception in 1910 until his death.

He was a busy practitioner, but he found time also for his main interest outside of medicine, namely, horticulture: he was a life member of the Minnesota Horticultural Society and for a great many years was the president and mainstay of the Montevideo Park Board.

He served for a time in the United States Army during World War I.

His daughter, Hildegarde, had married a doctor, Dr. W. A. Owens, and shortly before World War II, his son, Ludvig, Jr., was studying medicine. Dr. Lima's plans to retire, and turn his practice over to his son and son-in-law were completed. In fact, Dr. Owens had already become associated with him when the war spoiled the plans. Dr. Owens entered service, and Dr. Lima, instead of retiring to a well-earned rest, felt compelled to again take up the full burden of his medical practice. When Ludvig, Jr., completed his medical education and internship and joined his father, and when soon thereafter, Dr. Owens returned from army duty to enter the group, Dr. Lima, finally, was able to retire. In 1946, after 42 years of arduous practice, he was able to take time for the work with plants and trees that he so loved. However, within two years his death occurred on September 14, 1948.

His daughter, Antoinette, passed away in 1936. Mrs. Lima survived him for a few years, but she died in 1952.

Brunhild (Brown), Hildegarde (Owens) and Ludvig R., Jr. survive him, the two latter living in Montevideo, with their families. As previously noted, Dr. W. A. Owens (son-in-law) and Dr. Ludvig R. Lima, Jr., (son) are carrying on his practice.

Dr. Luthard N. Bergh was born at Stony Run, Yellow Medicine County, near Montevideo, September 27, 1880. His father, Ole Nielson Bergh, was a pioneer pastor in this part of the state. In 1883, the family moved to Audubon, Minnesota, where Luthard attended public school. He obtained his pre-medical work in Augsburg College in Minneapolis, and in 1906, he graduated from the University of Minnesota Medical School, and served a year as interne at Ancker Hospital in St. Paul.

He established his practice in Montevideo in June, 1907, being associated with his brother-in-law, Dr. L. R. Lima, for many years.

He served continuously upon the staff of the Montevideo Hospital from the time it was built in 1910 until his death. He was surgeon for the Milwaukee Railroad during the same period of time and served the Selective Service system throughout both World Wars. In April, 1946 he was awarded a Certificate of Merit and the Selective Service Medal, authorized by Congress and the President of the United States, in recognition of that service. He was also a lifelong member of the Camp Release District Medical Society, the Minnesota State Medical Association, the American Medical Association and the American Association of Railway Surgeons.

Dr. Bergh was wrapped up in his profession to the point where he took little time for travel, hobbies or other outside interests. For some years, until forced to stop by a lame shoulder, he played a good game of golf, finding the relaxation and fellowship that made those the happiest years of his life.

He was an excellent surgeon, and for many years, he had the largest surgical practice in the western part of the state.

He was married to Grunhild Sverdrup in 1907, and two children were born, George S. and Solveig, both eventually becoming doctors.

Dr. Bergh suffered a stroke some years before his death, but this did not incapacitate him for long. After some months, he returned to doing surgery, even with a slightly crippled arm. However, his forty-one years of strenuous practice finally took their toll, and he died of a heart attack in 1948.

His wife, his daughter, Dr. Solveig Bergh, and his son, Dr. George S. Bergh, with his family, all live in Minneapolis.

Dr. Henry T. Foshager was born at Pennock, Minnesota, July 13, 1881. He was educated in the rural schools of his home community, and attended St. Olaf Academy and St. Olaf College in Northfield, Minnesota, graduating in 1905. He attended the University of Minnesota Medical School, graduated in 1909, and served a year's internship in Ancker Hospital in St. Paul.

He first located for practice in Ipswich, South Dakota, leaving there after three years to become associated with Dr. B. J. Branton in Willmar, Minnesota. He remained there but a short time before opening an office in Clara City, where he practiced continuously for 32 years, until his death.

On August 7, 1918, he was married to Gertrude Habstritt of Clara City.

Dr. Foshager was a member of the Camp Release District Medical Society, the Minnesota State Medical Association and the American Medical Association. He served as mayor of Clara City in 1917 and 1918 and was its health officer for many years.

His work was extremely rigorous because he had a large practice, particularly in obstetrics, and an extensive territory to cover. Even when his patients were hospitalized, it meant an 18 mile drive to Montevideo to attend them.

That was the ultimate cause of his death, because it was in hastening to attend such a case, late in the evening of October 19, 1946, that his automobile collided with a truck whose driver had failed to see a stop sign. He was killed instantly.

Certainly his death must be recorded as "in line of duty." Mrs. Foshager resides in Clara City. There were no children.

For the information that I have obtained in preparing this account of our pioneer doctors, I must credit the "History of Minnesota," by William Watts Folwell, "History of the Minnesota Valley" by Neil and Bryant, and "History of Chippewa and Lac qui Parle Counties" by Moyer and Dale.

I am also indebted to many of the living relatives of these various physicians and I wish to express my gratitude to Ludwig Roe and Everett S. Mills, for allowing me to browse through the old files of their newspapers. And my especial thanks go to John W. Peterson, Winifred Barber Nelson and Ida Simpson Kohr, for calling upon the memories of their childhood days in this vicinity to help me in my work.

In Memoriam

PHILIP A. ARLING

Dr. Philip A. Arling, graduate of the Medical School of the University of Minnesota and former Minnesota resident, died September 20, 1955, after an automobile accident. He had been in practice in McMinnville, Oregon. He was forty-six years old.

Dr. Arling had been a member of the Hennepin County Medical Society, the Olmsted-Houston-Fillmore-Dodge County Medical Society and the Minnesota State Medical Association. After a period of military service, he was a resident in surgery at the Mayo Clinic before moving to the west coast.

Dr. Arling is survived by his brother, Dr. Leonard S. Arling, Minneapolis. He leaves his wife, three children; three other brothers: Henry W. Swenson and Walter D. Swenson, Minneapolis, Gilbert Swenson, Chisago City; three sisters: Mrs. Howard Reick, Minneapolis, Mrs. Werner Hedenstrom, Saint Paul, and Miss Mildred Swenson, Racine, Wisconsin.

JOSEPH LAWRENCE ARKO

Dr. Joseph L. Arko, eye, ear, nose and throat specialist of Hibbing, died September 9, 1955, at the age of forty-two. He was on the staff of the Morsman Clinic and the Rood Hospital.

Dr. Arko was born in Chisholm in 1913. He attended Chisholm schools, Hibbing Junior College and the University of Minnesota. He interned at Wilkes Barre General Hospital in Pennsylvania. In 1939 he started his practice in Hibbing.

During World War II, Dr. Arko served in the army as a captain in the medical corps. His duty was on hospital ships, and in England and Italy.

Dr. Arko was an active member of the Rotary and had served a term as president of the Hibbing club. When he became ill three years ago, the club presented him with a television set as a tribute to his work and as a sign of their regard for him. He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Arko is survived by his wife, the former Mary Lovaas; two children, Joseph and Betsy; his father, John C. Arko of Chisholm; two brothers, John and Frank, both of Virginia; and a sister, Mary of Hibbing.

EGIL BOECKMANN

Dr. Egil Boeckmann, prominent St. Paul eye, ear, nose and throat specialist, died September 3, 1955, at his home at Dellwood, White Bear Lake. Son of a distinguished Norwegian physician, Dr. Boeckmann came to St. Paul with his parents when he was five. At the time of his death he was seventy-four years old.

Dr. Boeckmann attended the University of Minnesota, the University of Pennsylvania medical school and did post-graduate work in Europe. While at the University of Minnesota, Dr. Boeckmann was famed as the athlete who ran the length of the field in the final seconds to tie the score of the Minnesota-Michigan football game of 1903. This was the game that started the traditional "Little Brown Jug" rivalry.

Dr. Boeckmann served on the Board of Regents of the University in the '20's. He was a member of the Ramsey County Medical Society, which his father helped found. He was a member of the Minnesota State Medical Association.

Besides his wife, the former Rachel Hill, Dr. Boeckmann is survived by two daughters, Mary Hill Boeckmann and Mrs. Peter Ffoliott, both of St. Paul; three sisters, Mrs. Charles Freeman and Mrs. Clarence Freeman, both of St. Paul, and Mrs. Julius Seyler, Munich, Germany, and five grandchildren.

HOWARD KRAMER GRAY

Dr. Howard Kramer Gray, fifty-four, a member of the staff of the Mayo Clinic since 1932, was drowned in the Mississippi River some 5 miles north of Lake City, Minnesota, on September 6, 1955, while swimming to recover a dinghy that had become detached from his cabin cruiser.

Dr. Gray was born at St. Louis, Missouri, on August 28, 1901, the son of Harriette Flora Gray and Carl Raymond Gray. He received the degree of bachelor of science from Princeton University in 1923, and from 1923 to 1925 was a student in medicine at the University of Nebraska. He then entered Harvard University, from which he received the degree of doctor of medicine in 1927. He served his internship at the Robert Packer Hospital in Sayre, Pennsylvania, from July 1, 1927, to July 1, 1928.

Dr. Gray came to Rochester, Minnesota, as a fellow in surgery of the Mayo Foundation on October 1, 1928. On July 1, 1931, he was appointed a first assistant in surgery, and from January to October, 1932, he was assistant resident surgeon at Saint Mary's Hospital. In 1932, he received the degree of master of science in surgery from the University of Minnesota. He was appointed to the staff of the Mayo Clinic in 1932, as a junior surgeon with the rank of instructor of surgery in the Mayo Foundation, Graduate School, University of Minnesota. In 1935, he was made head of a section of surgery, a post which he occupied until his death; in 1935, also, he was promoted to the rank of assistant professor. He became an associate professor in 1940, and a professor in 1947.

Dr. Gray, who took much of his training in operative surgery under the guidance of the late Dr. Edward Starr Judd, was attracted to surgery from the very beginning of his career. His first paper, published in 1931, dealt with external biliary fistulas. Surgical conditions of the gall bladder comprised a particular field that engaged his attention to the time of his death, and one in which he became widely known for his dexterity of operative technique and his profound knowledge of the underlying physiologic principles involved. In the same year, he published, with Dr. Waltman Walters and Dr. J. T. Priestley, a paper on total removal of the stomach, an operation in which he likewise acquired uncommon skill and wide recognition. In 1938, he operated on Col. James Roosevelt, eldest son of President Franklin D. Roosevelt, who came to Rochester with Mrs. Roosevelt to be near the son during and after the procedure.

Dr. Gray also did early work in surgical operations on the thorax. He maintained an active interest in advances in physiologic knowledge which could be applied to surgery, among them being his work with pedicle muscle grafts and, in recent years, the use of plastic tubing, in association with Dr. John H. Grindlay, for replacement of a section of the common bile duct from the gall bladder.

Dr. Gray had held a reserve commission in the Medical Corps of the Army, but on December 26, 1941 he entered the Medical Corps of the United States Naval Reserve with the grade of lieutenant commander. He was assigned as chief of surgery on the hospital ship, *Solace,* which was one of four such ships evacuating wounded men from the island of Saipan. Later, he was chief of surgical services at Aiea Heights Hospital in Honolulu, and was chief of surgery at the Naval Hospital in San Diego, California, at the time of his separation from the service in November of 1945. He was awarded the Navy Commendation Ribbon for "devotion to duty, able leadership in co-ordinating the surgical service of the hospital, and superior skill in his profession." In 1946, he was awarded the Legion of Merit, by direction of President Truman, "For exceptionally meritorious conduct in the performance of outstanding services to the United States. . . . His inspiring conduct throughout was in keeping with the highest traditions of the United States naval service." Dr. Gray held the grade of captain when he was released to civilian life in 1945.

Dr. Gray came of a family distinguished for its contributions to various aspects of American life. His father, Carl Raymond Gray, who died in 1939, was the son of a professor of mathematics; Carl Raymond Gray became president of the Union Pacific Railway System and later chairman of the board. He was awarded several honorary degrees, and served the railway until he was seventy years old.

Dr. Gray's mother, a native of Oswego, Kansas, was known nationally for her exposition of studies in the Scriptures. She gave a weekly radio broadcast in Bible studies from stations in both Omaha, Nebraska, and Kansas City, Missouri. In 1937, she was presented with the gold medal of the Golden Rule Foundation as "The American Mother for 1937."

A brother of Dr. Gray, Carl Raymond Gray, Jr., became vice president and general manager of the Chicago and Northwestern Railway Company and was a major general in the Transportation Corps during World War II. He was administrator of veterans' affairs in the Veterans Administration from 1947 to 1953, and has been decorated by several foreign governments.

Dr. Gray was a diplomate of the National Board of Medical Examiners, and was certified as a specialist in surgery by the American Board of Surgery, Inc., in 1941. He was a fellow of the American College of Surgeons, and a member of the American Surgical Association, the American Association for Thoracic Surgery, the Western Surgical Association, the American Medical Association, the Minnesota State Medical Association, the Southern Surgical Association, the Society of Clinical Surgery, the Nu Sigma Nu professional medical fraternity, Society of the Sigma Xi, the Boylston Medical Society of Harvard University, the Ivy Club of Princeton University and the Alumni Association of the Mayo Foundation.

Dr. Gray had been a member of the Board of Governors of the Mayo Clinic since 1948. He was awarded the honorary degree of doctor of science in 1949 from Lafayette College, and the same degree in 1954 by the University of Nebraska. In 1950 he was honored by appointment as the Lord Moynihan lecturer of the Royal College of Surgeons of England.

In 1942, with Drs. Waltman Walters and James T. Priestley as co-authors, Dr. Gray published a 576-page volume entitled "Carcinoma and Other Malignant Lesions of the Stomach," which was received as "an important contribution to useful knowledge on the subject of cancer of the stomach." In 1948 Dr. Gray was appointed special surgical consultant to the Union Pacific Railroad Employees Association.

Dr. Gray was married on September 2, 1925, to Miss Lila De Weenta Conrad. Two children were born to Dr. and Mrs. Gray: De Weenta Russell Gray (Mrs. Walter I. Bones, Jr., of Parker, South Dakota) and Howard Kramer Gray, Jr.

Because of his unfailing ability to instill both hope and cheerfulness in his patients, Dr. Gray was held in warm affection by countless patients whom he had treated surgically over the years. Because of his sincere and inborn courtesy and thoughtfulness, as well as his equanimity and evenness of temperament, he was held in the greatest esteem by the fellows, nurses and surgical colleagues who came into daily professional contact with him. He was a concerned citizen of his community, a devoted worker in his church, an excellent musician and a gracious host. "Howdie" Gray will be long remembered and grievously missed by myriads of people.

The Howard K. Gray Memorial Fund is being established by his friends to provide further postgraduate surgical experience to outstanding students of surgery.

JOHN N. LIBERT

Dr. John N. Libert, physician of St. Cloud since 1921, died September 2, 1955. He was sixty-two years old.

Dr. Libert was a native of St. Cloud. He attended St. Cloud Normal School before going to Creighton Medical College and interning at St. Joseph's Hospital in St. Paul. He served as a medical officer in the army for two years in the U. S. and France before starting his practice in St. Cloud.

Dr. Libert had been president of the Stearns-Benton County Medical Society and was secretary of the society for nearly twenty years. He was a member of the Minnesota State Medical Association, serving as vice-president in 1925, and the American Medical Association. He was a major in the national guard and belonged to the Fourth Degree Knights of Columbus, the Elks, the American Legion, and the 40 et 8. He was a member of the staff of the St. Cloud Hospital.

Survivors are his wife; his son, Harvey of Minneapolis; his mother, Mrs. Margaret Libert, and brother Paul, of St. Cloud; two sisters, Mrs. Robert Schmidt of Perham and Mrs. Frank Sullivan of Hamilton, Ontario, and four grandchildren.

DAVID EDWARD McBROOM

Dr. David E. McBroom, for many years superintendent of the Minnesota Colony for Epileptics at Cambridge, died September 3, 1955, in Minneapolis. He was seventy-one years old.

Dr. McBroom was born in Akron, Ohio, in 1883. He received his medical education at Ohio Medical University, graduating in 1907. He was on the staff of the School for Feeble-Minded at Faribault for three years, leaving to enter private practice. From 1910 until 1922 he practiced successively at Northfield and Adams, Minnesota, and Waterloo, Iowa. He was then recalled to the Faribault institution as assistant superintendent. In 1928 he moved to Cambridge where he served until 1941 when he was named director of the mental health unit of the Minnesota Division of Public Institutions. In 1945 he went to Redfield, South Dakota, to work for the State School for Feeble-Minded.

In 1941, Dr. McBroom was president of the East Central Minnesota Medical Society. He was a former member of the Minnesota State Medical Association, a member of the American Medical Association, the Minnesota Neurological Society and a fellow of the American Psychiatric Association. He was also a member of Tyrion Masonic Lodge, Knights Templar, Scottish Rite and Osman Temple of the Shrine.

Surviving are his wife, Medora, and a daughter, Mrs. Samuel Mayo.

EUGENE JOHN McCANN

Dr. Eugene J. McCann, Minneapolis physician, died August 21, 1955, in St. Barnabas Hospital. He had been ill for several months.

Dr. McCann was born in Shakopee, Minnesota, in 1904. He attended the medical school of the University of Minnesota, graduating in 1931. He interned at Minneapolis General Hospital. For three years he studied internal medicine at the University of Minnesota. He was a member of the staffs of St. Barnabas, St. Andrews, St. Mary's and Veterans Hospitals in Minneapolis.

For five years during World War II, Dr. McCann was an army colonel serving as commanding officer of the 162nd General Hospital unit, which served in England and France.

Dr. McCann had been a member of the Ramsey County Medical Society and the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was active in the United States army reserve.

He is survived by his wife; his mother, Mrs. Annie McCann, St. Paul; a sister, Mrs. William Muelken, Prior Lake, Minnesota; and four brothers, James, Joseph, Frank and Nicholas, all of St. Paul.

SIGSBEE RAYMOND SELJESKOG

Dr. Sigsbee R. Seljeskog, Minneapolis practitioner, died August 15, 1955. He was fifty-five years old.

Dr. Seljeskog was born in Waseca, Minnesota, in 1899. He attended schools in Pittsburgh, Pennsylvania, and the University of Pittsburgh. He graduated from the medical school of the University of Minnesota in 1936. After an internship at Lutheran Deaconess Hospital in Minneapolis, he did post-graduate work in physiological chemistry and hematology at the University of Minnesota, receiving a M.S. degree.

In 1941, Dr. Seljeskog entered military service. As a Lieutenant Colonel in the medical corps, he commanded a unit of the first field hospital to be set up on the continent after invasion of Normandy and remained with the unit in Belgium and Germany.

Dr. Seljeskog was a member of the Athletic club, Hugnad Lodge, Zarthan Lodge A.F.&A.M., Scottish Rite and Zuhrah Temple of the Shrine. He was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association. He was formerly superintendent of the staff at the Sister Kenny institute.

He is survived by his wife; a son, Edward, Minneapolis, two daughters, Elaine, Minneapolis, and Mrs. Anita Mittelsted, Chaska, and one grandchild; his father, Louis Seljeskog of Waseca; two sisters, Mrs. Sam Hagen and Mrs. Arthur Larson, Waseca; a brother, Theodore, Redlands, California.

CARLYLE TINGDALE

An unfortunate boating accident at Lake Vermillion took the life of Dr. Carlyle Tingdale, of Hibbing, August 8, 1955. Dr. Tingdale was forty-seven years old.

Dr. Tingdale was born in Minneapolis, son of Dr. and Mrs. A. C. Tingdale. He attended the University of Minnesota and interned at Minneapolis General Hospital. He had practiced medicine in Hibbing for eighteen years, first on the staff of the Rood Hospital, with the Mesaba Clinic and later in private practice. He was village health officer and school doctor. He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Dr. Tingdale is survived by his wife, the former Mary Catherine Bowen, daughter of Dr. Robert L. Bowen of Hibbing; his son, Patrick, and his father, Dr. A. C. Tingdale, Minneapolis; a brother, Randolph, New York City; a sister, Mrs. Owen Heath, St. Louis Park.

Reports and Announcements

MEDICAL MEETINGS

National

American Academy of Obstetrics and Gynecology, Conrad Hilton Hotel, Chicago, December 12-14, 1955.

American Association of Blood Banks, eighth annual meeting, Palmer House, Chicago, Illinois, November 19-21, 1955.

American College of Surgeons, sectional meeting, Milwaukee, Wisconsin, February 27-29, 1956.

American College of Surgeons, Conrad Hilton Hotel, Chicago, Illinois, October 30-November 4, 1955.

Association of Military Surgeons of the United States, 62nd annual convention, Statler Hotel, Washington, D. C., November 7-9, 1955.

Interstate Postgraduate Medical Association of North America, Minneapolis, Minnesota, November 1-4, 1955.

Mediclinics of Minnesota, postgraduate course sponsored by Academy of General Practice of Broward County, Fort Lauderdale, Florida, March 5-14, 1956.

National Society for Crippled Children and Adults, Palmer House, Chicago, Illinois, November 28-30, 1955.

Radiological Society of North America, Chicago, Illinois, December 11-16, 1955.

Southern Medical Association, Houston, Texas, November 14-17, 1955.

International

International Symposium on Enzymes: Units of Biological Structure and Function, Henry Ford Hospital, Detroit, Michigan, November 1-3, 1955.

AMERICAN COLLEGE OF CHEST PHYSICIANS TO HOLD STUDENT ESSAY CONTEST

The Council on Undergraduate Medical Education of the American College of Chest Physicians offers three cash awards for the best essay contributions by undergraduate medical students on any phase in the diagnosis and treatment of chest diseases (heart and/or lungs). First prize will be $250, second prize $100, and third prize $50. Winners will be announced at the annual meeting of the organization in Chicago, Illinois, June 7-10, 1956. Manuscripts must be submitted by April 10, 1956.

An application form, plus instructions and suggestions for manuscript preparation, can be obtained by writing the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

ASSOCIATION OF MILITARY SURGEONS

Physicians, dentists, veterinarians, nurses, women's medical specialists and medical service corps officers of the Army, Navy and Air Force Reserves are expected to attend the sixty-second annual convention of the Association of Military Surgeons. The meeting will be held at the Hotel Statler, Washington, D. C., November 7, 8 and 9, 1955.

The three-day meeting will include discussions of the medical problems facing the military services and the nation in an atomic war.

CONTINUATION COURSE

The University of Minnesota announces a continuation course in *Gastroenterology for General Physicians* to be held at the Center for Continuation Study from December 1 to 3, 1955. Dr. Henry L. Bockus, professor of medicine and chairman, Department of Medicine, University of Pennsylvania Graduate School of Medicine, Philadelphia, renowned gastroenterologist, will be guest speaker and will also deliver the annual *Journal-Lancet* Lecture on Thursday evening, December 1. The course, which will stress the management of the more commonly seen gastrointestinal disorders, will be presented under the direction of Dr. C. J. Watson, professor and head, Department of Medicine.

LYON-LINCOLN SOCIETY CLINIC COURSE

The fifty-second semi-annual clinic course of the Lyon-Lincoln Medical Society was held in September and October at the New Atlantic Hotel, Marshall. Speakers at the six weekly scientific meetings were Dr. Harold E. Miller, Minneapolis; Dr. B. F. Fuller, St. Paul; Dr. Ray C. Anderson, University of Minnesota; Dr. Bernard J. Spencer, Minneapolis; Dr. W. E. Wellman, Rochester, and Dr. Harry B. Hall, Minneapolis.

TWIN CITIES DIABETES ASSOCIATION

The Twin Cities Diabetes Association, a newly organized affiliate of the American Diabetes Association, will have Dr. Henry T. Ricketts of Chicago as guest speaker for a dinner meeting to be held at 6:15 p.m., November 16, 1955, in Coffman Memorial Union, University of Minnesota. Dr. Ricketts, former president of the American Diabetes Association, will speak on "The Problem of Vascular Disease in Diabetes." Dr. Ricketts is Professor of Medicine at the University of Chicago and is an outstanding authority in diabetes.

All physicians are invited to attend. Members of Hennepin and Ramsey County Medical Societies are invited to join the Twin Cities Diabetes Association. Information and reservations can be obtained through Dr. Wyman Jacobson, WA-6-6581, Minneapolis, or Dr. Marguerite Schwyzer, MI-8-3893, Saint Paul.

Woman's Auxiliary

COUNTY PRESIDENTS REPORT ACTIVITIES

Enthusiasm for progress, and also the desired participation of each individual member in the auxiliary program, seem to be the watchwords of the county presidents this year. At least this was very evident in conversations with the three responsible for the material on this page, namely—Mrs. Karl Walfred, Stearns-Benton; Mrs. Harold Peterson, Ramsey (reported by Mrs. L. G. Culver); and Mrs. L. R. Boies, Hennepin (reported by Mrs. C. W. Freeman).

Stearns-Benton

Stearns-Benton has outlined an active year, with Mrs. Thomas Dredge (formerly of Hennepin) acting as program chairman. They have monthly supper meetings, four members serving as hostesses each time, and have an average attendance of twenty-five. At their October meeting, they will present Dr. Mendelsohn, of St. Cloud Veterans Hospital, who will talk on mental health. He is an outstanding authority, having just recently come to St. Cloud from the Menninger Clinic in Kansas City. Other meetings will include a tea for prospective nurses, Christmas meeting at the Veterans Hospital when gifts will be brought for children of veterans, a book review, assembling of cancer dressings, and legislation. Their February meeting is a real treat, a Valentine party given by their husbands, which includes "dinner out," a corsage and a gift. Numerous new residences have been constructed, and members are opening them for auxiliary meetings. This adds up to an interesting and very worthwhile program.

Ramsey

The Fall Fashion Festival is over but will long be a topic of conversation. The proceeds from the show will go toward the Philanthropic Fund.

Congratulations to members of a great committee. Through their efforts and careful planning, the expenses were cut to a minimum. The very attractive table decorations, in the form of a wishing well with real little oaken buckets entwined with colorful paper flowers, were inspired by Mrs. Albert Hayes, chairman of decorations. These centerpieces were all made by the committee members. No time was wasted by Mrs. Hayes. It is reported that she carried crepe paper, wires, et cetera, with her in the car and while riding to and from her lake home, busied herself making buds and flowers.

Members from out-state might circle their calendars for next year's event, which will be held about the middle of September. Everyone is welcome.

The Year Book Committee members have been busy through the summer checking addresses and telephone numbers. The books will be ready for each member at the first meeting on October 17.

Members anticipate a happy and busy season this year with the very capable and pleasant Mrs. Harold O. Peterson at the helm.

Hennepin

Health Day—the major public education and public relations activity of the Woman's Auxiliary to the Hennepin County Medical Society—is well under way. Scheduled early in the fall, October 17, to co-ordinate with school and PTA programs, this day-long open house to acquaint Mr. and Mrs. Hennepin County and their families with the recent advances in medicine and dentistry, will emphasize the problems of adolescence this session.

Preparing for the event since last spring, the chairman, Mrs. Lyle French, and her committee of ten have contacted the principals of all public, private and parochial schools in the county, all PTA presidents, all the clergy, all women's and numerous other organizations, such as the American Legion. Mrs. French met with the School Health Committee of the Minneapolis Public Schools, in an effort to work out a program that would interest parents, teachers and students alike. Speakers were contacted, the main ballroom at the Radisson Hotel rented, publicity arranged.

Working with the Hennepin County Dental Auxiliary, the medical auxiliary is going all-out to make the event a success. Announcement cards will be sent out with doctors' September statements; house organs of local industries will carry notices; there will be a radio, television and newspaper publicity. Placards will be placed in drug stores and hospital lobbies.

The drawing card?—A top-notch program with excellent speakers. The morning will be devoted to "The Age of Turmoil"—adolescent problems. Speakers include Prof. Reynold Jensen, head of the department of child psychiatry at the University of Minnesota, on "Problem Children—Do They Exist?"; Judge Thomas Tallakson (tentative), head of juvenile court, on "Alcohol and Delinquency"; Dr. Harold Eberhardt on "Mouth Care for Adolescence"; Dr. Albert Stoesser on "Skin Care of Adolescence"; Judge Lindsey Arthur (tentative) on "Adult Responsibility for Teen Age Driving."

The afternoon program will include talks on rehabilitation of the handicapped by Mr. Schoenbaum, director of the Easter Seal program, and Dr. Frederic Kottke, professor of physical medicine and director of the Rehabilitation Center at the University of Minnesota.

A discussion of cancer will follow, with talks by Dr. Ralph Christensen on "Oral Cancer" and Dr. Stuart Arhelger (tentative) on "Cancer Research," plus a film titled "146,000 Could Live."

The finale of the day is an evaluation of Salk vaccine by Prof. Gaylord Anderson, director of the public health department at the University.

In addition to the program, there will be exhibits set up by several health organizations including the Blood Bank, Tuberculosis Association, and the American Cancer Society. The mobile x-ray unit of the Tuberculosis Association will be in front of the hotel to take chest x-rays.

The result?—Members hope to top the 1954 Health Day attendance record of 3,000!

General Interest

Dr. Gordon M. Martin, Rochester, was named president of the American Congress of Physical Medicine and Rehabilitation at its annual meeting in Detroit, Michigan, early in September. **Dr. Frederic J. Kottke,** Minneapolis, was elected third vice president of the organization.

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Dr. Thomas Johnson, Minneapolis, has been named head of medical services at Stillwater State Prison. Announcement of his appointment was made by Warden Douglas Rigg. Dr. Johnson, a native of Texas, graduated from the University of Minnesota in 1954 and recently completed his internship at Mount Sinai Hospital, Minneapolis.

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Dr. Ralph H. Buesgens, Waterville, has purchased a lot in the city and is in the process of erecting a new medical clinic building.

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"New Tuberculosis Legislation" was the title of a talk by **Dr. Mario Fischer,** public health director for Duluth and St. Louis County, at the Tuberculosis Nursing Council fall meeting of the Minnesota Nursing League on September 19 at Nopeming Sanatorium.

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Dr. Ernest A. Ager, Le Center, narrowly escaped serious injury when his car skidded on a rain-soaked highway, went out of control and overturned while he was returning home from a hospital call recently. Although the car was badly damaged, Dr. Ager escaped with bumps and bruises.

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Dr. J. A. Bargen, Rochester, has been named a member of the medical advisory board of the Sears-Roebuck Foundation. Function of the board is to assist the foundation in the development and administration of programs of interest to the medical profession.

* * *

Dr. R. A. MacDonald, Littlefork, was named vice chairman of a Koochiching County committee for the care of the aged and infirm when the committee held its organizational meeting at International Falls in August. Purpose of the committee is to make recommendations in regard to construction of new facilities for the aged.

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Dr. Thomas A. Peppard, Minneapolis, was presented with the St. Barnabas Hospital bowl award during the hospital's nursing school commencement exercises in Minneapolis on September 2. The award is given each year to a physician in Hennepin County who "by reason of his professional contributions on the basis of medical research, medical achievement or leadership, has become the outstanding doctor of this or other years." Physicians previously honored include **Dr. S. Marx White, Dr. Henry L. Ulrich,** and **Dr. E. J. Huenekens.**

Dr. Douglas L. Johnson moved from Little Falls to St. Paul on August 28. He will live there for the next year while completing a fellowship in ophthalmology at the University of Minnesota.

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Dr. Andrew C. Erlanson, St. Paul, has been appointed pediatrics consultant for the Minnesota Department of Health section of maternal and child health. He recently completed a three-year fellowship in pediatrics at the Mayo Clinic, Rochester.

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Dr. and Mrs. W. G. Benjamin, Pipestone, returned home late in August following a summer tour of Europe.

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Dr. Hilbert Mark, staff member of the Minnesota State Sanatorium at Ah-Gwah-Ching from 1946 to 1951, was recently named director of preventive medicine of Philadelphia, Pennsylvania.

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On a vacation trip to the West Coast, in August, **Dr. J. H. Dokken,** Windom, fractured his arm at Salinas, California, when he fell while being attacked by two dogs during a tour of a new construction project.

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Dr. F. W. Bachnik, formerly with the Adams Clinic in Hibbing and more recently a resident of Rochester, sailed for Europe on September 6 for a tour of European medical centers. On his return he will join Dr. C. B. Cunningham, formerly of the Lenont-Peterson Clinic in Virginia, in the practice of obstetrics and gynecology at St. Petersburg, Florida.

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Presiding over the third general assembly of the Council for International Organizations of Medical Sciences when it met September 30 and October 1 in Paris was **Dr. Maurice B. Visscher,** head of the University of Minnesota's department of physiology.

Dr. Visscher served as president of the second general assembly and is a member of the council's executive committee, which will confer September 28 and 29, and October 2, 1955.

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Dr. and Mrs. Virgil Schwartz left by airplane the first part of August for England. From there they visited Belgium, Holland, Germany, Switzerland, Italy and France. They arrived home the middle of September.

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Dr. Gordon R. Kamman, of St. Paul, addressed the Mid-continent Psychiatric Association at Tulsa, Oklahoma, on September 24, 1955. His subject was "Harmful Effects of Interpretive Psychotherapy in Certain Involutional Depressions."

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Principal speaker at a meeting of the Kiwanis Club in Rochester on August 18 was **Dr. Jan H. Tillisch,** Rochester, who discussed the accomplishments of the First Minnesota Regiment in the Civil War.

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Dr. William F. Braasch, Rochester, retired member of the Mayo Clinic staff, has been named to the University of Minnesota Alumni Association committee of past presidents.

NEW LOCATIONS

Dr. Winfield Williams, who recently completed a residency in internal medicine at the Minneapolis Veterans Hospital, has become associated with the Park Region Medical Center in Fergus Falls.

* * *

Dr. Carl N. Harris, senior physician of the Adams Clinic, Hibbing, has announced that **Dr. Laurence G. Thouin** has become associated with the clinic. Dr. Thouin, a native of Hibbing, was an instructor in obstetrics and gynecology at the University of Utah Medical School and has been in private practice in Honolulu, Hawaii, for five years.

MINNESOTA BLUE SHIELD

In the first six months of 1955, the amount paid to doctors for services rendered Blue Shield subscribers was 15 per cent greater than the payments during the same period of 1954. The total amount of Blue Shield claims paid from January 1 through June 30 of this year is $2,837,170. For the same period in 1954, the amount paid by Blue Shield for services rendered by doctors was $2,465,876. This increase in payments represents $371,294 more during the first six months of 1955 than during the same period of 1954. The average monthly Blue Shield payment during the first six months of 1954 was $410,000. In the same period of 1955, this average is $62,000 greater, or $472,860 per month.

One of the reasons for the increases in Blue Shield payments in 1955 over the comparable period in 1954 is the fact that a greater number of services were rendered by doctors to Blue Shield subscribers. For example, during the first six months of 1955, Blue Shield provided payment for 94,785 services rendered by doctors. In the same period in 1954, 81,679 services were provided Blue Shield allowances. This shows an increase of 13,106 services in the first six months of 1955 over the same period in 1954. This increase in the number of services paid is greater than can be explained on the basis of the enrollment increase.

In further study of Blue Shield payments, allowances for medical care during the first six months of 1955 are 17 per cent greater than during the same period in 1954. Surgical benefits increased 11 per cent and obstetrical payments almost 18 per cent. Blue Shield payments for such ancillary services as diagnostic x-rays, anesthesia, endoscopic examinations and consultations were 33 per cent greater during the first six months of 1955 than during the same period of 1954.

Inasmuch as the Blue Shield contract and Schedule of Payments were revised, providing additional and increased benefits effective July 1, 1955, it is expected that payments to doctors for services rendered during the last half of 1955 will surpass the amount paid in any previous six-months period.

Book Reviews

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

SURGERY OF THE SMALL AND LARGE INTESTINE. Charles W. Mayo, M.D., Section of Surgery, Mayo Clinic, Rochester, Minnesota; Professor of Surgery, Mayo Foundation, University of Minnesota. 340 pages. Illus. Price $9.00. Chicago: Year Book Publishers, Inc., 1955.

The long awaited companion book to the Year Book series on surgical technique is now available. It is a concise book with excellent illustrations of the more common surgical operations performed upon the small and large intestine and rectum. Included are many specific details regarding diet and preoperative and postoperative care that many similar books leave unclear or omit entirely. For example, the method of bowel preparation for colon surgery at the Mayo Clinic is described explicitly.

Preceding each segment of the gastrointestinal tract being considered, is a short, lucid discussion with illustrations of the blood supply, lymphatic drainage, and general anatomic relations. These few pages are exceedingly helpful and furnish the necessary background for an understanding of the operations to be described.

After reading that portion of the book pertaining to intussusception, one is left with the idea that the treatment of choice for intussusception in infants as well as adults is resection. In infants, there usually is no anatomical basis for the intussusception and I believe that resection in these cases is usually not necessary; if done routinely, they will lead to an undesirable mortality.

I would also differ with the author regarding the management of Gross' Type III imperforate anus with recto-urinary tract fistula. In most cases, a one-stage combined abdomino-perineal operation can be done primarily, rather than a preliminary colostomy followed by a waiting period of four to five years before definitive surgery.

The section on regional enteritis is exceptionally well done and furnishes a practical approach to the surgical treatment of a most perplexing disease. Much of the valuable data presented was obtained from the recent excellent review of 600 cases of regional enteritis at the Mayo Clinic by Van Patter.

The author's vast personal experience in lower gastrointestinal surgery has undoubtedly been the major source of information for this book. Therefore, the great value of this book is to the young surgeon who is given a step-by-step practical approach to the surgical management of lesions discussed. I would recommend the book most heartily.

Kenneth A. Storsteen, M.D.

Most multiple primary tumors arise in the bowel, bladder, and skin.

The Challenge of Arteriosclerosis

Challenge of Arteriosclerosis to Medical Education

WILLIAM F. MALONEY
Minneapolis, Minnesota

WHEN first assigned this title, my reaction was one of amazement. Did the program committee really think that our medical schools are afflicted with arteriosclerosis? Did they really want me to deliver a tirade about the hardening of the arteries of our educational system? This did not seem appropriate, nor did it seem that a degenerative process had begun its inexorable progress in the body of our medical educational system. I recognized, of course, (as we all do) that only by keeping curriculi flexible and adaptable, and by keeping faculty minds open to new educational methods and ideas in this swiftly changing era of medicine, can our schools fulfill their obligation to society; that only by conscientious effort to remain aware not only of the changes in medical knowledge, but also aware of the changing place of medicine and the physician in our society can the schools prepare doctors who will adequately carry out the task of ever-raising the health level of our people. Inelasticity and occlusion are not unknown in medical education, but this is a relatively rare occurrence, as distinguished from its incidence in the population.

But since arteriosclerosis is the subject of this symposium, I ruminated a bit about our medical educational system in terms of just how adequate, adaptable and dynamic it had been in dealing with this subject matter. It was soon apparent that the subject matter of the condition arteriosclerosis itself presented a tremendous challenge to teaching. It was also apparent that medical education had met this challenge well to date. And finally, it was apparent that the most appropriate discussion for today was one that centered around the challenge arteriosclerosis represented in terms of producing physicians who were knowledgeable about this subject.

Immediately obvious, is the fact the arteriosclerosis occupies many times more pages in the 1955 editions of leading textbooks of medicine than in the 1925, and that curriculum time devoted to this material has increased in several instances from as little as zero to a significant percentage during a comparable period. It is apparent also that the study of this entity now cuts across virtually all departmental lines—both basic science and clinical.

For purposes of discussion, let me propose one description of arteriosclerosis. It may be described as a condition of unclear etiology, involving fundamental physiological and anatomical alterations that are influenced by physical, chemical, genetic, environmental and psychological factors. A high incidence, a wide variety of clinical manifestations, and prolonged disability are characteristic. Arteriosclerosis as thus described is at every point a challenge to medical education.

The reforms in medical schools and medical education, stimulated by the Flexner Report in 1910, ushered in what has come to be known as "scientific" medicine. Biology, chemistry and physics have come to occupy central positions in medical teaching. Dr. Donald Anderson, the

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From the University of Minnesota Medical School. Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

former secretary of the Council on Medical Education, has considered "the thoroughness with which we have sought to apply the facts and techniques of the basic sciences to clinical medicine" as being "the outstanding feature of medical education in this era." In his words, "The transformation of clinical medicine into a real science, and conversion (at least partially successful) of the physician from an empiricist to a scientist who daily applies scientific methods and the techniques of the laboratory to the problems presented by his patients, is a development to which must be credited most of the gains in medical education in the past half century."[1]

The emphasis on basic experimental science in the pre-clinical years of medical education continues to be appropriate, particularly with regard to the subject matter of this symposium. The elucidation of the fundamental alterations in the physiological processes, and of the etiology of the anatomical alterations known to occur with arteriosclerosis can continue to be accomplished only by individuals thoroughly familiar with basic principles and techniques in the natural sciences, and with the ability to use this knowledge critically and with originality.

However, as defined by the Association of American Medical Colleges, it "should not be the aim of undergraduate medical education to present the complete, detailed, systematic body of knowledge concerning each and every medical and related discipline." Rather, emphasis must be placed on "learning fundamental principles applicable to the whole body of medical knowledge; of establishing habits of reasoned and critical judgment of evidence and experience; and developing an ability to use these principles and judgments wisely in solving problems of health and disease."[2]

Thus, the continued emphasis on basic science is important, but the emphasis on major principles is not only more productive in preparing young minds for original thinking, but also has been made absolutely essential by the continuing rapid accumulation of scientific fact and detail which threatens to overwhelm the educational enterprise.

Factual basic science knowledge is accumulating rapidly in the field of arteriosclerosis. This raises an even more fundamental problem. Just which "facts" does one teach? No educator that I know of has an entirely satisfactory solution to this quandary. There is, however, educational value in the very quandary itself. It serves brilliantly to assist in making students critical of experimental evidence, healthily skeptical of "facts," cautious in their judgment, and respectful of the magnitude of the problems to be solved.

Yet another facet of this problem must be dealt with. The accumulating evidence that hereditary factors, psychological elements, living habits and social forces may affect this process so increases the scope of the orientation necessary as to challenge the limits of a four year curriculum. Here again, undergraduate medical education does not and cannot attempt to provide a detailed presentation of all these factors in addition to the physical, chemical and biological agents and alterations that may affect the human being.

However, the recognition of the relevance of these other factors, in addition to the basic disciplines of "scientific" medicine, has brought to medical education a new period of development called by some "comprehensive" medicine. George Packer Berry, dean of the Harvard Medical School, expresses it in this manner:

> "The trend today is toward an internal synthesis of the scientific facts about man, and an expansion of our view to embrace what for simplicity can be described as the psychological and sociological facets of the patient. It is not so much that these aspects of the patient have been ignored in the past—they have been inherent in the doctor's appraisal of the patient since the time of Hippocrates—but the newer disciplines of psychiatry and social anthropology and allied fields now provide us with a great deal more insight into the nature of disease, into man's relationship with man, and the factors that make for healthy community life. To equip our doctors in training to meet the new challenge . . . we must broaden the base of our training, expanding the concept of scientific medicine to that of comprehensive medicine, where all the benefits that have accrued . . . will be retained with the addition of new studies of the patient in his socio-psycho setting."[3]

Degenerative vascular disease contributes a major and ever-increasing percentage of the problems with which comprehensive medicine concerns itself. The development in the student of a suitable orientation toward these problems, without interfering with a sound training in medicine as it has been developed on the basis of the natural sciences, is a difficult problem.

An increasing amount of experimentation has been taking place in medical education in recent years, in an effort to deal with this problem. Dr. Herman Weiskotten summarized this in a paper read at the Annual Congress on Medical Education two years ago. I quote him:

> "Frequently such new subject matter has not appeared to fit readily into the program of existing departments. Attempts have been made to present it to the students in didactic form, as separate courses detached and isolated from the pre-existing curriculum content. Such attempts have not been too successful, and the schools have been faced with the problems of attempting to incorporate such new subject material into the individual experiences of the student in the integrated programs of the basic medical science and clinical departments.
>
> "Here again, the solutions of these problems have required not only continued re-evaluation of the course content of the various departments, but also continued experimentation as to how in a given school important new subject matter and viewpoints can be best integrated into the basic training of the student.
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> "Just as individual faculty members are continually conducting research for the advancement of medical science in their particular fields, as faculty groups they should continually be conducting experiments in the field of medical education—for this is the primary responsibility of our Medical schools and must always be recognized as such."[4]

A natural outcome of the concept of comprehensive medicine has been a broader interpretation of medicine's role in the community. The possibility of control of some of the factors responsible for arteriosclerosis as our increased understanding suggests, has caused the science of preventive medicine to assume new and greater importance.

Another addition to the medical educational program has been the teaching of the science and art of rehabilitation. The prolonged disability which most commonly results from the occurrence of arteriosclerosis in several of its forms presents a challenging problem. It has been clearly demonstrated that rehabilitation has much to offer in this field.

Again, the objectives of medical education, as enunciated by the Association of American Medical Colleges, carry beyond teaching diagnosis and treatment to "helping the student to achieve basic skills in working in the medical team of physician, nurses, social workers, physical therapists and others; and in working in the community team of official and voluntary institutions and agencies providing health services; as well as that of helping the student to develop sound attitudes for basing the plan for cure, rehabilitation or adjustment to permanent handicap upon the joint efforts of the medical team and the patient."[2]

Not all educators have broadened their horizons to understand or teach in terms of comprehensive medicine. However, arteriosclerosis in its implications for the health of the total community will eventually be a principal force in compelling them to greater perspective. On the other hand, I submit that this nation's medical schools, in carrying out their functions of teaching, research and service, have for the most part accepted challenges so well that they have given themselves the greatest challenge of all—an ever-increasing prevalence of degenerative vascular disease as a result of the ever-lengthening life span. As our own professor emeritus of pathology, Doctor Bell, once remarked, "The only thing left to die of will be arteriosclerosis." This challenge should be enough to keep the arteries of medical education elastic and patent.

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3. Berry, George Packer: Committee Report, First Teaching Institute of the Association of American Medical Colleges (Oct.) 1953.
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A chronic disease or condition may be defined as one which lasts a long time or at least too long and which, while it is present, prevents the individual from operating at his optimum efficiency. The cause may be a disease, as in the case of poliomyelitis, tuberculosis or syphilis of the central nervous system. It may be a physical or mental injury or dysfunction as in the case of cerebral palsy, an accidental amputation, mental retardation, or excessive exposure to radiation.—DANIEL BERGSMA, M.D., *New Jersey Pub. Health News*, April, 1955.

Role of the National Heart Institute in Meeting the Challenge of Arteriosclerosis

JAMES WATT
Bethesda, Maryland

IN August, 1948, the Congress of the United States enacted legislation creating the National Heart Institute, a division of the National Institutes of Health, Public Health Service. As part of this legislation, the Congress specifically charged the National Heart Institute with the responsibility of improving the health of the people of the United States as related to disorders of the heart and circulation. The National Heart Institute was authorized to do this through the carrying on of research, by assisting and fostering research and other activities by public and private agencies, by providing training for research workers and clinicians, and by assisting the states and other agencies in the use of the most effective methods of prevention, diagnosis, and treatment of diseases of the heart and circulation.

In carrying out these responsibilities, the National Heart Institute has utilized several programs. Today, I would like to review for you some of the accomplishments, some of the frustrations, and some of the dreams that these approaches have nurtured in one area of cardiovascular disease—arteriosclerosis.

The largest of these programs, in terms of man-hours and dollars expended over the past six years, has concerned itself with the encouragement and support of research. The awarding of research grants to universities, hospitals, and laboratories throughout the country and the carrying on of research in the laboratories of the National Heart Institute at Bethesda, Baltimore, and Framingham are the main facets of the National Heart Institute research program.

Upon recommendation of the National Advisory Heart Council, the Surgeon General has awarded, since 1948, approximately 4.5 million dollars in grants for the support of research related to arteriosclerosis to laboratories other than those of the Public Health Service. These awards have provided funds for the organization and operation of new laboratories, for the stimulation of increased effort in already established laboratories, and for the encouragement of introducing new views and new skills to the basic and applied problems related to arteriosclerosis.

The results of these efforts are well documented in scientific and medical journals. A major step forward is the realization and demonstration that arteriosclerosis, particulary atherosclerosis, is not an inevitable part of aging, but rather a pathologic process afflicting some people at a relatively early age, others practically not at all. To study this pathologic process, techniques have been developed to produce atherosclerotic lesions in a variety of experimental animals. The utilization of these animals has permitted the establishment of controlled studies in which some of the variables associated with this disease can be studied separately. The artery wall has been introduced as an active factor in the production of the lesion; the role of the cellular and non-cellular elements circulating in the blood — phagocytes, cholesterol, phospholipids, lipoproteins, and neutral fats—are being studied; the physical forces—hypertension, blood eddying, friction, adsorption, and surface tension—are being looked at and evaluated in terms of this problem; the role of the liver, of the endocrine glands, of the gastrointestinal tract is being scrutinized. Some of the newer research techniques as used in biophysics, in ultamicroscopy, and in enzyme chemistry have yet to be extensively applied to the area of arteriosclerosis; the same can be said of some of the older established techniques, those of the geneticist, the epidemiologist, and the environmental engineer.

From the National Heart Institute, National Institutes of Health, Public Health Service, Department of Health, Education and Welfare, Bethesda, Maryland.

Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

In the laboratory of the clinical scientist, new methods of diagnosis and treatment are under surveillance. As yet, excluding histological study after biopsy, we regretfully realize that the only generally accepted method of diagnosis depends upon the onset of clinical symptoms or signs. The search for reliable tools or methods for preclinical diagnosis is being pursued, and we hope the results of these efforts will be available to the clinician before long. In the area of therapy, exciting surgical techniques for the repair of blood vessels have been reported. The artery bank is now a reality; the use of prosthetics for the replacement of injured blood vessels is no longer a dream. There have been many disappointments in drug and hormonal therapy for the prevention or treatment of atherosclerosis, but with each failure a glimmer of a new possibility arises. Techniques for the rehabilitation of those afflicted with this disease are slowly being developed.

As part of the research effort directed against arteriosclerosis, scientists working in the laboratories of the National Heart Institute are pursuing similar problems. A coordinated plan of investigation already under way at Bethesda has been greatly accelerated by the establishment of the Clinical Center. Here, preclinical and clinical scientists are able to integrate their activity in specially selected patients for periods of time considerably longer than those usually available to workers in the average hospital situation. Having animal and patient laboratories in close approximation has permitted parallel controlled investigations to be initiated. As yet, many of our laboratories are just now being completed, equipped, and staffed, but in those already in operation the mechanisms of lipid transport and metabolism are being studied. The relationship of proteins, heparin, clearing factor and certain drugs and hormones to the development of atherosclerosis has commanded our interest.

Many of you are aware of the heart disease epidemiology study being conducted at Framingham, Massachusetts. There, in cooperation with local physicians and community health agencies, the genetic and environmental factors believed to be significant in the development or progression of arteriosclerosis and hypertension are being investigated. About two-thirds of the adults in this community between the ages of thirty and fifty-nine, including several thousand volunteer participants selected by random sampling methods, are part of the study. These persons will be followed over an extended period, during which data of pertinent constitutional or conditioning factors will be recorded and analyzed.

These examples reflect a few of the ways in which the research grant and direct research programs of the Institute are assisting in the development of new knowledge about arteriosclerosis.

Another area of considerable interest to the National Heart Institute has been the need for additional research workers and clinicians leading to the establishment of its research fellowship and clinical traineeship programs. These training programs at the predoctoral and postdoctoral levels aid young men and women in obtaining training in laboratories and clinics concerned with arteriosclerosis. During this training period, a living stipend, tuition, and travel funds are made available. For the more experienced man, a special fellowship or traineeship can be provided. These awards permit the trainee to assimilate new techniques and obtain instruction in the disciplinary background needed to search out and investigate meaningful problems or to more skillfully utilize the tools of clinical medicine for better patient care.

Recognizing also the need for other training programs for scientists and clinicians, the Institute supports an undergraduate and graduate training grant program. Through this program, funds are made available to universities, hospitals, and laboratories for the establishment, expansion, or improvement of instruction in areas related to arteriosclerosis or other cardiovascular matters. These funds have been used for the support of trainees, of staff, and for the provision of the facilities needed to maintain these programs of instruction.

Thus, by means of the research fellowship, the traineeship, and the training grant programs, we hope to maintain and encourage an increase in the pool of highly skilled research workers and clinicians. We at the National Heart Institute consider these training programs essential, for we realize that the answers to the problem of arteriosclerosis and their application to the

(Continued on Page 742)

Life Insurance Looks at the Arteriosclerosis Problem

WILLIAM P. SHEPARD and
HERBERT H. MARKS
New York, New York

THE awareness and the concern of the life insurance industry over the problem of arteriosclerosis is perhaps best indicated by the Life Insurance Medical Research Fund organized ten years ago. Although the purpose of the fund is to support medical research in any field, its resources have been devoted to research bearing on diseases of the heart and arteries and fundamental related problems. In addition to supporting research, the fund also fosters the development and training of research personnel of high calibre by financing graduate fellowships. Over 140 life insurance companies, which account for 90 per cent of the life insurance in force in the United States, now contribute to the fund, which to date has appropriated more than $7,200,000 for its work. The companies are now contributing nearly $1,000,000 annually. While these sums may seem small in relation to the volume of business and resources of insurance companies, it must be remembered that the life insurance business is rigidly supervised and subject to strict laws with regard to expense limits. To date, 247 research programs have been supported by grants and 302 fellowships have been given to young men and women to enable them to obtain research training in the field of cardiovascular disease. Research has thus been aided in 110 institutions, most of them in the United States and Canada.[1,2]

Since I have been asked to talk on the problem of arteriosclerosis as seen from the point of view of life insurance, I have purposely picked out for my discussion certain aspects on which information available from life insurance records is particularly useful. These are (1) the extent of the problem in terms of mortality and morbidity, (2) the trends in mortality, (3) facts relating to prevention and control, and (4) facts relating to prognosis.

The facts I present will, of necessity, relate generally to aggregates such as cardiovascular-renal diseases or heart disease because conditions due to or associated with arteriosclerosis are not combined in a separate aggregate in the statistics on mortality and morbidity. Arteriosclerosis is, however, the predominant etiological factor in the categories covered in my material.

It is no secret that a major reason for the concern of the companies is that from the standpoint of claims, disorders associated with or due to arteriosclerosis constitute a large and increasing proportion of the total. This holds not only for death claims, but to an increasing extent for temporary and permanent disability and hospitalization.

Mortality from "Arteriosclerosis"

More than two billion dollars in death claims were paid to American families by life insurance companies during 1954.[3] While we do not know exactly the amount of money paid out for the various causes of death, cardiovascular-renal disease in 1954 accounted for well over half of the deaths among insured persons from all causes, and for 60 per cent or more of the deaths from disease. The major proportion of these deaths reflects the toll from arteriosclerosis. In addition, the condition is a factor in deaths from diabetes and certain other causes, especially in older persons.

Figures on deaths in 1952 among persons insured under *ordinary* policies, presented in Figure 1, show that cardiovascular-renal diseases accounted for 57 per cent of the total, the major item being 42 per cent from heart disease. In contrast, in 1927 cardiovascular-renal diseases

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From the Metropolitan Life Insurance Company, New York, N. Y.

Presented by Dr. Shepard in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

accounted for only 34 per cent of the total, and heart disease for only 17 per cent.[4]

In the Metropolitan's own experience in 1954,[5] of the total of $367,000,000 disbursed on account of deaths, 207 millions were accounted for by diseases of the cardiovascular-renal system. Of the total, more than 140 millions were paid specifically for deaths due to arteriosclerotic and degenerative heart disease and arteriosclerosis.

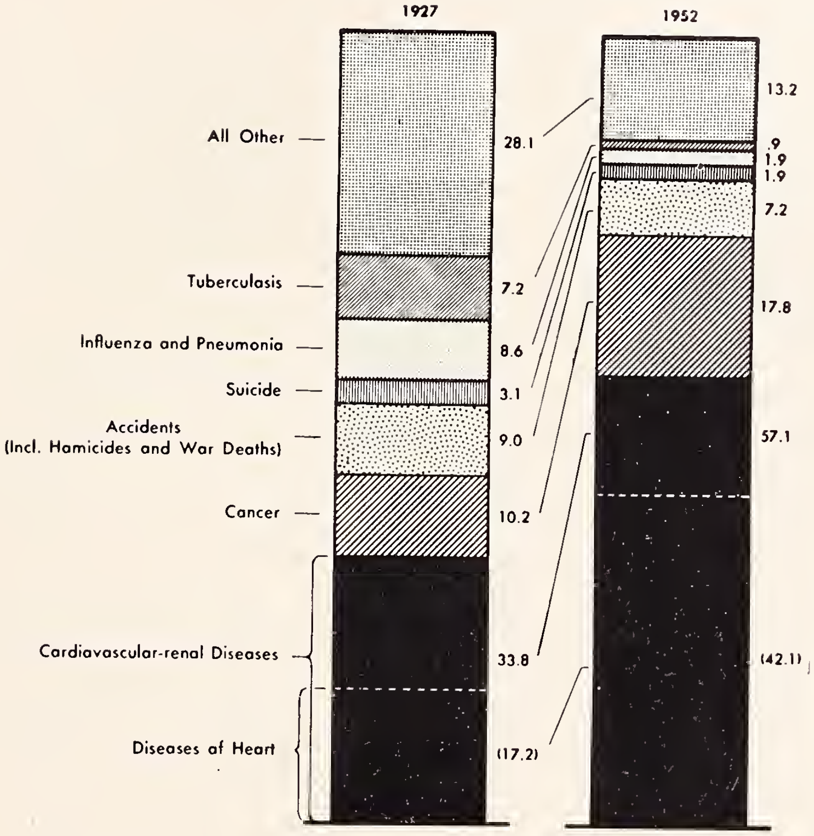


Fig. 1. Per cent distribution of death claim payments by cause of death. Ordinary insurance. (All companies reporting to Life Insurance Association of America).

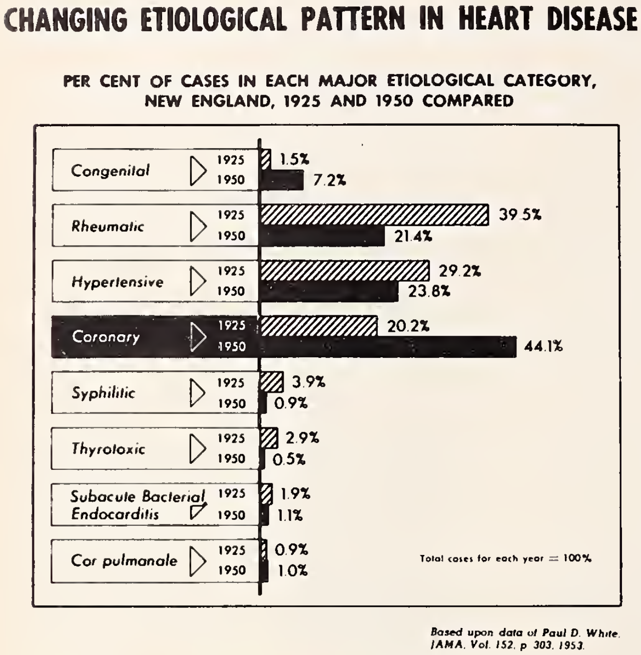
These figures present the current picture. It is important also to look at the over-all picture as measured by the chances of eventually dying from diseases of this nature. Under conditions prevailing in the general population in recent years, the chances of a white man aged twenty dying eventually from chronic or degenerative diseases are 87 in 100.[4] Twenty-five years earlier, this proportion was 75 per cent. The major items contributing to these high figures are diseases of arteriosclerotic origin. The latest analysis of this type by specific causes of death, based upon 1948 data, showed that for cardiovascular diseases this probability at age twenty for white men was 59 in 100.[6]

Morbidity from Heart Disease (Non-rheumatic)

Detailed statistics on morbidity from life insurance company records are only now beginning to be developed. Consequently, available figures are rather fragmentary. Among our own employees, disability due to heart disease, excluding rheumatic heart disease, accounted for 8 per cent of the disabilities lasting eight days or longer among men during 1954, but for 20 per cent of the time lost. At ages over fifty-five the number of cases of disability from heart disease was about one-eighth of the total, but the time lost was more than one-fourth of the total. A recent study of causes of hospitalization among the male personnel at ages sixty and over showed that diseases of the cardiovascular system accounted for more than 40 per cent of the non-surgical cases.[7]

Trend of Mortality—Cardiovascular-renal Disorders

Let us look next at the available information on the trend of deaths from cardiovascular-renal disease in life insurance experience. For this purpose I cite the long-term series of death rates among *industrial* policyholders of the Metropolitan Life Insurance Company. The facts given here will be based upon death rates adjusted to allow for changes in the age distribution and upon rates specific for age, in order to eliminate the effect of the increasing population of older persons on the situation. Among white males at all ages combined the death rates have shown a generally downward trend, although the rate of decline has been rather small in recent years. At ages 45 to 54, however, there has been scarcely any improvement among males during the past two decades. In contrast, the trend for females, regardless of age, shows distinct improvement over this period.



Based upon data of Paul D. White, JAMA, Vol. 152, p 303, 1953.

Fig. 2. Changing etiological pattern in heart disease.

Arteriosclerotic Heart Disease

It is of interest next to look at the available facts on heart disease specifically in terms of etiology as revealed in our claim records. This is brought out in an analysis of claims among *industrial* policyholders of the company in 1950-1952.[8] Among males at as early an age as thirty-five, arteriosclerosis is the predominant type in these fatal cases and accounts for more than 60 per cent of the cardiac deaths at ages beyond thirty-five. In addition, many of the cases classified as hypertensive are associated with arteriosclerosis, and many of those lacking information on etiology are probably of arteriosclerotic origin.

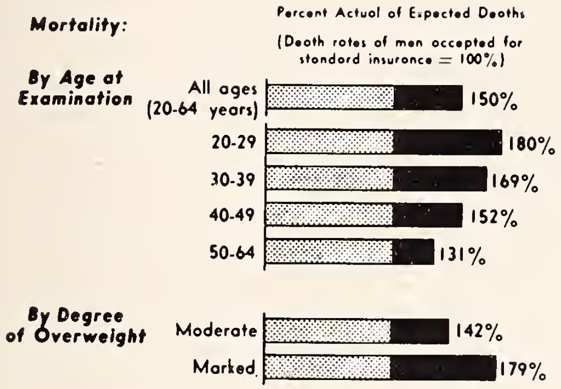
One of the gravest aspects of this whole problem is the excessive mortality among males. This shows up in fairly early adult life. Whereas in the experience among our *industrial* policyholders, the age adjusted mortality rate at all ages combined from arteriosclerotic heart disease among males is about two and a half times that among females, the ratio at ages 35 to 44 rises to 6 to 1.[8]

Unfortunately, the classification of deaths by cause in the past was rather inadequate as regards etiological factors. Accordingly, for earlier periods there are no data comparable to those just given. Perhaps the best and most succinct presentation of the changing etiological pattern in heart disease over the last quarter of a century is that portrayed in the figures published by Dr. Paul D. White a couple of years ago.[9] This is shown in Figure 2, which is based upon data obtained from cooperating physicians in New England on cardiac patients seen in clinic and private practice. The major change is a doubling of the proportion of cases due to arteriosclerosis to a point where it comprises nearly half the total cases seen.

While much thought has been given to the factors underlying the relatively unsatisfactory state of affairs as regards arteriosclerotic diseases, we have very little objective proof as to what has brought it about. Several hypotheses have been formulated, but these still require careful and prolonged testing.

Relation of Body Build to Mortality

In the circumstances, it might be thought that means of prevention and control of arteriosclerotic disorders would be out of the question. This is not altogether true. At least one important resource is available to us, as revealed by the experience of life insurance companies. This experience is embodied in a series of studies made at irregular intervals going back to the beginning of the century, the first of which included cases insured as early as the years immediately after the Civil War. These studies, it should be emphasized, are essentially long term follow-up studies involving observations on large numbers of persons. All of them have shown consistently that overweights have a mortality significantly higher than persons of lesser weight, and that this excess mortality is primarily due to cardiovascular diseases. The labels used in describing these cardiovascular conditions have changed over the years, but the pattern of mortality from them is most consistent.[10]



Experience of Metropolitan Life Insurance Company
Substandard Ordinary Issues, 1925-1934, traced to 1950

Fig. 3. Mortality of men rated for overweight.

It will suffice to illustrate the point from the recent experience of the Metropolitan Life Insurance Company based upon approximately 50,000 men and women who were charged an extra premium for their life insurance simply because they weighed too much.[11] These persons were first class risks in all other respects and were in safe occupations. The experience covers the period 1925 to 1950. As Figure 3 shows, the mortality among the men in this experience was 50 per cent higher than that among men accepted for standard insurance, that is, insured at the usual premium rates. The mortality was significantly above normal in every age group at issue of the insurance. Furthermore, mortality increased with the degree of overweight. The overweight women in the study showed about the same degree of excess mortality as compared with female standard risks.

In both sexes the bulk of the excess mortality was due to cardiovascular-renal diseases, primarily of arteriosclerotic origin. As in earlier studies, the greatest relative excess was in the mortality from diabetes, and today this primarily reflects the premature development of arteriosclerotic complications among diabetics.

Effect of Weight Reduction

Even more significant are the findings in a supplementary study of this experience.[11] We were able to identify those persons who because of reduction in weight were subsequently granted insurance at standard rates. As Figure 4 shows, the mortality among such persons was appreciably lowered—about one-fifth among the males and one-third among the females. A prime contributing factor was the reduction in the relative mortality from heart disease.

Experience on Persons Rated for Moderate Obesity Who Received Lower Ratings After Weight Reduction Compared with General Experience on Cases Rated for Moderate Obesity.

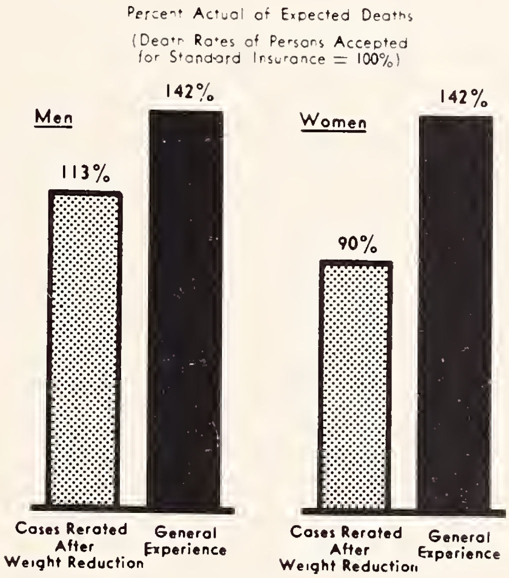
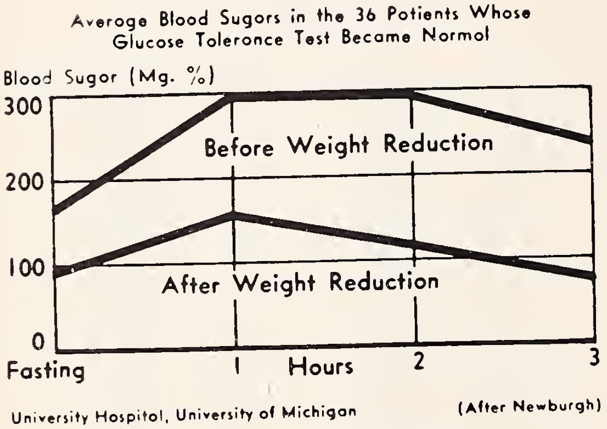


Fig. 4. Effect of weight reduction on mortality.

So far as we have been able to ascertain, this is the first objective and measurable evidence of the benefits of weight reduction among presumably healthy overweights, although the medical literature contains many statements, based upon clinical impressions, on the improvement in the physical condition of overweight patients who have reduced. There are some more specific clinical studies of such effects on persons with various types of impairments which have a bearing on arteriosclerosis. Thus, the studies of Newburgh at the University of Michigan Hospital, which are summarized in Figure 5, have shown that reduction in weight of obese diabetics greatly improves glucose tolerance to the extent that even those on insulin may get along with a lower dosage or even get along without insulin altogether if the weight is kept down.[12] Parallel results have been reported by others. Such diabetics who regain weight show a loss of tolerance and frequently an increase in insulin dosage.[13] Studies have likewise been made of the effect of the reduction of weight in hypertensives.[14,15] These have shown a lowering of the blood pressure level in many cases.

Change in Tolerance	Number of Patients
Total	47
Became normal	36
Improved	5
Unimproved	6


Average Blood Sugars in the 36 Patients Whose Glucose Tolerance Test Became Normal

University Hospital, University of Michigan (After Newburgh)

Fig. 5. Glucose tolerance test results among obese diabetics after reduction to normal weight.

Prognosis in Heart Disease

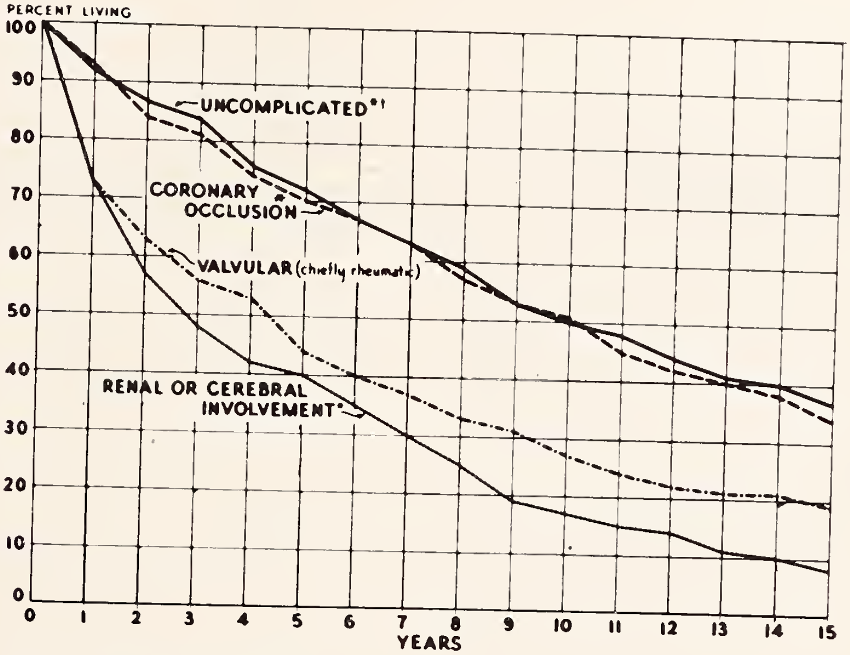
Life insurance records of an entirely different character provide some information on the long-range outlook for men with arteriosclerotic heart disease. For example, Robb and Marks[16] studied the after-history of two groups of male policyholders of the Metropolitan Life Insurance Company who had total and permanent disability from heart disease and were eligible for certain insurance disability benefits on that account. The first group consisted of 540 men admitted to disability between 1934 and 1936 who were traced to the anniversary of admission in 1952. Thus, for them there is a minimum follow-up period of more than fifteen years. The second group consisted of 649 who had recovered from such disability between 1925 and 1949 and traced to the 1952 anniversary; for them the period of follow-up ranges from a minimum of three years to more than twenty years.

Figure 6 shows the survivorship record of the cases admitted to disability in 1934-1936. Of those with arteriosclerotic or hypertensive heart disease who had no other vascular or no renal complications and who at the time of admission to disability had not experienced an acute coronary episode, about half survived ten years or longer after becoming disabled, and more than a third were living at the end of fifteen years. The presence of cerebral or renal complications adversely affected the results, but even in this group 40 per cent survived five years, and 17 per cent, ten years or longer. The proportion of survivors was generally greater for men who were between 40 and 49 when disabled than for those who were 50 to 64. The difference, however, was less than expected when account is taken of the normal difference in mortality by age.

The recovered cases were classified somewhat differently in that attempt was made to segregate those disabled specifically as a result of arteriosclerotic heart disease. These cases were further subdivided according to the presence or absence of occlusion and also according to the reported presence of hypertension. It was found that among cases with occlusion, where there was no record of hypertension, the proportion surviving at the end of five years was 80 per cent, and at the end of ten years nearly 60 per cent. The record was somewhat poorer among cases with known hypertension. The smaller experience on cases without a history of occlusion was on the whole somewhat better.

Among the recovered cases with a history of occlusion, the survivorship record was slightly better for those fifty and over at recovery than for those between ages 40 and 49. The reverse was true in cases without a history of occlusion but this experience was smaller in volume. When comparison is made of the two age groups according to the presence or absence of a history of occlusion, it is found that on cases with occlusion the survivorship record was as good as that in cases without occlusion up until the fourth year. Subsequently the latter showed the higher survivorship rate. At ages fifty and over the results in the two groups were generally similar.

While the mortality in all of these groups, as compared with that of standard risks generally, is very considerably elevated, nevertheless, it is clear that a significant proportion of these men do live many years after the attack and often are able to resume a reasonably good level of activity, even to the extent of going back to work and continuing at it.



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*Arteriosclerotic and hypertensive.
†Excluding those with coronary occlusion and with renal or cerebral involvement.

Fig. 6. Survivorship of white men disabled by heart disease. Metropolitan Life Insurance Company, Ordinary Department, disability cases reported and/or admitted in 1934-1936 and traced to anniversary in 1952.

These results are in substantial conformity with those based upon the experience of the Mutual Life Insurance Company, as reported by Waldron and Constable.[17] They confined their study to cases of proved myocardial infarction causing disability. The diagnosis was based for the most part on electrocardiographic findings. Their study, which covered the period 1934-1949, included 1,551 cases. The subsequent mortality was unquestionably high. The average expectation of life for those 30 to 39 at onset was estimated at 11.5 years; for those 40 to 49 at onset, it was 10.5 years; and for those 50 to 60 at onset, 8½ years. All these figures are much below the average for the age but are distinctly counter to the widely-held concept that the remaining lifetime of persons following myocardial infarction is very brief.

Summary

To sum up, the insurance industry has a real stake and real concern in the problem of arteriosclerosis, because arteriosclerosis is responsible for more deaths than any other form of cardiovascular disease, and is frequently a deleterious factor in other disorders characteristic of middle and later life. An earnest of the deep and continued concern of the industry in this problem is the establishment of the Life Insurance Medical Research Fund, the resources of which during the ten years since its founding have been devoted entirely to problems in the field of cardiovascular disorders. Insurance records are a usable and accessible source of information on many aspects of the problem, especially from the long range point of view, and have been the basis of many important scientific studies. I have reported here what our mortality and morbidity records reveal with respect to the dimensions of the problem, its characteristics and its trends. I have cited life insurance studies which have shown that, as a practical measure, control of body weight is probably the most significant and readily available approach in attacking the premature development of arteriosclerotic disease. These records have provided the best available and most concrete evidence that weight reduction pays. I have been able to give you also from our studies of life insurance records, facts on the longevity of persons with arteriosclerotic heart disease that have been useful both to physicians and public health workers.

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NATIONAL HEART INSTITUTE

(Continued from Page 735)

patient will depend upon a continuing supply of trained young men and women.

Another important approach to problems of arteriosclerosis has been through the heart disease control program. Funds are here made available through awards to state and territorial departments of health for the prevention, diagnosis, and treatment of diseases of the heart and circulation. Through this program, the health officer of the community is assisted in the development of needed health facilities. Working with local medical, scientific, and community organizations, the health officer can tailor a program to the specific needs or potential of the local community. It is through this workshop of the city, county, and state that the health needs of the individual are expressed and the final results of scientific investigation put to the acid test. The health officer and the community health program are thus aided in working with the individual, who is after all the beginning and the end of all health programs.

Thus, by the conduct and support of research and training and public health activities, the role of the National Heart Institute in meeting the challenge of arteriosclerosis, and other cardiovascular problems, is played. In principle, in law, and as administered, this role has been conceived and carried out as one of partnership in a great nationwide endeavor against diseases of the heart and circulation. Those concerned with the National Heart Institute in 1948 when it was established were proud to see it join the federation of interests then mobilizing for a comprehensive effort against heart disease. Those concerned with it today are, I believe, just as proud of the gains the partnership has won against arteriosclerosis and of the strong promise of greater advances in the coming years.

Strategy and Tactics of Research in Arteriosclerosis

IRVINE H. PAGE
Cleveland, Ohio

IN the past decade research has become almost "big business." And during this period research in arteriosclerosis has expanded phenomenally. It is an interesting commentary on physicians and investigators that the most common fatal disease known to man, described 5,000 years ago in Mesopotamia, is only now being studied, not merely from the viewpoint of its effects, but its causes and mechanisms. Clinicians have been content too long to observe its ravages without even a nod given to attempts at understanding the why and how of the disease. It seems that a latent period of 4,990 years is a pretty fair one.

Where are we in our efforts to understand arteriosclerosis? Well, certainly not very far. Discovery in general can, I think, be divided into four stages. The first is the period of uncoordinated fact collecting — the naturalist or "butterfly net" period. This is the period of magnificent indifference; in it lie the seeds of discovery but the inner structure is barely discernible. The second is the period of controversy—the "rhubarb stage" of the ball game. This stage is often called one of "scientific controversy" to make it sound less like the rhubarb that it really is. It has the merit that there is probably something worth arguing about if this stage is reached. It is healthy so long as young investigators understand its nature and not take it, or themselves, too seriously. The third is the period of definition and clarification—the "white of an egg stage" because someone clarifies the problem by precipitating the irrelevant sediment. The fourth and last is the period of solution or fruition—the "bravo stage" in which everyone, or nearly everyone, agrees, applauds and goes home and from then on nobody does much more about it, except in some rare cases, collects royalties.

I think you will agree that research in arteriosclerosis stands about half way between the first and second stages.

There are other adjuncts of modern methods of discovery, some of them often sneered at as mere trappings by our colleagues but most of them eagerly sought after once they have come into being. I refer to societies, committees and even to money, all the paraphernalia associated with that dreadful creature, the politician, and even worse, in the malignant form of the medical politician.

Again the field is bestirring itself because it now has the American Society for the Study of Arteriosclerosis to represent it and provide the rostra necessary to the communication of one's own work. The society has been, as many of you know, extremely successful. The meetings have steadily increased in quantity, but more importantly, in quality. Like so many young fields there is the usual tendency to scientific opportunism, i.e., dropping everything you are doing to get on board every new band wagon as it comes along. There is the usual re-doing by fancier methods of some of the older chestnuts. Pedestrian research is present but not in too depressing volume. Nonetheless, on the whole, it has seemed to me that sound ideas and good workmanship have prevailed and that the crackpots, exhibitionists, and opportunists seem to have been kept reasonably under wraps. The American Heart Association provides still another rallying point for those more concerned with the clinical effects of arteriosclerosis. This interest is especially focused in its Council on High Blood Pressure Research and Section on Circulation. As the section on basic science matures, it is more likely to take an interest in the mechanisms of arteriosclerosis. A time may come when it will be desirable for the American Heart Association and the American

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From the Research Division of the Cleveland Clinic Foundation and the Frank E. Bunts Educational Institute, Cleveland, Ohio.

Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

Society for the Study of Arteriosclerosis to fuse their interests. In my opinion it is not yet. But the American Heart Association does something more that is important; it collects and spends money for the support of research in arteriosclerosis. This I need not detail to you because you are all familiar with its extent and because many of you are recipients. Thus, a certain amount of power and responsibility accrue to this organization. I say this because of my "original" observation, made thirty years ago, of the positive chemotactic effect of the dollar on scientists.

Hand in hand, and I might say, hand in pocket with the American Heart Association, is the United States Public Health Service as specially represented by the National Heart Institute and the National Advisory Heart Council. Dr. James Watt, its able director, has told you of its activities. Having been intimately associated for a number of years with this organization and now having no ax to grind, I will say that no more honest, hard working and intelligent group have I seen. It is only a pity all government agents aren't like them, or are they? Perhaps more than our contacts with internal revenue would lead us to think. Oddly, the great pile of brick at Bethesda that houses their intramural program is productive due, I think, to the accumen of men like Dr. Sebrell, Dr. Van Slyke and Dr. Shannon, who chose their investigators well. I say "oddly" because most of us deplore the greater and greater size of so-called "research centers." The atmosphere of bigness itself often overwhelms and obliterates the creative one in which discovery is so apt to occur. The responsibility of these tax supported investigators is great and because they are tax supported they will have the added burden of minding their scientific manners, possibly more than most.

These institutions constitute the major agencies supporting research in arteriosclerosis. Other sources should be concerned more than I think they are. For example, the dairy industry may well find itself very much involved and perhaps like the tobacco industry, it will show signs of waking up to its responsibilities very late in the game. I would hesitate to think what this great industry would have to say about our chairman's views on the fat content of the diet. The insurance companies, involved to the extent of billions of dollars, have given only token assistance, much less than a million dollars annually for research in all cardiovascular diseases.

There are other aspects of the strategy and tactics of research in arteriosclerosis, equally, or more important than, the dollar. The chief of these is the creation of an environment which is attractive and intellectually satisfying for the basic sciences. If research in this field is forever dominated by clinicians, vigorous growth cannot be expected. It is to the chemists, biophysicists, physiologists, pharmacologists, pathologists and the like that we must look. To build the needed laboratory facilities is easy but to build the intellectual and social environment in a world dominated by physicians is far from it. And this is no reflection on physicians; it is simply that they have not yet learned how to give their support to research without keeping strings on it which allows them to control the work, if not in fact, at least by indirection. The scientist is not allowed his self-respect; he is made to feel he is permitted the privilege of work at the clinician's behest.

This may sound trivial to you but from long experience I can assure you it is not. The most virulent cases of Ph.D.itis occur in those associated with physicians. To me one of the really important aspects of the basic science section of the American Heart Association is that it was set up exclusively for the purposes of scientists. Unfortunately even it hasn't found the secret yet of luring and holding the chemist's and physicist's interest and affection. In twenty years I would hope that scientists of all variety will occupy important positions in all parts of the American Heart Association. If we are willing to spend millions of dollars on them, and this would presumably indicate our belief in them, then we should be willing to make them participants in the management and future planning of our endeavors. There are a host of problems such as long term support, overhead to institutions, fluid funds, annuities, public responsibilities, et cetera, which need their wisdom. Their participation will teach them in turn some of the realities of life, spread the burden of research management and help to create that necessary atmosphere of discovery of which I spoke.

It could be hoped that the planning of the attack on arteriosclerosis would be aided by cooperation of investigators with industry. This is in some cases true and is almost certain to lead to valuable results. Unfortunately another large group of companies are seizing upon the vast economic importance of the problem and our gross ignorance of both diagnosis and treatment, to sell all sorts of nostrums. These mixtures are purveyed not only as preventives for the "degenerative diseases of old age" but as out and out cures for arteriosclerosis. I think you are all too familiar with the "health and vigor" in the "second forty years" types of advertising.

Mixtures of androgens, estrogens, vitamins, lecithins, minerals, adrenal hormones, thyroid, ephedrine and liptropes purport to provide all the ingredients for not getting old and if taken from thirty-five to forty years of age are almost "guaranteed" to achieve both "subjective and objective" improvement. You are also familiar with the list of somewhat dubious references to the literature, either published in not well recognized journals, presented at meetings or not really pertinent to the argument. In short, the evidence to back the claims are shabby in the extreme. I have often asked the representatives of the drug houses if they would substitute the name cancer for the vague terms degenerative or circulatory disorders, etc., and see if the claims would be equally appealing. No, this is an abuse and those reputable companies who do this sort of thing should be made to understand that the peddling of these unproven mixtures adds nothing to their luster and takes many dollars needlessly from our patients. The same dollars put into sound research would yield dividends which no company would regret.

I can't let this opportunity go without a word about a new and vital problem facing the field of arteriosclerosis. It concerns suggestions, and indeed, recommendations, emanating from investigators in the field, for this or that change in the way of life of our citizens calculated to prevent arteriosclerosis. Good and wise public relations means the most careful screening and checking of all recommendations for widespread changes in public behavior. Are we in a position to recommend dietary changes yet? Are there laboratory tests yet available for the measurement of atherogenesis? Are any of the agents on the market purporting to be prophylactic or therapeutic of proved value?

And now the strategy and tactics of research in the problems of arteriosclerosis. I would be brash indeed, if I assayed to give you such. I can only give you a few of my opinions as an individual investigator. No one dares, I hope, hand down from the heights, even if he can get there, plans for the future of research.

As many of you know, I personally like to think of the problem as multifaceted. It is not alone one of lipids but of proteins and carbohydrates as well. It is not a question alone of lipoproteins in the blood but one of the reaction of the blood vessels to these lipoproteins as well. To me the problem is much like that of a public water works. Plasma with a wide variety of physicochemical properties is being pumped around in conduits which act as filter beds. The conduits are of widely varying properties so we have at least two large groups of variables. And then there are the pressure and flow relationships which seem important and highly variable. And finally there are a host of secondary factors such, for example, as peculiarities of anatomy which may at times become decisive in the effects of arteriosclerosis. The type of epidemiological approaches prosecuted by Keys and by Morris seem to me of great value for we must know of those environmental factors which aggravate the trouble. As many of you know, Paul White and I have seen eye to eye for many years in our desire to keep your arteries flexible by using the God-given form of locomotion. With all this in mind it is hard for me to accept the primacy of any especial approach. The only danger is that a single approach may exclude others with attendant failure to think creatively from a broader perspective. Too hasty codification of ideas ultimately leads to solidification. Ideas have a way of becoming "concrete" all too soon.

I think we must neither be surprised, nor too disturbed, when discoveries of the cause and cure of arteriosclerosis are announced reasonably regularly. But we must be eternally vigilant that privilege be preserved of freedom to speak out. With this privilege goes the responsibility of keeping emotional responses within reasonable bounds. In this we have been lax and as proof of it read your daily mail for the

(Continued on Page 808)

Pleomorphism of the Lesions of Arteriosclerosis

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HARDENING of the arteries, or rather arteriosclerosis, is a broad concept that in man encompasses pathological processes of the most varied sorts. Among the arteriosclerotic changes that may be observed, those lesions that contain an appreciable amount of lipid material in the arterial intima have been segregated from the remainder and called atherosclerotic. However, atherosclerosis itself is a very complex change that cannot be regarded as a simple pathological or disease entity. Even in the same individual the natural history of atherosclerosis from its commencement to its various complicated end states may involve many variable causal factors and developmental patterns.

Atherosclerosis is usually defined or assessed by the pathologist as a lesion. It is usually considered by the clinician to be a disease state that can be defined or assessed in the functional terms of its own complications or sequelae. It is, of course, appreciated that the sequelae may partially depend upon circumstances that are more or less independent of atherosclerosis *per se.* For example, the sequelae or complications of hemorrhage into atherosclerotic plaques, of thrombosis, of organization of thrombi and of infarction are only partially dependent upon the atherosclerotic lesion itself. Except in general terms, it is not possible to equate the lesions of atherosclerosis with their possible sequelae because the association between them is neither qualitatively nor quantitatively constant.

Ludwig Aschoff[1] has spoken of arteriosclerosis as an arterial dystrophy "which manifests itself by deposits of the most varied kinds in the vascular walls . . . " Some of these accumulate during the uncomplicated development of an atherosclerotic lesion while others may be super-added as a result of some local complication such as haemorrhage, necrosis or thrombosis at the site of a lesion. With the passage of time the material products of these complications may be incorporated in the lesion, become an integral part of it and contribute to the state of sclerosis.

I would like to illustrate for you something of the pleomorphism of the lesions that may be observed in arteries when a pathological or clinical diagnosis of simple or complicated atherosclerosis is made. Most of the material is from the major coronary arteries of man since these are of the greatest practical interest. The slides illustrate something of the natural history of the condition.

(*The colored slides that were shown with this paper could not be reproduced here. However, a brief description of each slide is presented to indicate the material that was covered.*)

1. *Coronary Artery.* Fat stain. To illustrate two large lipid-rich atheromatous lesions and marked stenosis of the lumen. In spite of the large content of lipid there is also a large amount of fibrous tissue present constituting at least one half of the bulk of the lesions.

2. *Coronary Artery.* Fat stain. A large crescentic atherosclerotic plaque narrows the lumen to about one half its normal size. The plaque contains a small amount of lipid at its junction with the media, but the majority of the lesion is fibrous and does not contain visible fat.

3. *Coronary Artery.* Fat stain. To illustrate a common association of a moderate amount of lipid, some macrophages and round cells and a moderate amount of fibrosis. Small vascular channels can be seen within the atherosclerotic intimal thickening. An uncommon feature of this slide is the presence of lipid-rich atheromatous material in the media of the artery beneath the center of the plaque.

4. *Coronary Artery.* Fat stain. To illustrate at larger magnification the lipid infiltration of the media referred to in the previous slide.

5. *Cerebral Artery.* Fat and elastic tissue stain. To illustrate lipid infiltration of the media of the cerebral arteries occurring through a gap in the internal elastic membrane. The fatty material has escaped from the

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intima through the elastic tissue defect into the media and to the adventitia. This type of lesion is common in cerebral arteries.

6. *Cerebral Artery.* Elastic tissue stain, paraffin section. To illustrate a lesion similar to that observed in the previous slide. The lesion has an hour-glass or button-hole shape. Lipid-filled macrophages or foam cells extend to the junction of media and adventitia.

7. *Coronary Artery.* Fat stain. Polarized light. To illustrate an accumulation of crystalline cholesterol material in an atherosclerotic plaque. This accumulation unlike most, contains only a very small amount of sudanophilic fatty material. It is surrounded by fibrous tissue.

8. *Crystals.* Polorized light. Crystals of cholesterol obtained by extraction with solvents from an atherosclerotic aorta. The crude material was purified slightly and recrystallized slowly. The notched square plate-like shape is characteristic. The unusual color effects are due to retardation of light.

9. *Surface Preparation of Aortic Endothelium.* Silver, sudan and hematoxylin stain. To illustrate an approximately normal endothelial pattern (female, aged seventeen years; congenital aneurysm of cerebral artery with rupture).

10. *Surface Preparation of Aortic Endothelium.* Silver, sudan and hematoxylin stain. To illustrate a bizarre endothelial pattern with the formation of multinucleated and giant endothelial cells. Such changes, like those of atherosclerosis, are associated with increasing age, but it is not established that the endothelial change and an atherosclerotic change are intimately related (male, aged forty-three).

11. *Surface Preparation of Aortic Intima.* Chlorazol black E stain. To illustrate some aspects of the proliferating reactive tissue in the atherosclerotic intima. The large bizarre stellate cells are probably smooth muscle cells. They lie among fibroblasts and numerous large and small round mononuclear cells.

12. *Surface Preparation of Aortic Intima.* Chlorazol black E stain. Polarized light. Another area of atherosclerotic intima composed largely of fibroblastic and collagenous elements with no muscle cells and few round cells. Lipid material is seen as bright, glistening droplets in large stellate cells. None of the more common globular lipid-filled macrophages or foam cells are present.

13. *Surface Preparation of Aortic Intima.* Silver, sudan and hematoxylin stain. Another area of aortic intima illustrates a focus where numerous large and small mononuclear round cells have collected under the endothelium in the intima. Only minute amounts of fat are present.

14. *Coronary Artery.* Hematoxylin eosin stain. This profoundly stenosed artery is from a 14-year-old female who suffered from nephrosis and chronic nephritis with hypercholesterolemia. The lesion shows a profound atrophy of the arterial media which is attenuated almost to complete disappearance. Fibrous tissue has proliferated to almost occlude the vessel. It contains moderate amounts of lipid but is not excessively fatty. Some lipid has penetrated to the adventitia.

15. *Coronary Artery.* Hematin-phloxin-saffron stain. The arterial intima contains two superimposed atherosclerotic lesions. The outer one is somewhat calcified. Both are heavily embedded in fibrous tissue. The lumen is stenotic. Adjacent lies a small extramyocardial branch of the coronary tree. It is almost obliterated by connective tissue proliferation.

16. *Coronary Artery.* Hematin-phloxin-saffron stain. To illustrate the complication of haemorrhage into a relatively non-fatty atherosclerotic lesion. Thrombosis has not occurred at this particular site. The products of the haemorrhage will contribute to the sclerosis at the site.

17. *Coronary Artery.* Hematin-phloxin-saffron stain. To illustrate the complication of intramural haemorrhage with disruption of the atherosclerotic plaque and thrombus formation.

18. *Coronary Artery.* Larger magnification of vessel shown in Slide 17. Haemorrhage has disrupted a pultaceous fatty plaque. Cholesterol crystal clefts are numerous. Thrombus material is seen where the plaque has broken into the lumen. There is some clotted blood attached to the thrombus.

19. *Coronary Artery.* Hematoxylin-eosin stain. To illustrate atherosclerosis complicated by thrombosis and organization of the thrombus. There is a small lumen surrounded by dense collagenous tissue. The majority of the occlusive lesion is composed of loose myxomatous connective tissue.

20. *Coronary Artery.* Hematin-phyloxin-saffron stain. Illustrating arteriosclerosis complicated by thrombosis. The arteriosclerotic intima shows extensive fibrotic, hyalin and calcific changes. Thombosis has occurred and a rather acellular "jelly" or hyalin material has replaced it. Large thin-walled revascularization channels have been formed.

21. *Coronary Artery.* Larger magnification of Slide 20. To illustrate the relatively acellular jelly-like material that has replaced the thrombus.

22. *Coronary Artery.* Hematin-phloxin-saffron stain. To illustrate atherosclerosis complicated by thrombosis. There is fibrosis, hyalinization and calcification in the intima. Thrombosis has occurred and extensive organization and recanalization has replaced the thrombus material.

23. *Coronary Artery.* Larger magnification of Slide 22. To illustrate the fibroblastic and collagenous nature

of the reorganized mass. There is extensive development of smooth muscle about many of the channels of revascularization.

24. *Coronary Artery.* Hematin-phloxin-saffron stain. To illustrate complete occlusion of an artery by a large amount of hyalin fibrous tissue and equally extensive deposits of calcium salts. Very little lipid is present. The vessel has been rendered extremely sclerotic.

The lesions you have seen are more or less common in the experience of the pathologist. They are very different one from another and manifest many vagaries of composition and content. They represent the lesions that one seeks to prevent or to cure. The features common to almost all of the lesions are the presence of mixed lipid material in the arterial intima, a cellular reaction manifest by the presence of large and small round cells—that is, macrophages, lymphocytes and indifferent mononuclear cells, and a proliferation of fibroblastic tissue. The proportions of these various elements to each other is rather unpredictable. Upon these common features may be superimposed a variety of degenerative changes such as necrosis; hyaline, fibrinoid or mucoid degeneration; and pathological calcification. Further changes may incorporate products of blood or thrombus material in the lesions. Cellular reactions may attempt to form new vascular channels that may develop abundant smooth muscle, connective tissue and endothelium within the original lumen of the artery. Varying forms and degrees of sclerosis of the vessel result.

The conclusions to be drawn from the lesions that you have seen are, in one sense, very simple. It is obvious that no simple etiological or pathogenetic concept will explain fully the pleomorphism of the lesions. According to our current understanding of the development of atherosclerosis, the most important single etiological factor is the abnormal focal accumulation of lipid material in the arterial intima. The lipid accumulation is followed by an irritative and proliferative cellular reaction in the intima and this in turn may be succeeded by the most varied degenerative changes in the lesions, by haemorrhage and by thrombosis.

From the prophylactic point of view one may seek to prevent or at least lessen not only the lipid deposition but also the subsequent proliferative and degenerative stages of the development of atherosclerotic lesions as well as the possible occurrence of intramural haemorrhage or thrombosis. Theoretically there is no compelling reason to maintain that an approach to the prevention of atherosclerotic disease must be limited solely to those aspects concerned with lipid accumulation, although this is obviously a valuable approach that lies close to the root of the matter.

From the point of view of the cure or removal of advanced or clinically important lesions the pleomorphism that you have seen precludes any simple metabolic approach. The composition of such lesions is so variable from person to person and from lesion to lesion that any metabolic procedure directed towards removal or reduction of one constituent, while possibly beneficial, cannot be expected to remove all of the other components; in any case, the nature of the major components of the lesions may not be known.

It is apparent from a pathological examination of a variety of simple and complicated atherosclerotic lesions that the prevention of the lesions is, in theory, a very different matter than their removal. Consideration of the prevention of a sequel such as thrombosis has still different theoretical bases.

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HEART-TY APPETITE

It has long been recognized that obesity produces detrimental effects on cardiac patients. Overweight heart patients have a much higher mortality rate. Additionally, "Obese people often have symptoms suggestive of heart disease, but without any demonstrable cardiac abnormality. . . . Obesity reduces work tolerance and cardiac reserve; it increases oxygen consumption, cardiac work, blood pressure and cardiac rate." Conversely, "Undernutrition lightens the work of the heart and has been of benefit to those with heart disease, . . ."—Master, A. M., and Jaffe, H. L.: Physiological Effects of Obesity Upon the Heart, *Journal of the American Geriatrics Society,* 3:299 (May) 1955.

Factors Other than Cholesterol in Arteriosclerosis

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WHEN one first begins to study arteriosclerosis experimentally, enthusiasm for one's own particular method of attacking the problem runs high. The horizon looks bright and the prospect of early success is encouraging, but before long even the most optimistic investigator becomes aware that the problem has many aspects he had not anticipated and he gradually realizes that perhaps "many roads lead to Rome." The program of this symposium, with papers on such diverse topics as metabolic disturbances, genetical effects, environmental factors and the baneful effects of our mundane way of life, clearly reflects this varigated aspect of the problem of arteriosclerosis. This catholicism of outlook and diversity of investigative approach to the problem seems to me most desirable for certainly we have not yet discovered many of the facts we must have in order to plan properly for the treatment and control of arteriosclerosis.

With these thoughts in mind I have chosen to tell you not about the discoveries made by traveling the main highway of the investigation of lipid metabolism, but rather to describe some of the discoveries made by exploring the byways of experimental arteriosclerosis. However, because of time limitations it will not be possible to consider all of these discoveries and so I shall limit myself to the following because it seems likely that they may play an important part in the pathogenesis of arteriosclerosis.

1. Ischemic Injury of the Arterial Wall
2. Metabolic Changes in the Arterial Wall
3. Thrombosis as a Cause of Arteriosclerosis
4. Vascularization and Hemorrhage of the Intima

Ischemic Injury of the Arterial Wall

For many years we have known that severe arteriosclerosis may follow injury of an artery and that often the sclerosis is sharply confined to the site of injury. Syphilitic aortitis provides a familiar example of this. Syphilis attacks chiefly the ascending part of the thoracic aorta and the arteriosclerosis which develops later also occurs in the same place—a location that is quite different from the usual site of aortic arteriosclerosis which is in the abdominal portion. Arteriosclerosis also occurs after other forms of primary arteritis such as rheumatic fever (Karsner and Bayless, 1934) and acute and chronic inflammations (Saphir and Gore, 1950). Experiments have also given confirmation of this view, showing that arterial cauterization (Ssolowjew, 1930, and Schlichter, 1946) and freezing (Kelley, Taylor and Hass, 1952) will cause localized arteriosclerosis and that rabbits fed cholesterol after arterial cauterization develop atheromas confined to the site of injury (Schlichter, Katz and Meyer, 1949; Kelley, Taylor and Hass, 1952; Ssolowjew, 1930). All these workers have independently suggested that the pathogenesis of this type of arteriosclerosis is ischemia of the arterial wall produced by injury of the vasa vasorum. Other evidence also supports the idea that local or diffuse ischemia of the arterial wall may cause arteriosclerosis. For example, in the neighborhood of infarcts of the kidney and myocardium, where the tissues are presumably poorly oxygenated, the intima of the arteries becomes greatly thickened (Rotter, 1949). Likewise, the extensive experiments of Hueper, in which he produced generalized arteriosclerosis in dogs by the administration of polyvinyl alcohol and other macromolecular substances, seem best explained on the basis of anoxia (Hueper, 1941a, 1941b).

Another mechanical factor which also ap-

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pears to have an important effect on the development of arteriosclerosis is high blood pressure. This concept is well supported by clinical (Dock, 1946), pathological (Baurle, 1951; Blumenthal, Handler and Blache, 1954, and Dock, 1946) and experimental evidence (Bronte-Stewart and Heptinstall, 1955; Ferguson and Varco, 1955).

Metabolic Changes in the Arterial Intima and Media

Until quite recently many students of arteriosclerosis have regarded the arterial wall as a passive participant in the development of the lesions and have placed emphasis on primary injury of the vessel by toxic substances in the blood stream. Now, however, evidence has accumulated which suggests that abnormal metabolic processes within the arterial wall itself may be of primary importance. For example, the results of experiments in which P^{32} was injected into cholesterol fed rabbits indicate a marked derangement of phospholipid metabolism in the aorta. Phospholipid was formed five times faster in the atheromatous than in the normal aorta and seemed to be synthesized in the wall of the vessel itself (Shore, Zilversmit and Ackerman, 1954). Other experiments, using P^{32} and acetic acid in which both carbons were labeled with C^{14}, indicate that the surviving aorta of rats can convert acetate to fatty acids and also readily incorporate P^{32} into the phospholipid molecule (Chernick, Srere and Chaikoff, 1949). Such experiments suggest that arterial tissue may actively metabolize lipids. However, we can not yet consider this suggestion as proved, for other experiments with Tritium labeled cholesterol indicate that exogenous cholesterol forms the bulk of the atheromatous deposits in cholesterol fed rabbits (Biggs and Kritchevsky, 1951).

Another interesting metabolic change occurring in the aorta concerns the metachromatic ground substance. This is a mucoid material which is thought to be an acid mucopolysaccharide and most likely chondroitin sulfate. It is probably a normal constituent of the aorta for it is present during the fetal period and at all ages thereafter in both humans and animals. Under abnormal conditions it may increase abundantly and form conspicuous deposits both in the media, where it lies closely applied to the elastic fibers and the elastic lamina, and in the intima, where it accumulates particularly in fibrous and hyalinized areas (Bunting and Bunting, 1953; Taylor, 1953).

Interest has centered around the arterial mucopolysaccharides for several reasons. In the first place, studies of early human coronary and aortic arteriosclerosis show that several pathological processes participate in its development and that they occur in an orderly sequence (Moon and Rinehart, 1952; Taylor, 1953). The first observable change consists of simultaneous proliferation of subendothelial fibroblasts, fragmentation of the internal elastic membrane and increase in mucoid ground substance. Lipid deposits form only after these changes have occurred and hyalinization and calcification appear only in the late stages. Indeed there is evidence to suggest that hyalinization of the tissues results from abnormal polymerization of the mucoid ground substance (Moon and Rinehart, 1952).

There is another aspect of the acid mucopolysaccharides that may have importance for the problem of arteriosclerosis. Accumulating evidence shows that they may be one of the tissue factors which determine the localization of cholesterol or other lipid deposits. For example, morphological observations and chemical determinations of the sulfate content of human aortas, which are a measure of the acid mucopolysaccharides, indicate that such a tissue factor probably determines the localization of the lipid deposits in hereditary xanthomatosis, syphilitic aortitis and the atherosclerosis of hypertension (Faber, 1949). In addition experiments have demonstrated that rabbits, which have been fed amounts of cholesterol insufficient to produce atheromatosis, will nevertheless accumulate cholesterol in the aorta following experimental procedures that produce reparative processes in which the acid mucopolysaccharides are increased. This has been shown in epinephrine sclerosis (Erb, 1905) and during repair of cauterized wounds of the aorta of rabbits (Ssolowjew, 1926, 1930).

Two important tools for the experimental study of the part played by the mucopolysaccharides in the pathogenesis of arteriosclerosis have recently been provided. Thus, it has been discovered that rats fed Lathyrus odoratus meal develop aortic aneurysms, and edema and hemorrhage of the media associated with increased chondroitin sulfate B and degeneration of the elastic fibers (Churchill, Gelfant, Lalich and Angevine, 1955). In addition, it has been found that arteriosclerosis develops in pyridoxine deficient monkeys (Rinehart and Greenberg, 1951).

Thus a considerable body of evidence indicates that an early change in human arteriosclerosis and possibly also in rabbit and fowl (Paterson, Slinger and Gartley, 1948) cholesterol atherosclerosis, has to do with an increased deposition in the media and intima of mucopolysaccharides. Other evidence also exists that the primary lesion of arteriosclerosis may occur in the media, but time does not permit my reviewing it (Gray, Handler, Blache, Zuckner and Blumenthal, 1953). In passing, I should like to comment that this aspect of arteriosclerosis has not been studied as sufficiently as the known facts indicate it should.

Thrombosis as a Cause of Arteriosclerosis

One of the most interesting new ideas about the cause of arteriosclerosis is the suggestion that thrombi, and in particular fibrin thrombi, instead of being exclusively the result of arteriosclerosis may be the cause of it. According to this concept small thrombi of fibrin or blood are deposited on the arterial intima, then organized and covered with endothelium and eventually incorporated into the arterial wall as focal, eccentric, thickened areas. Similar episodes repeated over the years eventually give rise to arteriosclerotic plaques. Rokitansky in 1852 first suggested such an explanation to account for the development of arteriosclerosis and his suggestion came to be known as the "encrustation theory." Virchow, however, repudiated this theory and as a result of his influence the encrustation theory was either forgotten or mentioned only briefly for the sake of completeness.

However, in 1946 Duguid brought forth morphological evidence which strongly suggested that under certain conditions organization of thrombi deposited on the walls of arteries did occur and that the morphological results of the organization were indistinguishable from arteriosclerosis. According to the thrombosis hypothesis when a mural or occlusive thrombus forms in an artery, it soon retracts in order to allow restoration of the circlation, and the new "channel becomes lined with endothelium and a new lumen is thus formed, with the thrombus now lying external to it and forming as it were a part of the vessel wall. Organization then follows, the subendothelial layer of fibrin on the free surface of the thrombus being transformed into fibrous tissue by what appears to be a direct metamorphosis, while around the attached surface of the thrombus a vascular zone of organization is formed which advances into the mass, converting it into fibrous tissue. Since this new tissue is similar to, and continuous with, that of the original intima, it has the appearance of an intimal overgrowth.

"In some instances a complete organization of the mass is brought about in this way; but more often, before the process has reached the center, fatty degeneration and sometimes softening take place, and thus the picture of atherosclerosis is produced. While these changes are in progress the new lumen under the influence of the blood pressure gradually widens, while the thrombus shrinks and becomes moulded to the shape of the vessel wall, so that in the end the mass which once filled the lumen is reduced to a crescentic thickening of the intima." (Duguid, 1949).

Other observers have verified Duguid's morphologic findings so that there is now little reason to doubt the accuracy of his observations or the correctness of his interpretations of their significance (Heard, 1949; Crawford and Levene, 1952; Geiringer, 1951, and Wagner, 1954). Other evidence also supports this view. For example, it has been found that in humans both large and small emboli to the lungs may undergo just such endotheliazation, organization and incorporation into the arterial wall (Krutzsch, 1920; Goedel, 1930; Ljungdahl, 1928; Belt, 1939a, b; Castleman and Bland, 1946; McKeown, 1952; Mautz and Craige, 1951; Saphir, 1932). Several authors have also described tumor emboli in the small pulmonary arteries being surrounded by hyaline thrombi that undergo organization and cause local intimal thickenings which resemble very closely pulmonary arteriosclerosis (Saphir, 1947; Krutzsch, 1920; Barnard, 1954a). In addition cases have been described in which repeated showers of fibrin emboli during pregnancy have lodged in the lungs and there undergone organization (Schneider, 1950, 1951).

This concept, that arteriosclerosis may result from organization of thrombi, is also susceptible to experimental testing and numerous experiments have shown that under certain conditions both thrombi and emboli may, indeed, become organized and produce significant arterial disease in exactly the way Duguid described (Harrison, 1948; Wartman, Jennings and Hudson, 1951; Wartman, Hudson and Jennings, 1951; Muirhead and Montgomery, 1951; Heard, 1952; McLetchie, 1952; Barnard, 1953, 1954).

The evidence, then, clearly shows that thrombosis must be considered as a possible factor of primary importance in causing arteriosclerosis. Indeed the thrombosis hypothesis explains some of the facts that other theories do not. In the first place, the organization of thrombi of fibrin and blood can actually be observed in many cases of human arteriosclerosis. In the second place, local lesions can be easily explained although the theory does not explain why certain locations are so consistently affected. In the third place, the theory accounts adequately for the narrowing which occurs so frequently in the human form of the disease. This fact is difficult to explain on the basis of primary injury of the vessel wall since destruction of tissue and replacement fibrosis in vessels subjected to expansile movement and considerable internal pressure should lead to dilatation rather than narrowing. No other theory of the pathogenesis of arteriosclerosis accounts for this feature so well—an observation which Duguid has repeatedly made. And finally it seems likely that many and perhaps most cases of pulmonary arteriosclerosis result from organization of multiple showers of small emboli or thrombi.

At first sight the thrombosis hypothesis seems to stand apart from other concepts and even to conflict with them. But recently other lines of investigation have begun to converge with it. Especially important from this point of view is the work on fibrinolysin activity during life and on fibrinolysin and the fluidity of blood post mortem (Biggs, Macfarlane and Pilling, 1947; Mole, 1948). It is obvious that if we accept as a working hypothesis the concept of the thrombotic origin of arteriosclerosis then we must admit the importance of factors controlling the formation of fibrin in the circulating blood. Macfarlane's investigations have shown that "fibrinolysis activation in the blood during life may be the initial result of a variety of stimuli having in common the exposure of body or mind to unusual stress.

"Mole points out that during life there is a continual production of fibrinogen by the liver, that this must involve a continual removal from the blood stream, and that normally some deposition of fibrin would be expected. Having investigated the relation between fibrinolysin and the fluidity of blood at necropsies, he considers that fibroinolysin is most probably produced by the vascular endothelium and that this may be the natural physiological means of preventing undue deposition of fibrin in health. Failure of fibrinolysis will promote the deposition of mural thrombi, which may form the initial lesion in atherosclerosis. Such failure, for example, occurs often in infection and cachexia, conditions which favor thrombosis." (Brit. Med. J. 1949).

The results of the experiments of Fullerton, Davie and Anastasopoulas (1953) not only confirm this view but extend it by showing a possible relationship to the lipid-cholesterol factor. They found that lipemia is accompanied by increased coagulability of blood and suggested that cholesterol and lipemia may produce arteriosclerosis by leading to intravascular fibrin formation and the initiation of the sequence of events suggested by Duguid. All this work needs further confirmation and the many interesting possibilities suggested by these concepts need to be put to the test of controlled experimentation.

Vascularization and Hemorrhage of the Arterial Intima

Finally I wish to discuss briefly the possibility that vascularization and hemorrhage of the arterial wall may be factors in the pathogenesis of arteriosclerosis. I believe it is fair to say that most students now accept the fact that the diseased arterial wall frequently becomes excessively vascularized with thin-walled, tortuous capillaries which are prone to rupture and bleed into the intima and media. Further, much evidence indicates that an expanding hematoma within the intima causes the obstruction in a small proportion of cases of coronary occlusion while in a larger proportion of cases intimal hemorrhage precipitates massive thrombosis (Wartman, 1950, for review of older literature; King, 1952; Yater et al, 1948; Paterson, 1952; Schlichter, 1948; Geiringer, 1951; Waters, 1948;

Lindbom, 1950; Papcharalampous and Zollinger, 1953).

All of these observations have been explained on the assumption that vascularization and hemorrhage are secondary phenomena occurring rather late in the development of arteriosclerosis. However, some observers have expressed a somewhat different point of view. They have suggested that disease of the vasa vasorum with subsequent abnormal vascularization and intramural bleeding may be events of primary rather than secondary importance. Winternitz (1938) points out that arteries are normally possessed of a capillary bed and thus are able, like other tissues, to respond to injury by the two fundamental processes of exudation and proliferation. He further observes that in the case of arterial injury exudation often predominates, that the exudate is often serofibrinous or hemorrhagic and that since it cannot always be easily removed it may accumulate in the vessel wall. Later these accumulations undergo fatty degeneration, necrosis and calcification and form atheromatous plaques. Increased serum lipids might hasten or exaggerate this process, but would not be essential to it. Many different etiologic agents might start such a process—vitamin deficiencies, a hemorrhagic diathesis, or a multitude of different microorganisms. In support of this concept Winternitz also draws attention to the fact that the development of the morphologic lesions of infective endocarditis bears a striking resemblance to the development of arteriosclerosis. A similar sequence of events is also known to occur in the thyroid where degenerating and organizing hemorrhages produce lesions indistinguishable, except for location, from arterial atheromas.

I believe I have given enough evidence to indicate the attractiveness of this concept of the pathogenesis of arteriosclerosis. Certainly it seems worthy of being used as a working hypothesis for other investigations.

Summary

In this paper, I have presented evidence which indicates that causes other than disturbed lipid metabolism may also be important in the initiation and development of arteriosclerosis. These causes include ischemic injury of the arterial wall, tissue factors such as the acid mucopolysaccharides, organization and incorporation into the intima of mural thrombi, and organization and vascularization of serous and hemorrhagic exudates in the intima. Evidence obtained from observations in humans and experiments in animals indicates that each of these possibilities could be used profitably as a working hypothesis for further study of the problem of arteriosclerosis.

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Atherosclerosis

The Atherosclerosis Problem

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ATHEROSCLEROSIS is the hub of the arteriosclerosis problem. Once it is conquered, a tremendous dent in morbidity and mortality rates will follow, and the average life span will be greatly lengthened. Are we making progress? I think we are. Evidence for this has been or will be presented during this conference.

Let us see wherein this progress lies. It lies first of all in the rejection of the view that atherosclerosis is merely aging which is inevitable and irreversible, and, more important, it lies in the acceptance, instead, that it is a disease which is reversible and, we are convinced, preventable. Basically it is a metabolic disorder involving lipid-cholesterol-lipoprotein metabolism. The theory upon which the work of the Michael Reese group* is now based considers that, when dealing with population groups, altered lipid-cholesterol-lipoprotein metabolism is engendered by a luxus of calories, lipid and cholesterol in the diet over the life span. This acquired rich and unbalanced diet, particularly in the sedentary, with its high fat content (constituting up to 50 per cent or more of the caloric intake) influences the metabolism of lipid-cholesterol-lipoprotein and so sets the background for the ready genesis of atherosclerosis in the population. Dietary deficiencies of some vitamins and amino acids may act to accelerate atherogenesis. Certain diseases, habits, stresses, genetic traits and local factors (such as vascular trauma and vascular infections) may act as triggers of this process in individual cases. Hypertension too is an accelerator of atherosclerosis. But basic to all of these triggers and accelerators, in most cases at least, is the dietary pattern over the life span, consisting of an excess of calories, lipid and cholesterol.** In the last few years the evidence in support of this theory has been summarized by my associates and myself (see Bibliography).

On this basis further studies must be directed along three principal lines, namely: (1) clinico-pathological, (2) epidemiological and (3) experimental. From the first two we obtain the background of atherosclerosis. The clinico-pathological and epidemiological studies pose the problems to be tested. In the experimental studies we put questions derived in this way and seek to obtain answers concerning etiology, pathogenesis, treatment and prophylaxis. After detailed analysis and adequate confirmation in the animal, we are prepared to verify our concepts of treatment and prophylaxis by cautious probes in man. This has been the modus operandi of the work of our group in the last two decades. It is my considered judgment that only by such a broad perspective can the problem of atherosclerosis be conquered. This approach plus care in designing the research program and care in interpreting its results will avoid fruitless — as contrasted with useful — bickering and "theorizing" ("idle speculation") which divert so much energy from the primary task of filling in gaps in our knowledge.

The problem of atherosclerosis brings together investigators from many disciplines and points up the trend toward narrow specialization which has crept into medical science recently. Appreciation of the views of persons from other disciplines working on atherosclerosis and understanding of the methodologies and "jargon" of the multiplicity of disciplines essential for understanding atherosclerosis will go as far as

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*The group consists at present of Drs. R. Pick, J. Stamler and myself and a number of dedicated technicians. In the past it included Drs. D. Dauber, L. Horlick and S. Rodbard.

**There is no doubt that atherosclerosis develops more readily in certain abnormal blood vessels than in normal ones, but we hold the view that it depends here too on the character of the lipid-cholesterol-lipoprotein metabolism.

any single ordinary discovery in accelerating our conquest of this disease. It definitely points up the need of overcoming the defects inherent in excessive specialization without loss of its benefits. Group research is one answer; a group which combines many talents can accomplish much, when supplemented by brilliant individualistic effort. Who amongst us knows which bit of knowledge, or, more likely, which combination from many disciplines will unlock the closets where the solutions of atherosclerosis are hidden? It follows, therefore, that we desperately need a meeting of minds, a return to broader perspectives in science, a counterbalance to the trend to specialization in research.

Clinico-pathological studies had a dominant role in atherosclerosis for many years. They helped define the correlation of this vascular disorder with the diseases affecting man. Morphological pathology has been useful in this regard when it recognized that its examination revealed only the anatomical character of this vascular process. From the anatomical examination one can only conjecture the dynamic biochemical, biophysical and physiological processes, which over the lifetime of the deceased led to the necropsy findings. The findings at autopsy must not be mistaken for the living process.

Clinical studies have been useful too in outlining the manifestations and sequelae of atherosclerosis, as well as the circumstances under which they become apparent. A moment's reflection will convince one that there is a vast domain of subclinical atherosclerosis that cannot now be definitely revealed by present methods of clinical study. Clinical manifestations and sequelae must not be confused with the disease itself, and procedures affecting the former must not be wrongly interpreted as necessarily reflecting effects upon the basic pathogenesis of the disease itself. The value of alleviating detrimental manifestations and of avoiding unwanted sequelae is obvious, but this does not necessarily mean getting to the bottom of the problem. Tests, even elaborate ones, and the factors altering the tests, do not necessarily reveal the pathogenesis, treatment or prophylaxis of the disease process itself. It is not from the manifestations nor from the sequelae that the answers will come, but only from an understanding of the total organism in its environment, on the one hand, and the biological process in living cells, on the other. And, by the way, it is necessary to remind oneself constantly, over and over again, that changes in the blood do not always reflect the changes occurring within the living cells nor the adaptations of the entire organism.

A great deal of information is obtainable from the study of man in his natural environment by methods which have proven so effective in the infectious and contagious diseases and which have been fruitful in the science of ecology as far as plants, and animals below man, are concerned. Man is of different ethnic origins, of two sexes, and of various ages. In the world today, and even in the U. S. A., man's environment is extremely variable as regards to climate, occupation, mores, diet and other stresses. This too is true of earlier periods of man's existence. Some of this data can be procured from available archeological and anthropological studies. More can be gotten, from vital statistics, especially in recent years, death certificates and census figures. Field studies need to be developed. A concerted attack in an expanding amount needs to be developed to pluck out the information buried in these rich sources of data. This will depend on correlations of differences obtained. By going from the study of the individual to that of large groups and by using the statistical approach based on probabilities, we may be able to derive as exact mathematical leads to the factors resulting in atherosclerosis, as is derived by the physicist and chemist when dealing with populations of molecules in different environments. Of course, epidemiology applied to atherosclerosis is a young and virgin field. But it is vigorous and already has made important contributions. The information available has pitfalls for the unwary. But with time, diligence and skill, these can be recognized and discounted. Concentration by properly trained scientists in this underdeveloped discipline should change the trickle to a veritable bonanza of new, needed, vital information from which questions can be put to the experimentalists to get answers from their animal studies.

The greatest impetus to the problem of atherosclerosis has come recently from the experimental approach in the animal laboratory. This is because of three fundamental advantages of this type of approach, namely:

1. The experiment can be designed to answer specific questions.

2. Controls can be run to exclude extraneous influences, and by using large enough samples and by random selection of "aliquots," statistically significant changes can be distinguished from sampling errors.

3. The lesions of atherosclerosis themselves can be studied in all its stages rather than some secondary manifestation or sequelae, or a presumed associated index.

It goes without saying that experiments must be properly conceived and executed. A mass of information can come from properly conducted animal experiments concerning: (1) the role of dietary and other ingested materials, (2) the effect of various microorganisms resident in the gastrointestinal tract, (3) the influence of hormones and other humoral and neurogenic regulators of bodily function, (4) the intermediary metabolism of lipid, cholesterol and phospholipids, their kinetics, energetics and enzymology, (5) the biology and metabolism of the blood vessels, (6) the transport of fatty substances in the blood and tissues, et cetera. Much information has already been gained but many gaps remain and new problems have arisen as old ones were solved. Thus, the revelation of differences in biological response of different arterial beds—as for example between the coronary arteries and the aorta—has posed the problem of the cause of this difference.

Many findings in man, especially as to sex, have been confirmed and analyzed in animals and new insight into its mechanism obtained. The role of age and species differences has received growing attention. Some therapeutic and prophylactic potentialities have been revealed so that some investigators may feel that animal experimentation has all the answers to atherosclerosis. Far from it. The mere fact that species differences do exist demand that all animal observations of significance must be reexamined in the species we are interested in, man. Otherwise ghosts may be created which fade in the piercing light of clinical experience.

Obviously, crystallization of the previous information concerning atherosclerosis will come only by the proper free interplay and adequate fusion of the three major approaches to the problem, epidemiological, clinical and experimental. We are getting to our goal—but we have not reached it yet. We have a motif, the life span unbalanced luxus diet, rich in calories, fats and cholesterol. We must look into other aspects, as for example: (1) the causes for localization of lesions in particular sites, (2) the causes for intimal ulceration, (3) the influence of hemorrhage and thrombosis, (4) the influence of physical and physical stresses. These are, as it were, decorations to beautify the motif, they must not be mistaken for the motif itself.

We have come a long way along the road to the solution of the problem of atherosclerosis in the last two decades. The goal is dimly glimpsed through the haze ahead. But we are not there yet, considerable labor on the part of many, carried out in a truly scientific atmosphere still lies ahead. But the reward will more than repay the effort still to be employed.

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If one were to use as criteria the amount of life spoiled by disease, instead of measuring only that destroyed by death; or the number of days lost from pleasure and work because of so-called minor ailments; or merely the sums paid for drugs, hospitals, and doctors' bills, the toll exacted by microbial pathogens would seem very large indeed. Microbial diseases have not been conquered. Rather, scientists have resigned themselves to the belief that a relative protection against them can be had only at the cost of a huge ransom.—RENE J. DUBOS, Ph.D., *Journal of the American Medical Association*, April 23, 1955.

Mode of Life and the Prevalence of Coronary Heart Disease

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IT IS generally agreed that neither infection nor external physical trauma can be blamed for any appreciable primary role in the development of the great majority of cases of atherosclerosis and coronary heart disease. Moreover, we have fortunately abandoned the view that aging, *per se*, is responsible. Finally, there is abundant proof that individuals and whole populations differ greatly in this tendency to develop the basic, primary lesions and the resulting consequences.

Accordingly, the responsible factor or factors must be sought for either in heredity or in the mode of life, or in both. I shall discuss heredity shortly and may persuade you that even though heredity influences the outlook for some people, most of us are *not* doomed simply because of the genes we have inherited. But we still must ask, "What kind of people develop coronary heart disease?"

The popular picture of the coronary heart disease victim is that of a burly business or professional man, fat and soft from overeating and lack of exercise, who smokes and drinks too much because he is tense with the stresses of modern life in his effort to get to the top. Obviously he is a white, urban American and very likely in his family tree there are forebears who died suddenly.

Perhaps there is something in this description but as a totality it is a caricature. Such men do exist and some of them die of coronary heart disease. But many men of this type escape and the great majority of coronary victims are not so readily pigeonholed as to type. The diversity is apparent from a survey of patients in a few general hospitals.

In a recent spot check of all patients in the medical sections of five hospitals in the Twin Cities we found eighty-two patients with coronary heart disease. Among these less than a third were or had been obese, 42 per cent were women, and among the men many were non-smokers and only a minority were in the business or professional class. Obviously, the problem is complicated.

There is a host of reasons to insist that the mode of life must be important and, I think, dominant. But what factors in the mode of life can be singled out for suspicion or for outright blame?

Besides heredity *per se*, let us list the characteristics of people that may deserve consideration. These include the habits of diet (both quantity and character), of physical activity, of emotion and of drinking, of smoking and of sex indulgence, all of which, theoretically, could be changed if need be. To these may be added sex itself and the body build, meaning the skeletal type, neither of which can be considered an expression of the mode of life though both merit a comment in passing.

Sex

In populations with much coronary heart disease, as in the United States, the mortality from this cause at relatively young ages is much higher in men than in women, and we say that females are protected because of their sex hormones. Actually, females are by no means immune. In 1952, 4,862 American women under fifty are recorded as dying of coronary heart disease and "myocardial degeneration." Nor do they rapidly lose their relative protection as soon as they reach the menopause. The female death rate from coronary heart disease does not equal that of the male until after the age of eighty years.

All of this is in the U.S. in populations where coronary heart disease is less common than here,

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TABLE I.

Mortality ascribed to arteriosclerotic heart disease in Italy (1950) and among Whites in U.S. (1952). Rates per 100,000, by sex.

	50-54 years			55-59 years		
	M	F	M/F	M	F	M/F
U.S.	544.1	183.8	2.96	877.9	331.0	2.65
Italy	117.1	51.6	2.27	187.3	107.9	1.74
U.S./Italy	4.65	3.56	—	4.69	3.07	—

the sex ratio of coronary heart disease is also less than here, as indicated in Table I.

These are vital statistics. The general accuracy of the picture they present has been checked by several methods—consultation with many physicians, surveys of many hospitals, coroners' reports, the experience of insurance companies in the two countries and by detailed analysis of the statistics.

There are only two possibilities to explain the facts. Either Americans tend to have a remarkably bad heredity in this respect or there is something in our mode of life that promotes the deposition of cholesterol and the formation of thrombi in our coronary arteries. And this detrimental influence is prominent among our men but our women are also affected.

Body Build or Type

It has been reported that overweight is not important but that men of stocky or heavy muscular, "mesomorphic" build tend to have coronary heart disease at an early age (Garn et al, 1951; Gertler, White, et al, 1954). If this is so, some of the trait may be hereditary, but we may ask whether the mode of life in childhood—the diet and habits of exercise, for example—influences the eventual skeletal type. And we can still insist that body build cannot be a dominant factor because there are plenty of mesomorphs in populations, like the Italians and Bantu, who suffer far less coronary heart disease than we do. In other words, the body build may be partly an expression of the mode of life and in any case it is certainly not fatal in itself.

Race and Heredity

It is easy to fall back on "race" and "heredity" as explanations for the obvious differences between individuals and between populations in their susceptibility to arteriosclerotic heart disease. Several excellent studies (e.g., Boissevain, 1931; Boas et al, 1948; Wilkinson, 1950; Adlersberg et al, 1950), have painstakingly traced many members of a number of unfortunate kindreds in which coronary heart disease is prominent, cutaneous xanthomata are not rare and high blood cholesterol values tend to be the rule. Citation of these cases as indicating a genetic basis for our number one cause of death is gratifying to the professional ego of the geneticist and absolves the medical researcher from any pressure to find out why people and populations differ. A few generations ago the attempt to learn how to prevent tuberculosis could have been opposed on equally meretricious facts and argument.

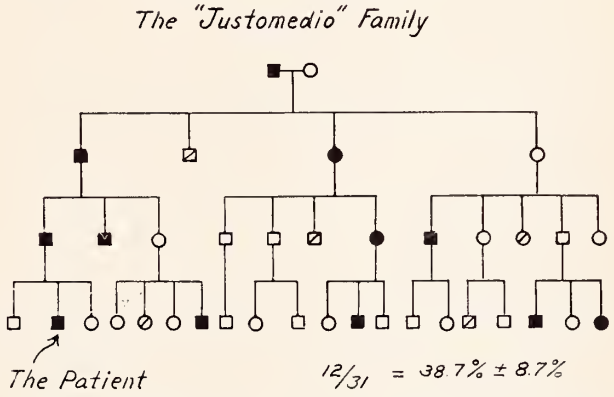

Fig. 1. Family tree of the "Justomedio" family. Squares represent males, circles females. Members dead of coronary heart disease are indicated in solid black. The symbols with a diagonal line indicate persons who died in youth.

It should be noted that the discovery of any family which apparently is coronary-prone is always good for at least a paper or two in the medical literature. This fact is eloquent testimony to the rarity of cases where peculiar heredity seems to be the basic cause of the trouble. Moreover, the freqency of coronary heart disease in our general population is so high that one may question how much of the coronary heart disease in so-called coronary families actually represents a great excess beyond ordinary expectations.

Consider the family tree, covering four generations, given in Figure 1. The physician called to attend a man in his forties dying of coronary heart disease is impressed to discover that the patient's father and an uncle died of coronary heart disease. When the family is traced further it appears that the patient's grandfather was found dead in bed one morning and that his great grandfather also died suddenly. The upshot is that among thirty-one descendants of the suspect great grandfather, excluding five who died in youth, there is good reason to label twelve dead of coronary heart disease, or a possible incidence of 38.7 per cent.

This is impressive indeed. Or is it? We find that the standard error of this incidence value is ±8.7 per cent and that for the U.S. white population as a whole the vital statistics show that nearly 30 per cent of all deaths beyond youth are ascribable to coronary heart disease.

The family illustrated in Figure 1, in fact, does not deviate significantly from our national picture. The "Justomedio" family is fictitious. This chart was devised to illustrate our *national* plight and not that of a particularly coronary-prone family.

Now these critical remarks are not to be taken as a denial that heredity operates in coronary heart disease. Unquestionably a serious abnormality in cholesterol metabolism or of vascular architecture leading to coronary heart disease *can* be inherited. But I am unaware of any evidence to show that this is anything more than a rare occurrence. It recalls the *one* case of fatal coronary occlusion found in a large series of autopsies on Bantu natives in Johannesburg. The victim was a twenty-three-year old girl! Very probably she suffered from a "metabolic fault" and it might even have been hereditary.

A stronger case can be made for the existence of familial tendencies, as opposed to strict inheritance, towards more or less coronary heart disease. But for these it is enough to cite the influence of a common mode of life imposed by family custom in food, activity, and so on.

Objection may be made that we still must credit racial genetic inheritance as a *possible* explanation of the difference in coronary proneness between people in the United States and those in Japan or the Bantu or even in Italy. In opposition are the following points.

1. Such information as we have indicates that American Negroes, unlike the Bantu, are by no means immune. Dr. Jeremiah Stamler has recently obtained evidence, confirmatory of other findings, indicating that Negro men in Chicago have an age specific mortality from arteriosclerotic heart disease at least as high as white men in Chicago. If it is argued that the American Negro has much other negroid blood besides Bantu, it must be pointed out that the general picture of the absence of coronary heart disease among the Bantu seems to prevail among all of the negroid people of Africa who furnished the slaves brought to the United States.

2. Larsen (1955) finds that the incidence of severe atherosclerosis and coronary heart disease among the semi-Americanized Japanese in Hawaii is lower than that of white Hawaiians but it is much higher than found in Japan.

3. Physicians anywhere in the United States who have many Americans of Italian descent among their patients will testify that coronary heart disease is far from a rarity among these people. In selected samples of Americanized Italians and Jews in New York, Epstein and Boas (1955) found no difference in the incidence of atherosclerosis among the men but evidence for coronary heart disease was more frequently seen among the Jewish men, but not among the Jewish women, than among the Italo-Americans. The Jews and Italo-Americans did not differ in the incidence of either hypertension or diabetes.

4. Within Italy there are great differences in the prevalence of coronary heart disease which are readily explicable, not by race, but by the customary local diets.

5. The experiences of the World War and after have shown that among Nordic and Teutonic peoples, too, coronary heart disease can decline greatly in spite of their race if they change their manner of life, particularly their diets.

To these points may be added other bits of information that may be relevant. In studies on population samples we have repeatedly found that at equal age, both the serum cholesterol concentration and the mortality rate from coronary heart disease are much higher in the United States, England and Sweden than in various parts of Italy and Spain. We believe that dietary differences are adequate to account for this, but the question may be asked whether it is possible that Nordics or Anglo-Saxon-Teutonic peoples have a genetic tendency to higher cholesterol than the Latin or Mediterranean peoples.

Firstly, it may be replied that within the Spanish and Italian populations, at least, there are differences in serum cholesterol and coro-

TABLE II.

Serum total cholesterol concentration, in mg. per 100 ml., in clinically healthy, middle-aged businessmen in Minnesota, classed as to eye color and matched as to relative obesity and age.

Eye Color	No. of Men	Serum Cholesterol	
		Mean	S.E.
Blue	70	251.8	±5.2
Brown	48	257.8	±5.4

nary heart disease prevalence that are explicable by dietary but not by racial differences (cf. Keys, 1952, 1953, 1954; Keys et al, 1954, 1955).

Secondly, we have attempted to approach a classification of Minnesota men into the more "Nordic" and the more "Mediterranean" types by eye color, men with clear blue eyes being considered to be more Nordic than those with deep brown eyes. Table II summarizes the serum cholesterol values in these two groups, matched in age and in relative obesity. The *brown* eyed men tend to have higher serum cholesterol values but the difference between them and the blue eyed men is not statistically significant.

Far more research is needed to show how much or how little of our tremendous incidence of coronary heart disease depends upon heredity. A few people are working on the question but all signs point to heredity as being only a minor factor and, moreover, one that can be overcome. I insist as forcibly as I can that our research effort must be geared to the proposition that heredity will not prevent success in the prevention of coronary heart disease.

Overweight and Obesity

Following the lead of life insurance companies, it has been widely believed in this country that a high frequency of overweight and obesity in the American population explains to a considerable degree the high frequency of coronary heart disease in the United States.

Elsewhere I have pointed out some of the most serious questions about the insurance company data used to support this thesis (Keys, 1954, 1955). While there will be little dissent from the opinion that extreme obesity is a health hazard in general, and perhaps for heart disease in particular, the evidence from various sources is far from convincing about coronary heart disease. Among other things, we may ask whether insurance applicants are a truly representative sample of overweight persons. In any case only a small fraction of our total burden of coronary heart disease could possibly be ascribed simply to overweight or obesity.

TABLE III.

Frequency of overweight among clinically healthy men 20-60 years old. Values listed are percentages of the men in the samples studied who were 10 per cent or more and 20 per cent or more heavier than the average for height and age given in the U.S. Medico-Actuarial tables. "Police-Fire" are men in the city fire and police departments.

Occupation	Region	Overweight By	
		10%	20%
Sedentary Work	Naples, Italy	28.1	10.3
Sedentary Work	Bologna, Italy	40.0	21.8
Sedentary Work	Malmö, Sweden	13.9	3.7
Sedentary Work	Twin Cities, Minnesota	22.5	5.3
Police-Fire	Naples, Italy	29.4	9.4
Police-Fire	Bologna, Italy	32.1	19.5
Police-Fire	Malmö, Sweden	21.4	10.0
Police-Fire	Twin Cities, Minnesota	42.0	18.0
Heavy Work	Naples, Italy	16.2	6.3
Heavy Work	Malmö, Sweden	14.0	4.3

At most it might be claimed that grossly overweight men in the U.S. have a mortality rate from coronary heart disease at relatively early age some 50 per cent higher than the general population of men who are not so obese. The question then turns on the proportion of the population that is grossly overweight. The insurance companies have selected 20 per cent over the "standard" average body weight for given height and age as the point beyond which they attach a penalty. One estimate, from the Metropolitan Life Insurance Company, is that perhaps 7 per cent of American men are in this category though the proportion among insurance applicants is smaller than this. It is easy to calculate then, that over 95 per cent of the coronary heart disease mortality among American men occurs in persons who are not obese enough to be specially "rated" by the insurance industry.

Moreover, in all studies so far reported on actual coronary patients the distribution of relative body weight among these patients proves to be similar to that of the rest of the population of the same age who do *not* have coronary heart disease (cf. Keys, 1954, 1955).

Finally, let us ask whether the lower incidence of coronary heart disease in some other populations can be explained by the absence of overweight persons in those populations. Actually, "overweight" occurs in all countries and even among such special groups as Bantu in South Africa overweight is not rare. But for the present

TABLE IV.

The distinction between obesity and relative overweight as illustrated by four men, all of the same height and age.

Body Weight lbs.	% of Weight in		Obese	Overweight
	Fat	Muscle		
160	20	50	No	No
160	30	40	Yes	No
180	20	50	No	Yes
180	30	40	Yes	Yes

purpose some of the most useful data are from recent surveys in Italy and in Sweden as summarized in Table III. "Overweight" was calculated from the ordinary U.S. Medico-Actuarial Tables of weight for height and age.

It is obvious that Americans have no monopoly on overweight. Among sedentary men the Italians sampled in Naples and in Bologna exceed Minnesotans as well as southern Swedes in this regard. Many members of the Police and Fire Departments in all of the cities are overweight, but Minneapolis and Bologna are neck-and-neck for overweight top honors. Finally, it will be noted that the heavy workers of Naples tend to be overweight more often than their counterparts in Malmö.

Dr. Brock may choose to say something about overweight among the Bantu and Cape Coloured men in South Africa. But it may be permissible here for me to cite some figures Dr. Bronte Stewart and I obtained last spring when we were working in Dr. Brock's laboratory. Among the first 90 Cape Coloured men we studied, 16 were 10 per cent or more overweight and 6 were more than 20 per cent overweight. Among the first 119 Bantu we studied, 13 were 10 per cent or more overweight and 6 were more than 20 per cent overweight. One of these Bantu was 5 feet 8 inches tall and weighed over 260 pounds.

These data certainly do not suggest that overweight is a primary factor in the development of coronary heart disease. For there is no doubt that the relative prevalence of coronary heart disease is very different in these populations, with Minneapolis at the high end and the Bantu at the bottom. Bologna is above Naples but far below Malmö in the prevalence of coronary heart disease. And I suppose that the frequency of coronary heart disease among the Cape Coloured men is somewhere between the values for Naples and Bologna.

Nothing of what I have said should be taken to mean that overweight is of no importance or that we should stop condemning obesity. Gross obesity, like any other marked departure from the central tendency of mankind, is undesirable and may be unhealthy too. But in regard to coronary heart disease, it seems impossible to assign to it more than some aggravating or accelerating influence to a process, that is to atherosclerosis, that is primarily conditioned by other factors.

It may even be that obesity, *per se,* has no influence but that in some populations men who are obese also tend to have other characteristics that lead to trouble. They may, for example, indulge in high fat diets as well as merely eating a lot. And, if we credit lack of exercise as having a real influence, it is significant to note that most fat men tend to move slowly and infrequently. In any case there is no reason to believe at present that the American problem of coronary heart disease will be solved, or even reduced much, by "curing" our obesity.

One final word on this subject, now that I have seemingly used the terms obesity and overweight interchangeably. Actually, obesity means fatness and it is only at the extremes that fatness and overweight tend to be closely related. We may ask our insurance friends how they would predict the prospects for coronary heart disease for the four men in Table IV.

These men are all of the same age and height. Their fatness is indicated as the percentage of their body weight made up of fat. Two men are obese but only one of these is overweight. Two men are overweight but only one of these is obese. The point is that neither insurance nor any other data on coronary heart disease have made this distinction, and all of the data that purport to show excessive mortality among fat people actually refer only to relative body weight.

Smoking

The reports by Hammond and Horns (1954) and by Doll and Hill (1954) on the mortality experience of men of known smoking habits have greatly increased interest in the theory that smoking may produce or promote coronary heart disease. In spite of the absence of concrete evidence, or a plausible theory, of mechanism for a true cause-and-effect relationship, it is not easy to disregard the finding that the death rate ascribed to coronary heart disease is

50 to 100 per cent greater among the smokers than among the non-smokers in the large population samples studied.

Berkson (1955) has severely criticized statistical shortcomings in these studies, arguing that the reported association between coronary heart disease and the habit of smoking could be spurious. Extreme assumptions about bias in the selection of the participants in the studies are required to defend this ultra-skepticism. In any case, it is useful, temporarily, to accept the facts as they appear and to proceed further to inquire what they may mean. The findings as they stand would be consistent with any one of three hypotheses:

1. Smoking, directly or indirectly, causes coronary heart disease, which means that smoking *per se* promotes atherosclerosis and/or thrombosis in the coronary arteries.
2. Smoking is a secondary aggravating factor in the presence of primary forces tending to produce atherosclerosis and/or thrombosis.
3. Smoking *per se* has no influence but the smoking habit is favored by men who, because of heredity and/or mode of life in other respects, are unusually prone to coronary heart disease.

I believe there is already evidence at hand to disprove the hypothesis that smoking has an important primary effect in the development of coronary heart disease. At least we can show that great differences in the prevalence and mortality of coronary heart disease in different populations are quite independent from any differences in the prevalence of smoking.

Over a great part of the world the majority of the men smoke cigarettes. In poorer countries fewer cigarettes are smoked but they are more completely smoked. In terms of nicotine and irritant received, the net effect of smoking ten cigarettes to the last millimeter may be greater than smoking twenty cigarettes in the manner common in the United States.

In Italy we have found by personal surveys that 40 to over 70 per cent of the men are smokers, as shown in Table V. From one-fourth to over half of all men studied were found to smoke from ten to fifty cigarettes daily and the habit had been established in youth. Even among Sardinian shepherds and coal miners, whose occupations and economic circumstances tend to discourage smoking, at least 20 per cent of the men were heavy smokers.

TABLE V.

Percentage of men under 60 years of age in various populations who smoke more than 10 cigarettes daily or more than 0.5 pound of pipe tobacco per month.

Twin Cities, Minnesota, Business Men	52%
Bologna, Italy, Employees	68
Cagliari, Sardinia, Employees	73
Sardinia, Coal Miners	63
Sardinia, Shepherds	43
Nicotera, Italy	63
Cape Town, Europeans	72
Cape Town, Coloured	81
Cape Town, Bantu*	72
U.S.A. Surveys, ages 50-59	53

*Mostly pipe smokers

Obviously, in regard to smoking, there is no great difference between men in the United States and men in Italy, or between men in southern Italy and those in Bologna. But, as has been noted, these areas differ greatly in the prevalence of coronary heart disease.

Even greater differences in coronary disease prevalence are found within populations in Cape Province, South Africa, but again, as shown in the table, there is no corresponding difference in the custom of smoking. The Bantu, at the bottom of a steep economic ladder, can rarely afford to be a heavy cigarette smoker, but over half of them in our sample smoke, in pipes, at least half a pound of tobacco a month. And in these men coronary heart disease is extremely rare. The Cape Coloured man, whose tendency towards coronary heart disease is certainly greater than the Bantu but is far less than that of the local European, is similar to the local European in his smoking habit. More often than not, the Cape Coloured man is a heavy smoker of cigarettes.

In Japan, too, heavy cigarette smoking is common but the data of Kimura (1955) confirm the long-standing opinion that coronary heart disease and severe coronary atherosclerosis are very uncommon among Japanese men in Japan.

These data do not, of course, answer all of the questions about the effects of smoking. It is possible to suggest that if the Bantu or Japanese or the southern Italian did not smoke they might be even freer of coronary heart disease. But it is not possible to deny that some factor other than smoking must be the dominant reason for the high incidence of coronary heart disease in the United States and other high coronary countries.

A trial calculation is informative. Suppose we accept the most optimistic interpretation of the Hammond and Horns data and assume that if our American men stopped smoking they would experience only the coronary mortality observed among the non-smokers in the Cancer Society study. In other words, for roughly half of our men in the fifties the mortality from coronary heart disease would be reduced by about 40 per cent. The total coronary mortality rate for all of the men in the fifties would be reduced, then, by some 20 per cent and this is certainly not negligible. But to equal the Italians' experience, *including* the 50 per cent or more of them who are smokers, we should have to effect three to four times this reduction!

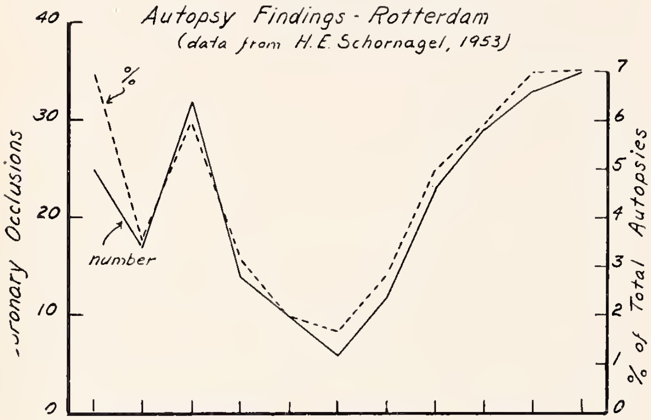

Fig. 2. Number of coronary occlusions, and percentage of total autopsies represented by them, in the Municipal Hospital records at Rotterdam, the Netherlands, for the years 1940 to 1950. Data from Schornagel, 1953.

There is as yet no evidence at all that stopping smoking would have *any* effect on our current or future burden of coronary heart disease. But, as indicated above, the best result we could hope for would still leave us with a greatly excessive mortality.

It has been suggested that the great decrease in smoking in some European countries during the war might have been influential in producing the sharp declines in coronary heart disease seen in those countries at the time. But, as pointed out by Schornagel (1953) for the Netherlands, this theory cannot be defended by a critical analysis of the data. In the Netherlands the wartime decline in coronary heart disease, and the post-war rise, affected both men and women equally but only a negligible proportion of the women in the Netherlands smoked before, during or after the war. Further, in Denmark the wartime decrease in smoking was the same as in other parts of Northern Europe but there was no decline in coronary heart disease in that country during the war. But it should be noted that the Danish *diet* was not changed during the war.

Alcohol

Relative rarity of severe atherosclerosis and coronary heart disease among alcoholics has been commented on occasionally in the literature. In Japan, Kimura (1955) observed marked atherosclerosis at autopsy more frequently among men who habitually drank *and* smoked cigarettes than among men of the same age who indulged in neither. Among elderly men in Britain, the average serum cholesterol concentration was considerably lower among the heavy drinkers than among abstainers and moderate drinkers (Hobson et al, 1953). Finally, it has come to be a common custom of competent cardiologists, presumably justified by clinical experience, to permit, or even to advise, their coronary patients to use alcohol but, of course, in moderation.

The evidence on alcohol is not great but it seems safe to conclude that the use of alcohol does not promote either atherosclerosis or coronary heart disease.

Physical Activity

It is widely believed, and some impressive evidence can be cited in support, that men whose occupations involve physical work are less prone to coronary heart disease than are sedentar men. But the best evidence is from countries such as England and Sweden where the distinction between manual and physical work is greate than in the United States and where other differences in the mode of life are more obviousl associated (Hedley, 1939; Morris and Heady 1953; Morris *et al,* 1953; Biörck *et al,* 1954).

In the United States these distinctions between manual and mental labor are disappearing Our mental workers are not apparently doin more manual work but our manual workers ar required to exert less and less physical effor to do their jobs. Perhaps this is why it is mor difficult in this country to demonstrate the majo differences between classes of workers in regard to the incidence of coronary heart disease Or is it because we are also more homogeneou in regard to income, diet and general mode o life?

For instead of physical activity as the criterion, in many countries a social or economic classification would show differences in the prevalence of coronary heart disease at least as great as can be demonstrated when men are classed according to physical activity. Malhotra's (1951) data from India, summarized in Table VI, are typical.

Morris (1955), has recognized the serious analytical problem resulting from the fact of high intercorrelation between a number of factors in the mode of life that may singly or together promote atherogenesis and coronary heart disease. My own belief is that physical activity is a real factor, but far more research is needed to show how great, if any, may be its effect.

Several items of information suggest that physical activity is not, by itself, a dominant factor. First, Kimura (1955) at Kyushu Medi-al School in Japan, where 100 per cent autopsy s the rule, found no significant difference be-ween manual workers and mental workers in the ncidence of severe atherosclerosis. Second, either in Naples nor in southern Sweden were here significant differences between the serum holesterol levels in men of equal age habitually ngaged in different levels of physical activity, hough there were major differences between the wo countries at the same level of physical activ-ty (Malmros and Biörck, 1954). Third, it is dif-icult to suggest that Englishmen and Swedes are ery much less active than Italians or Spaniards nd therefore the differences in the prevalence of oronary disease may be explained. Finally, in apan, where coronary heart disease is so rare, here are hordes of men who do very little phys-cal work. Clerks, petty officials, sedentary tradesnen, and relativelv sedentary artisans form a ery large group in Japan.

Emotion—"Stress and Strain"

Hours could easily be spent arguing about the uestion as to the role, if any, of emotion and he "stress and strain" of modern life on the roduction of coronary heart disease. One is empted to quote the familiar verse:

> "Myself, when young did eagerly frequent,
> Doctor and Saint and heard great argument
> About it an about, but evermore came out
> By the same door as in I went."

But opinions are strongly held and stated in pite of the complete absence of objective evidence. Melville Arnott (1954) has well stated the case, from what is known at present, against blaming stress and strain. This should not deter us from urging that this shadowy area be subjected to systematic critical inquiry. Until then, however, there is really almost nothing to say beyond noting that life in Japan scarcely seems free from stress and strain and that even the Bantu has emotional problems.

TABLE VI.

The socio-economic factor in the distribution of kinds of heart disease as illustrated by data for Amritsar, India, on the percentages of coronary heart disease patients and of patients with rheumatic heart disease contributed by the three major social classes. Data from Malhotra (1951).

Class	Coronary	Rheumatic
I	31%	7%
II	54%	49%
III	15%	44%
All	100%	100%

Diet

So far this discussion has led mainly to negative conclusions. No major, primary role in atherogenesis or in the production of clinical coronary heart disease can be assigned to race, nationality, obesity and overweight, cigarette smoking or indulgence in alcohol. Some of these factors may make a secondary contribution, perhaps contingent upon the existence of a particular set of circumstances.

The relegation of physical activity to a minor place is made with more reservation but we still have little that is positive to go on. Finally, even sex may have its limitations.

But when we come to the diet, apart from sheer calories, there is much firmer ground to conclude that it is of major, primary importance. The evidence is consistent in theory, in field studies, in statistics, and in experiments, both on man and animals. But the evidence on the diet, is now so extensive that I refrain from attempting to summarize it here. I have written a good deal on the subject (Keys, et al, op. cit.; Keys, op. cit.).

During the past several years I have attempted to test the theory of a dominant effect of the dietary fat in studies on various natural populations as well as in controlled laboratory experiments. And so far all of the new evidence conforms to the concept presented in lectures in 1952 in New York, in Edinburgh, and in Amsterdam.

TABLE VII.

Serum total cholesterol concentration, mg. per 100 ml., in clinically healthy men habitually engaged in moderate physical activity. For 118 men of Cagliari, Sardinia, the values under "A" were obtained by analysis of the fresh serum in Italy while those under "B" were from aliquots sent to Minneapolis and analyzed there. The values for 150 Swedes of Malmö were obtained by Doctors H. Malmros, G. Biörck and B. Swahn by the same method as used in Italy.

Age	Cagliari		Malmö
	A	B	
19-29	160.7	160.6	189.5
30-39	170.2	168.5	210.3
40-49	193.8	194.7	237.2
50-59	190.4	190.4	236.0

Later in this symposium, Dr. Brock will present some of the new data from South Africa. Here I shall mention briefly only a few other points.

First, exact comparability of serum cholesterol values in the different field studies has now been checked. An example is given in Table VII. We have been able to make duplicate analyses at the place of blood sampling in the field and, again, at the laboratory here in Minneapolis. Measured aliquots of serum are dried on filter paper and sent by letter mail. The example summarized here is from Sardinia. Similar good agreement was obtained in the same way with bloods drawn in other parts of Italy as well as in South Africa.

Second, the data from Sardinia illustrate again the rule that when two populations are compared, the average serum cholesterol concentrations at given age correspond with the percentages of calories provided by fats in the habitual diets of those populations. Table VII compares these men of Cagliari, Sardinia, with men of Malmö, in southern Sweden, who were studied last year by Malmros, Biörck and Swahn after our joint work in Naples. All of these men were engaged in moderate physical activity in their jobs, the Swedes being firemen, the Sardinians being mainly firemen and policemen on outside duty. The general dietary level of the Swedes provides about 38 per cent of calories as fats, while the figure for the Sardinians is about 22 per cent. Both Professor Malmros and Dr. Paul White are here and may wish to comment on the obvious fact that coronary heart disease in southern Sweden is a great deal more prevalent than in Sardinia. The Sardinians are similar to the Neapolitans in this respect.

Third, there is no time here to discuss the interesting question as to the relative cholesterogenic action of different dietary fats. After five years of controlled dietary experiments on human subjects here in Minnesota we can say positively that all fats are *not* equivalent in this respect but that the differences in their effects are not explicable on a simple basis such as relative saturation or iodine number. For example, it may be noted that in controlled experiments on man there is little difference between cottonseed oil and olive oil in their effects on the blood cholesterol. These two fats are interesting, of course, because they differ greatly in the degree of saturation of the fatty acids.

In this connection, it is relevant to cite recent results of Donath et al (1954) who have carefully studied the diets and serum cholesterol of *true* vegetarians in Leiden, the Netherlands. As compared with ordinary Dutchmen (skilled laborers) in the same area, neither the average serum cholesterol nor the proportion of fat calories in the diet differed significantly. But, of course, the vegetarians got only vegetable fats while most of the fat in the diets of the non-vegetarians was provided from animal sources.

Summary and Conclusions

This discussion has reviewed, all too hastily, evidence and reasoning that must be considered in any attempt to explain why there are such great differences between men, and women too, in the tendency to develop severe atherosclerosis and die of coronary heart disease. No convincing brief can be filed for primary importance of heredity, overweight *per se,* the body form, the use of tobacco or alcohol. And it is obvious that being a male is not necessarily fatal.

Physical activity, too, as a factor may be questionable if we demand to be shown that the relatively favorable mortality of manual workers in some populations is unrelated to dietary and other factors. Future research should be planned to test more rigorously the hypothesis that the habitual physical activity has an important influence.

I believe that a powerful effect of the diet on human atherogenesis can no longer be doubted. Though it is easy to point to many unanswered questions about the effect of the diet, it is more and more clear that there is a broad interrelationship between the dietary fats,

(Continued on Page 783)

Lipoprotein Metabolism in the Etiology of Atherosclerosis

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THE occurrence of abnormalities in lipoprotein metabolism may bear a direct causal relationship to atherogenesis. At the present time, however, such a relationship is almost entirely inferential and it is the purpose of this summary to consider, as objectively as possible, the experimental findings and personal convictions which have led to this point of view.

It is now abundantly evident that lipids exist in animal tissues and fluids in the form of lipoprotein complexes and that the metabolic reactions which these substances undergo in an aqueous environment are made possible by the existence of such macromolecules. The direct or indirect involvement of lipids and, in particular, of cholesterol, in atherogenesis is strongly indicated by the chemical and pathologic data that have accumulated from investigations on vessel wall tissue exhibiting atheromatous degeneration. The study of metabolic derangements resulting in such lipid deposition were given fresh impetus by the introduction by Pedersen,[1] Gofman et al[2] Russ, Eder and Barr[3] and others, of the concept of a cause and effect correlation between the levels of certain circulating lipoproteins and the incidence of atherosclerotic heart disease. The elegant methodology introduced by Gofman and his collaborators for the analysis of plasma for its various lipoprotein components must be given particular mention in connection with the stimulation of new approaches to the study of the biochemical and physiological background of this disease. Studies with these physical techniques, as well as by other less complicated but equally useful chemical methods[4,5,6] have also shown a relative elevation of low-density lipoproteins in the plasma of males in relation to females, in the aged in relation to the young, and in connection with those diseases which appear to involve abnormalities in lipid metabolism and predispose to atherosclerosis, such as diabetes and nephrosis. An extensive consideration of these physiological studies and of the chemical literature pertinent to them cannot be attempted in the present discussion. Such summaries have appeared in a number of review articles.[7,8,9,10,11] We shall deal here, instead, with the enzymatic and physicochemical aspects of lipid absorption and transport in an attempt to evaluate the present state of knowledge of these processes in terms of the physiologic and pathologic situations to which they appear to be related.

Chemical Nature and Enzymatic Degradation of Lipoprotein

In a consideration of lipoprotein metabolism we must necessarily confine ourselves to the lipoproteins of plasma and lymph since, although obviously of fundamental importance, the lipoproteins of tissue cells have not been adequately studied. As has been determined in a large number of laboratories, lipids are transported through the extracellular fluids in the form of three major macromolecular classes. The first class is comprised of the extremely large particles, generically known as chylomicrons, which appear post-absorptively in the normal lymphatic drainage of the intestinal wall. Although the basic chemical composition of these triglyceride-rich substances is relatively well known, their detailed architecture is at present a complete mystery and, as will become evident in the subsequent discussion, stands as a major missing item of information necessary for a rational description of lipid transport and mobilization.

The second class, termed beta lipoproteins, are molecules of uncertain heterogeneity containing protein to the extent of about 25 per cent of the particle weight and including varying quantities of cholesterol, phospholipid and triglyceride under different physiologic circumstances. The low-density lipoproteins found in elevated con-

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TABLE I. EFFECT OF PREINCUBATION OF LIPOPROTEIN LIPASE WITH PYROPHOSPHATE

Experiment No.	Sodium Pyrophosphate	Heparin	Glycerol Production
	..moles	..	..mole
1			0.49
2		5	0.57
3	0.5		0.50
4	0.5	5	0.58
5	5.0		0.19
6	5.0	5	0.50
7	0.5*		0.27
8	0.5*	5	0.43

All the vessels contained 0.1 ml. of lipoprotein lipase.

* In these two experiments, 0.5 micromole of pyrophosphate was preincubated with 0.1 ml. of lipoprotein lipase for 30 minutes at room temperature. In the absence of pyrophosphate, there was no decrease in enzymatic activity during a preincubation periol at room temperature.

centrations in certain metabolic disorders are generally of this class, but contain unusually high levels of triglycerides.

The third class, the alpha lipoproteins, contain relatively more protein, and relatively less lipid than the beta lipoproteins and are, consequently, characterized by a higher density. Although the chemical composition of the protein moieties of the alpha and beta lipoproteins is reportedly quite similar, end-group analysis[12] and immuno chemical[13,14] study indicate a major structural and functional difference between these two classes of substances.

Both the relative[6,9] and absolute levels of the alpha and beta lipoproteins of plasma may be closely related to the occurrence of vascular degenerative changes. It is likely, however, that a fundamental understanding of atherosclerosis must finally rest on the elucidation of the mechanisms controlling the synthesis, degradation and resulting extracellular fluid levels of these materials. It is the conviction of most of the workers in this field that the transport and mobilization of lipids in the normal and abnormal animal subject may furnish the most easily approached reflection of these unknown controlling mechanisms.

Our own efforts during the past three years have been concerned to a large extent with the characterization of an enzyme system originally termed "clearing factor" but more accurately, in the light of recent findings, lipoprotein lipase.[15a,b] Our interest in this system developed from the original observations by Hahn[16] and from subsequent studies by Weld,[17] Anderson and Fawcett,[18] Graham et al,[19] and others. The basic observation that alimentary lipemia as well as lipemias present in certain diseases involving disorders in lipid metabolism could be "cleared" by the administration of heparin to the animal or patient led, initially, to the concept of a heparin-induced or activated plasma enzyme which attacked the triglycerides of chylomicrons and low-density beta lipoproteins. Various tissue factors and plasma proteins were postulated as essential components of this system in the early study of the clearing phenomenon.[20] However, only after the application of classical enzyme techniques has the true nature of this system been more closely delineated. Recent work by Edward Korn[15,21,22] in our laboratory suggests very strongly that clearing factor is a tissue enzyme rather than a plasma enzyme. Thus, extracts of acetone powder preparations of heart muscle and, particularly, of adipose tissue[23] yield clearing factor preparations of very high specific enzyme activity which can be shown to depend upon the presence of heparin for normal lipolytic action against the triglycerides of chylomicrons, low-density beta lipoproteins, and synthetic "chylomicrons" prepared by incubation of oil emulsions with certain plasma protein fractions. The heparin dependent enzyme is characterized by its specific ability to attack the triglycerides of lipoproteins and chylomicrons in contradistinction to the ordinary non-heparin dependent lipases present in intestinal secretions and, ubiquitously, in tissues whose specificities are directed toward the hydrolysis of non-protein bound triglyceride compounds. This marked specificity is the basis for the term "lipoprotein lipase."

In view of the fact that lipoprotein lipase is predominantly localized in tissues it now appears that, except for the low levels of this enzyme which have been demonstrated in some instances in normal plasma, the high levels of activity in post-heparin plasma samples probably indicate a spilling over of activated lipoprotein lipase from tissue sites into extracellular fluids. The activity of lipoprotein lipase prepared either from post-heparin plasma[25] or from tissue extracts[21] is strongly inhibited by protamine and elevated concentrations of inorganic salts as well as by a number of phosphate derivatives including pyrophosphate and various nucleotides. The question of the presence or absence of heparin as a component prosthetic group of the enzyme will only be unequivocally resolved

TABLE II. ABILITY OF DIFFERENT SERUM FRACTIONS TO ACTIVATE COCONUT OIL.

Experiment No.	Fraction	Serum Equivalent	Glycerol Produced
		ml.	..mole
1	None	0	0.06
2	Whole serum	0.2	0.86
3	..1-lipoproteins	0.2	0.54
4	..1-lipoproteins	0.4	0.86
5	..-lipoproteins	0.2	0.14
6	..-lipoproteins	0.4	0.14
7	Residual serum after removal of lipoproteins	0.1	0.06

The coconut oil was preincubated for 30 minutes at 37° with the fractions indicated before incubation with lipoprotein lipase.
The substrate, enzyme, and other components were mixed at 0° and then incubated at 37°. All the vessels contained 0.2 ml. of 10 per cent albumin (pH 8.5), 0.02 ml. of 1 M CaC12, 10 micromole of heparin, and 0.28 ml. of 0.25 M NH4Cl-NH3 buffer (pH 8.5) in a total volume of 1 ml.

when the enzyme has been purified to a state permitting direct analysis for this material. The evidence at present available permits only the conclusion that heparin is required for lipoprotein lipase *action* since the pyrophosphate inhibited enzyme may be reactivated by the addition of small amounts of heparin[21] (Table I).

The over-all reaction carried out by lipoprotein lipase can be summarized in the following equations:

A. Substrate

1. Chylomicrons, low-density β-lipoproteins
2. Synthetic "chylomicrons."
   Triglyceride emulsion + α-lipoprotein → "activated" triglyceride

B. Hydrolysis

$$\begin{array}{l}\text{"Activated"}\\ \text{triglyceride}\\ \text{Lipoprotein}\\ \text{lipase}\end{array} \xrightarrow[\text{or Ca}^{++}]{\text{albumin}} \begin{array}{l}\text{Complexed fatty acids}\\ \text{+glycerol (+mono \&}\\ \text{di-glycerides during}\\ \text{early stages)}\end{array}$$

Experiments on the "activation" of synthetic oil emulsions by various plasma protein fractions prior to lipoprotein lipase action have indicated, as shown in the equations above, that the most potent activating component in plasma is the alpha lipoprotein moiety. One such experiment on the triglyceride-activating effect of various plasma components is shown in Table II.[22] It can be seen from the data in this table that the alpha lipoprotein component accounts for essentially all the activating effect of serum since both the beta lipoprotein fraction and the residual serum proteins present after removal of the lipoproteins by ultracentrifugal flotation are almost completely without effect. These findings on the activation of triglyceride emulsions for lipoprotein lipase action suggest that the system described in the equations above may, indeed, be a model for a cyclic mechanism for the transport of neutral fat from the intestine to metabolically active tissues (Fig. 1).

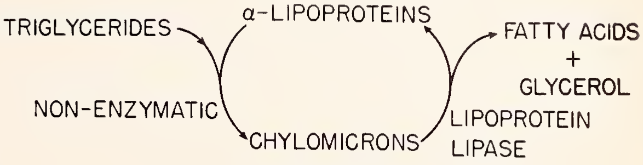

Fig. 1.

As can be seen from the equations above, the hydrolysis of lipoprotein-triglyceride requires the presence of certain fatty acid acceptors and binders which may be either divalent cations or serum albumin. Although possible fatty acid binders during the intracellular action of lipoprotein lipase may be divalent cation, or specific cellular proteins, the major transport form in the circulating blood of the unesterified fatty acids resulting from lipoprotein lipase and other possible lipolytic action is almost certainly the serum albumin-fatty acid complex.[26,27]

In earlier experiments on the electrophoretic mobility of plasma proteins following the administration of heparin to hyperlipemic patients it was found that the mobility of a portion of the beta globulin peak was markedly shifted as a result of complex formation with fatty acids liberated during the clearing reaction. Thus, in Figure 2 it can be seen that a portion of the beta globulin peak has shifted to a position in the electrophoretic pattern into and beyond the area occupied by serum albumin (Fig. 2, b, c, d). With time the mobility of this protein component returns to its initial value as in Figure 2f. Such electrophoretic changes could be induced equally well by the addition of an amount of sodium oleate approximately equivalent to that released under the conditions of the *in vivo* clearing. In later experiments, however, it has been shown that serum albumin, possessing a higher affinity for fatty acid is the major vehicle for fatty acid transport. As has been demonstrated a number of times previously, the electrophoretic mobility of serum albumin varies in proportion to the unesterified fatty acid level with which it is in equilibrium up to the maximum level of its effective binding capacity. In general, it would appear that, except for the situation in which large doses of heparin are administered to hyperlipemics, unesterified fatty acid transport in plasma is normally carried out entirely by serum albumin.

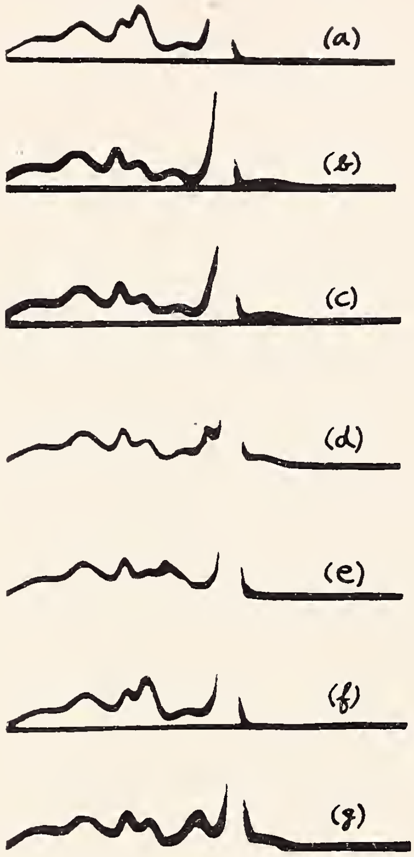

Fig. 2.

Physiological and Clinical Evidence Supporting the Importance of Lipoprotein Lipase in Normal Fat Metabolism

The importance of the lipoprotein lipase system in the regulation of fat transport and metabolism is suggested by several physiological observations in both animals and humans. In the light of *in vitro* chemical studies on the inhibitory effect of protamine[20] on lipoprotein lipase, Bragdon and Havel have carried out experiments[28] on the nature of the plasma lipoprotein picture following protamine administration in fasting rats. Their results are summarized in Figure 3. It can be seen that the administration of protamine resulted in a rapid and quantitatively significant increase in total plasma lipids which decreased to normal levels with time. They found, in addition, that this transient increase in plasma lipids could be accounted for by a specific rise in the triglyceride-rich beta lipoproteins of low density. The effect of protamine was reversed by the administration of heparin.

A clinical observation has recently been made by Havel and Gordon[29] which may represent an abnormal metabolic situation which is mimicked by the protamine experiments above. In a patient with idiopathic hyperlipemia and in two hyperlipemic siblings it was shown that the administration of anticoagulant doses of intravenous heparin did not lead to the production of more than minute traces of plasma lipoprotein lipase although the abundant appearance of this enzyme has invariably been observed in other individuals given such treatment. The excess lipid in this patient's plasma was present entirely in particles having the flotation and chemical characteristics of chylomicra. Alpha and beta lipoprotein concentrations were markedly reduced. On a fat-free diet his serum triglyceride content fell to within normal limits and the alpha + beta lipoprotein concentrations increased. Caloric restriction and repletion while the patient was on the fat-free diet failed to produce significant alterations in serum lipid and protein concentrations although the R.Q. during restriction was reduced to 0.73. This suggests that endogenous lipid transport was being effected but perhaps by a mechanism differing from that of exogenous lipid transport. Acute and intensive heparin administration failed to reduce significantly the patient's abnormal lipoprotein and triglyceride concentrations. Although an assumption must be made in suggesting that the observed defect in lipoprotein lipase

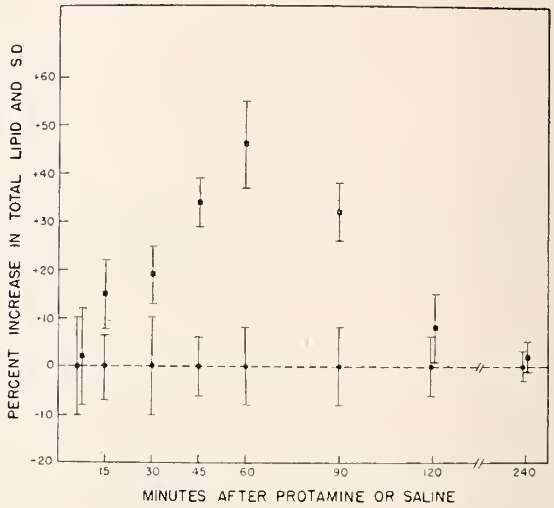

Fig. 3.

activity may account for the slow rate of removal of ingested fat from the blood in this patient, such an assumption does receive a certain degree of support from our present knowledge concerning the properties and distribution of lipoprotein lipase.

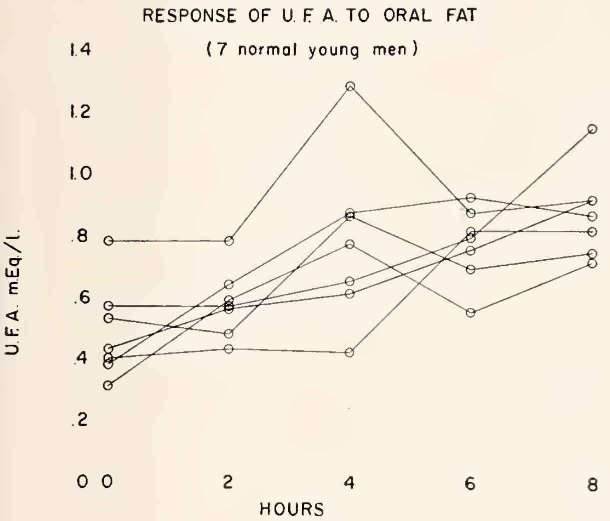

Fig. 4.

Finally, let us consider a series of observations[30a,b] recently made by R. Gordon and Amelia Cherkes in connection with the regulation of the unesterified fatty acid levels of circulating blood. The unesterified fatty acids (UFA) of normal blood, both in males and females, is approximately 0.5 milliequivalent per liter. Following the administration of oral fat to individuals fasted overnight, there is a moderate increase in this level, which persists for many hours (Fig. 4). Further experiments, however, showed a similar increase in blood unesterified fatty acids following fasting (Fig. 5), and suggested that the increased blood fatty acid level observed following oral fat was not due to the fat itself, but to the homeostatic influences leading to the degradation of depot fat in response to reduced blood glucose levels. The administration of oral glucose (Fig. 6) led to a marked decrease in the blood unesterified fatty acid level, followed by a marked rise after approximately two hours. In view of the well-known delayed hypoglycemia observed following glucose administration, which is believed to involve a homeostatic release of epinephrine, experiments were carried out on the effect of infused epinephrine on the plasma levels of unesterified fatty acids. It can be seen in Figure 7 that epinephrine has a marked effect, doubling or more than doubling the concentration of these substances in the individuals tested.

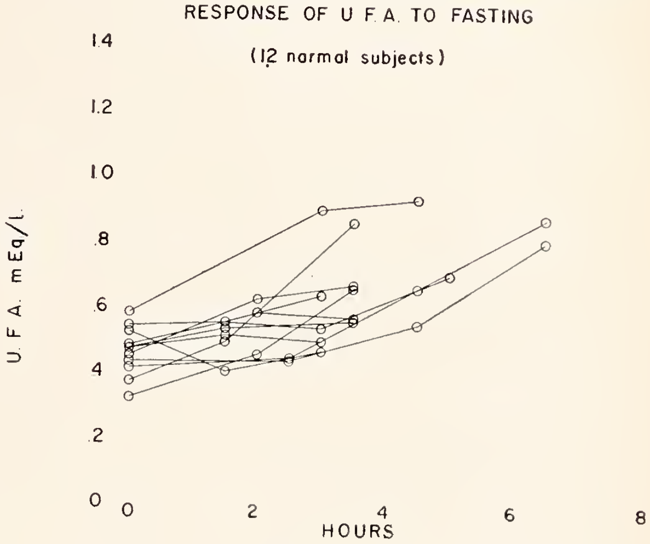

Fig. 5.

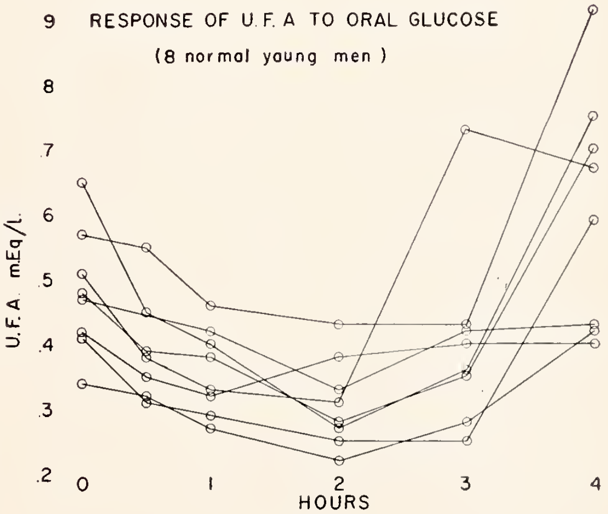

Fig. 6.

Studies with C^{14}-labelled long-chain fatty acids have been carried out in conjunction with these experiments by Havel and Fredrickson (unpublished results) as a means of labelling the triglycerides present in chyle. A study of the distribution of radioactivity in the various lipid fractions of blood following the infusion of such labelled chylomicrons suggests that the triglycerides of chyle are carried to tissues (mainly adipose?) and there deposited and, when required for nutritional purposes, re-secreted into the circulating blood as unesterified fatty acid. This over-all process may occur without direct metabolic involvement of the normal alpha or beta components of plasma since the specific activity of the blood unesterified fatty acids was nearly as high as the specific activity of the fatty acids in the triglycerides of the infused chylomicrons (free fatty acids were present in almost undetectable quantity in the administered chylomicrons) and since the radioactivity present in the plasma unesterified fatty acid fraction was found to increase earlier and more rapidly than that of the lipoprotein components of plasma.

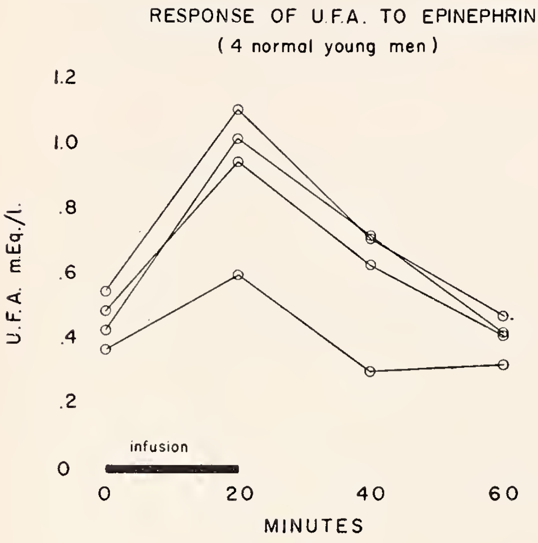

Fig. 7.

Experiments on the arteriovenous differences in plasma fatty acid levels[30a] have emphasized their metabolic importance and short half-life. Indeed, it can be calculated, in the case of myocardium, that the fatty acids removed by this tissue can account in several instances for the major share (as much as 70 per cent) of its caloric requirements. In the case of arteriovenous difference studies on samples drawn from arterial blood and from the inferior or superior vena cava, a negative arteriovenous difference is observed, indicating the release of fatty acids from the adipose-rich areas supplied by this circulatory pathway.

General Consideration of Triglyceride Transport and Metabolic Homeostasis

It is of interest to attempt to trace the path of ingested lipid material from the chyle through the metabolic tissues of the body in relation to the enzymatic, physical and physiological observations outlined above. Evidence from a number of laboratories supports the view that triglycerides are probably extensively hydrolyzed in passing from the gut through the intestinal wall into the chyle possibly in combination with alpha lipoprotein (as in Figure 1) and surrounded with a serum albumin sheath. Such a conclusion is based on the finding that triglycerides, labelled by radioactive means or fed with a known synthetic arrangement of fatty acids in the triglyceride molecule,[31] are randomized in the chylomicron triglycerides with respect to the hydroxyl groups on the glycerol moiety to which they are esterified. The studies with radioactive isotopes described in the previous section suggest that this newly formed triglyceride passes as chylomicrons to metabolizing tissues and depot areas as shown in Figure 8 and that subsequently, depending on nutritional situations in the plasma and on resulting hormonal control (Regulatory Mechanism No. 1 in Figure 8) as suggested by the epinephrine studies, are either stored as triglyceride or secreted as unesterified fatty acid for transport to other areas of the body.

Direct measurements of the arteriovenous differences across the myocardium of unesterified fatty acids, glucose, and oxygen have added support to this scheme. Thus under conditions of lowered blood sugar levels, fatty acid utilization accounts for a large share of the caloric requirements of the myocardium. Upon intravenous administration of glucose, the energetic economy of the muscle shifts to carbohydrate, and the arterial and venous levels of unesterified fatty acids are essentially identical. Of particular interest is the finding that the UFA level *entering* the myocardium is markedly decreased following glucose administration, suggesting that the blood carbohydrate level has, through hormonal interactions, decreased the release of UFA from adipose areas.

The physiological observations described earlier, in which either protamine administration in animals (and perhaps in what may be construed as a genetic defect in the patients unable to produce lipoprotein lipase) suggest that in addition to the triglyceride-fatty acid transport system conditioned by glucose levels in the blood, there may also be a secondary mechanism in which lipid is transported (Regulatory Mechanism No. 2) when needed, in the form of lipoprotein-bound neutral fat. Such a mechanism would, then, become operative when the lipoprotein lipase system was either absent, or unable to supply adequate supplies of foodstuff in the form of fatty acids. Such a mechanism would imply further the operation of phagocytic uptake of triglyceride-containing lipoproteins with subsequent metabolism through non-heparin-linked lipase systems. It is tempting to speculate whether or not the lipemia of starvation might not represent a situation in which elevated levels of plasma lipid are due, not to the absence of lipoprotein lipase as in the hyperlipemic siblings described above, but rather to a deficiency in hydrolyzable substrate in the extensively depleted depots.

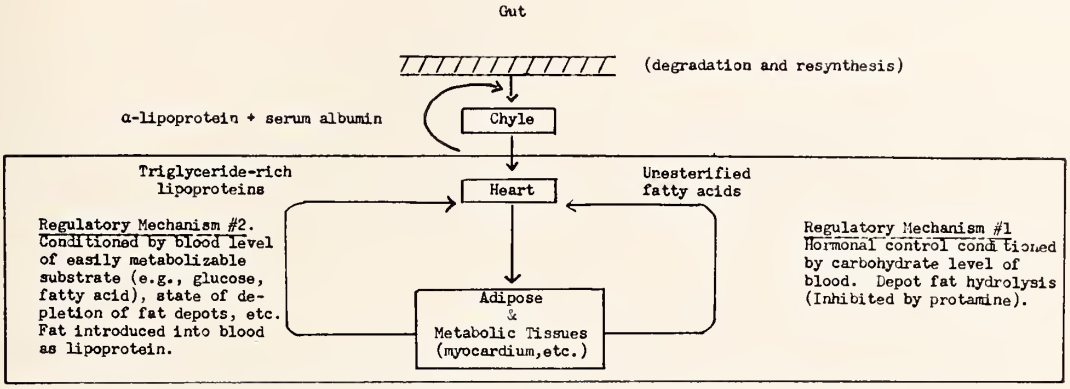
Gut
(degradation and resynthesis)
α-lipoprotein + serum albumin
Chyle
Triglyceride-rich lipoproteins
Heart
Unesterified fatty acids
Regulatory Mechanism #2. Conditioned by blood level of easily metabolizable substrate (e.g., glucose, fatty acid), state of depletion of fat depots, etc. Fat introduced into blood as lipoprotein.
Adipose & Metabolic Tissues (myocardium, etc.)
Regulatory Mechanism #1 Hormonal control conditioned by carbohydrate level of blood. Depot fat hydrolysis (Inhibited by protamine).

Fig. 8. Scheme for triglyceride transport and metabolic homeostasis.

Direct support for Regulatory Mechanism No. 2 has come from further study of the protamine experiments described above (in which a transient lipemia was induced by administration of this material to fasting rats). Gordon and Bragdon have recently found that glucose, given simultaneously with protamine, completely prevents the lipemia observed in the protamine treated control rats with fasting blood sugar levels.

The discussion above cannot obviously be based in fact at the present time. Nevertheless the existence of the complex system of checks and balances known to be operative in carbohydrate metabolism must, it seems likely, find its analogy in the case of lipid metabolism. The specificities and properties of the lipoprotein lipase system may furnish a useful experimental wedge in the study of these phenomena, particularly in the light of the admittedly meagre though stimulating physiological observations that have begun to accumulate.

Atherosclerosis and Lipoproteins

It is clear that little more can be said, specifically, to relate lipoprotein metabolism to the process of atherogenesis. The suggestion has been made[32] that the slow but significant filtration of plasma proteins through tissues into the lymph might, in part, account for the early stages of vascular degeneration. Thus large lipid-laden lipoproteins are incompletely removed from the wall of the vessel or from the intervascular tissue areas through which they have filtered and may remain, after variable degrees of degradation, as centers for subsequent fibrosis and degeneration. Such a mechanism might well be augmented by local tissue damage or by hydrostatic effects peculiar to the area in question.

A great deal has been said in the past about the possible role of chemically and metabolically specific low-density beta lipoproteins as etiologic agents in atherosclerosis. The now well-established physical heterogeneity of the low-density beta lipoprotein spectrum of proteins considered in conjunction with the apparent immunochemical identity of these substances, make it begin to appear that the family of low-density beta lipoproteins may differ from one another only in the degree to which they are laden with non-specifically bound triglyceride, phospholipid and cholesterol. Should it evolve, through further study, that low-density lipoproteins are, indeed, a direct causal agent in atherogenesis, it seems not unlikely that their deleterious effects might be due in large part to their molecular size and to the metabolic problem they present to the tissues into which they penetrate. Even the much maligned cholesterol molecule may ultimately become secondary in importance to the problem of neutral fat transport and metabolism.

The author is extremely grateful to his colleagues, Drs. Bragdon, Gordon, Havel, Korn, Fredrickson and Steinberg for their generous help and advice in the assembly of this discussion.

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COLDS AND RESPIRATORY INFECTIONS IN THE CARDIAC PATIENT

The question, "How important are respiratory infections as a cause of heart failure?", is examined in a recent issue of *Diuretic Review*.

Recently published papers in the professional literature point to general agreement, *Diuretic Review* reports, that respiratory infections, particularly colds, are the most common irritating and aggravating factors in congestive heart failure.

In a study of more than 300 cases of heart failure, a British medical investigator found more than half with some type of respiratory infection.

Furthermore, it was established that the infection was the precipitating cause of decompensation in 156 of the 300 cases. In a similar study conducted by three investigators and reported in the *American Journal of Medical Science,* a direct relation was found between the frequent occurrence of heart failure among cardiac patients and onset of "even minor colds."

The British investigator, Dr. F. J. Flint, believes respiratory infection in the person with a weak heart may initiate cardiac failure by causing damage to the heart muscle or by favoring congestion of the lungs.

Under any circumstances, *Diuretic Review* points out, these studies emphasize the importance of looking for and treating respiratory infection in cardiac patients.

The Lipoprotein System

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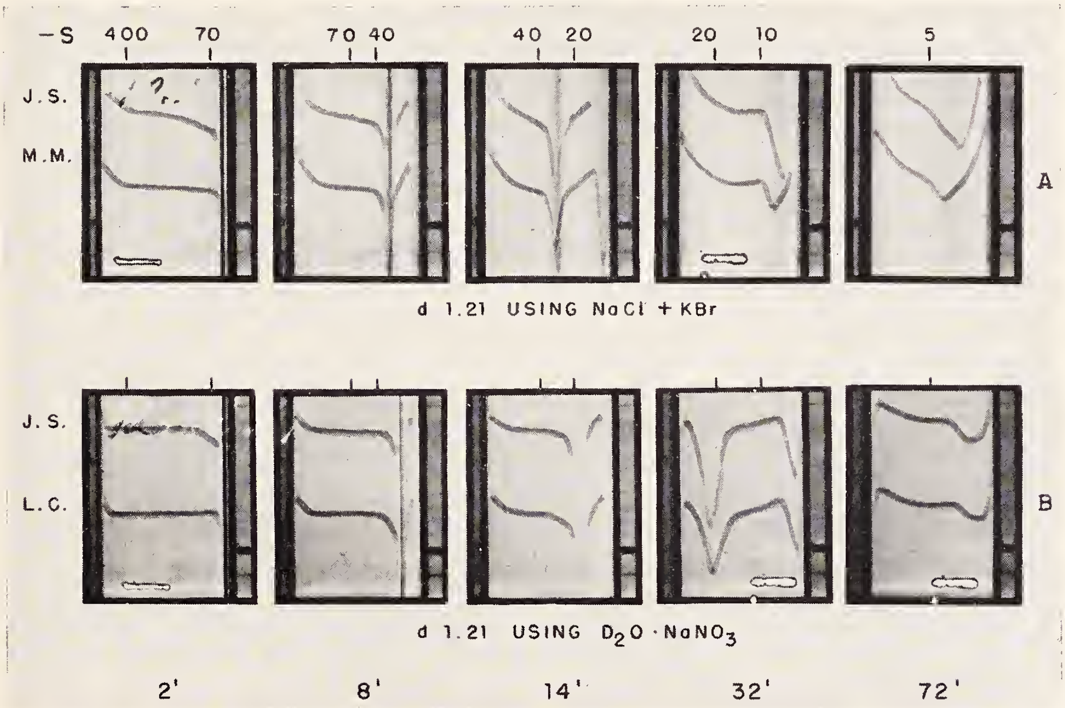
Cleveland, Ohio

MANY methods of investigation of lipoproteins have been developed since the original description of a lipoprotein in 1929 by Macheboeuf.[1] The application of high density ultracentrifugation for the concentration of serum lipoproteins by Pedersen,[2] and of the analytical ultracentrifuge for their characterization by Gofman and co-workers,[3] provided a great impetus to further studies as to their nature and physiology.

The studies of serum lipoproteins to be reported here were carried out by ultracentrifugation either at a density of 1.063 using NaCl, as originally described by Gofman,[2] or at d 1.21 using NaCl and KBr.[4] If the preparative ultracentrifugation is carried out for seventeen hours at 16° to 18° C., a lipoprotein concentrate is obtained, which in the case of KBr·NaCl system contains most of the serum lipoprotein. Figure 1 (a) shows the type of pattern obtained on normal serum using d 1.21, KBr·NaCl. A few studies were also made at density of 1.21 using D_2O and $NaNO_3$.[5] When this system is employed, longer preparative centrifugation at a higher "g" is necessary to obtain satisfactory lipoprotein concentration. Comparison of flotation rates at d 1.21 with the two systems (Fig. 1, a and b) shows a considerably slower rate with the D_2O-$NaNO_3$ system. If centrifugation is carried out for a sufficiently long time, a like degree of resolution is obtained.

The question, "What is the serum lipoprotein pattern of the normal adult human being?", is frequently asked. A group of normal controls



Comparative flotation rate of human serum lipoproteins studied at a density of 1.21 using, (a) NaCl + KBr, and (b) $D_2O \cdot NaNO_3$, in the analytical ultracentrifuge at 52,640 r.p.m.

From the Research Division of the Cleveland Clinic Foundation and the Frank E. Bunts Educational Institute, Cleveland, Ohio.

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

TABLE IA. SERUM PROTEINS, ELECTROPHORETIC TECHNIQUE, OF CLEVELAND CONTROLS, PERUVIAN AND NAVAJO INDIANS AND CLOISTERED MONKS.

	Total Protein	$A+\alpha_1$-globulin		α_2-globulin		β-globulin		γ-globulin		
		gm.	%	gm.	%	gm.	%	gm.	%	Age
Cleveland Controls Mean	7.44±	4.74	63.5±	0.54±	7.2±	1.11±	15.2±	1.04±	14.1±	29.5±
S. E. of Mean	0.15	±0.11	1.09	0.04	0.36	0.03	0.40	0.05	0.56	1.72
Peruvian Indians Mean	7.53±	4.34±	57.8±	0.52±	6.93±	1.17±	15.6±	1.49±	19.0	27.5±
S. E. of Mean	0.17	0.12	2.2	0.04	0.40	0.09	0.65	0.11	±1.57	2.6
Navajo Indians Mean	6.69±	3.25±	48.6	0.66	9.9	1.21±	18.2±	1.56±	23.2±	56.0±
S. E. of Mean	0.23	0.08	±1.0	±0.04	±0.52	0.03	0.54	0.08	1.08	3.2
Monks Mean	6.92±	4.44±	64.1±	0.39±	5.8±	1.08±	15.1±	1.00±	15.4±	30.3±
S. E. of Mean	0.11	0.10	1.40	0.05	0.70	0.05	0.72	0.06	0.75	3.4

TABLE IB. SERUM LIPOPROTEINS, ULTRACENTRIFUGAL TECHNIQUE D 1.21, USING KBR + NACL, OF CLEVELAND CONTROLS, PERUVIAN AND NAVAJO INDIANS AND CLOISTERED MONKS.

	Total Chol. mg./100ml.	-S70-400 mg./100ml.	40-70 mg./100ml.	25-40 mg./100ml.	20-25 mg./100ml.	1-10 mg./100ml.
Cleveland Controls Mean	229.7±	36.7±	24.2±	>220	11.6±	176.9±
S. E. of Mean	4.4	3.8	1.8		1.8	6.5
Peruvian Indians Mean	186.0±	91±	33±	208±	18.0±	197.5±
S. E. of Mean	11.8	14.1	3.6	16.7	4.2	17.6
Navajo Indians Mean	175±	17±	23±	206±	13.0±	172.9±
S. E. of Mean	6.2	2.6	2.4	9.1	1.5	9.7
Monks Mean	184±	34±	34.6±	197±	14.0±	124.2±
S. E. of Mean	14.9	4.5	4.6	18.7	2.0	20.7

was selected from the personnel of the Cleveland Clinic, and of a Cleveland industrial concern in whom serum lipoprotein and cholesterol determinations were made. Two-thirds of this group were male. A similar group had previously been analyzed as normals for serum electrophoretic proteins. We have also compared sera of fourteen normal male Peruvian Indians living at high altitudes with those of fifty-five Navajo Indians of mixed sexes. Serum analyses were done on nineteen normal cloistered monks on a uniform, well-balanced dietary regimen which contained about protein 51, fat 34 and carbohydrate 267 gm. per day, equivalent to 1,591 cal.

Diet of the Peruvian natives contains very little fat and the main caloric source is vegetables and a little meat. The Navajo Indian diet, unlike that of the Peruvian Indians, is not low in fat but similar to that common to Americans generally.

Table I, a and b, summarizes the lipoprotein and electrophoretic protein data obtained on the four groups. The serum total protein of the Cleveland controls and that of the Peruvian Indians were nearly the same, except that the concentration of albumin was lower and that of the γ-globulin higher in the Peruvians. These differences were even more intense in the Navajo. The total protein of the monks was lower than that of the Cleveland controls, but the relative proportions of the various proteins were very similar.

The means of total serum cholesterol in all three selected groups were lower than that of the Cleveland group. The means of -$S_{25\text{-}40}$ lipoproteins of the three special groups were consistently lower than that of the "controls." The -$S_{1\text{-}10}$, α_1-lipoprotein, was lower in the monks than in the other three groups. The α_2-lipoprotein concentration in all four groups was the same. The -$S_{40\text{-}70}$ concentration of the Peruvians and monks was very slightly higher than the Cleveland control and Navajo groups. The -$S_{70\text{-}400}$ fraction of the Peruvians was higher than the other groups. All blood samples were taken in the fasting state; this higher value is not attributable to an alimentary lipemia.

Atherosclerosis, for a number of reasons, is relatively uncommon in the American Indian. The data indicate an association of this trend in Navajos and Peruvians with a relatively low concentration of serum β-lipoprotein and with relative increases in γ-globulin.

Some of the differences between the groups may be due to their varied dietary habits; in the case of the Peruvian Indians who lived at an altitude of approximately 3,750 meters environment may have played a part and genetic factors may have their effect.

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Minneapolis District Dental Soc.
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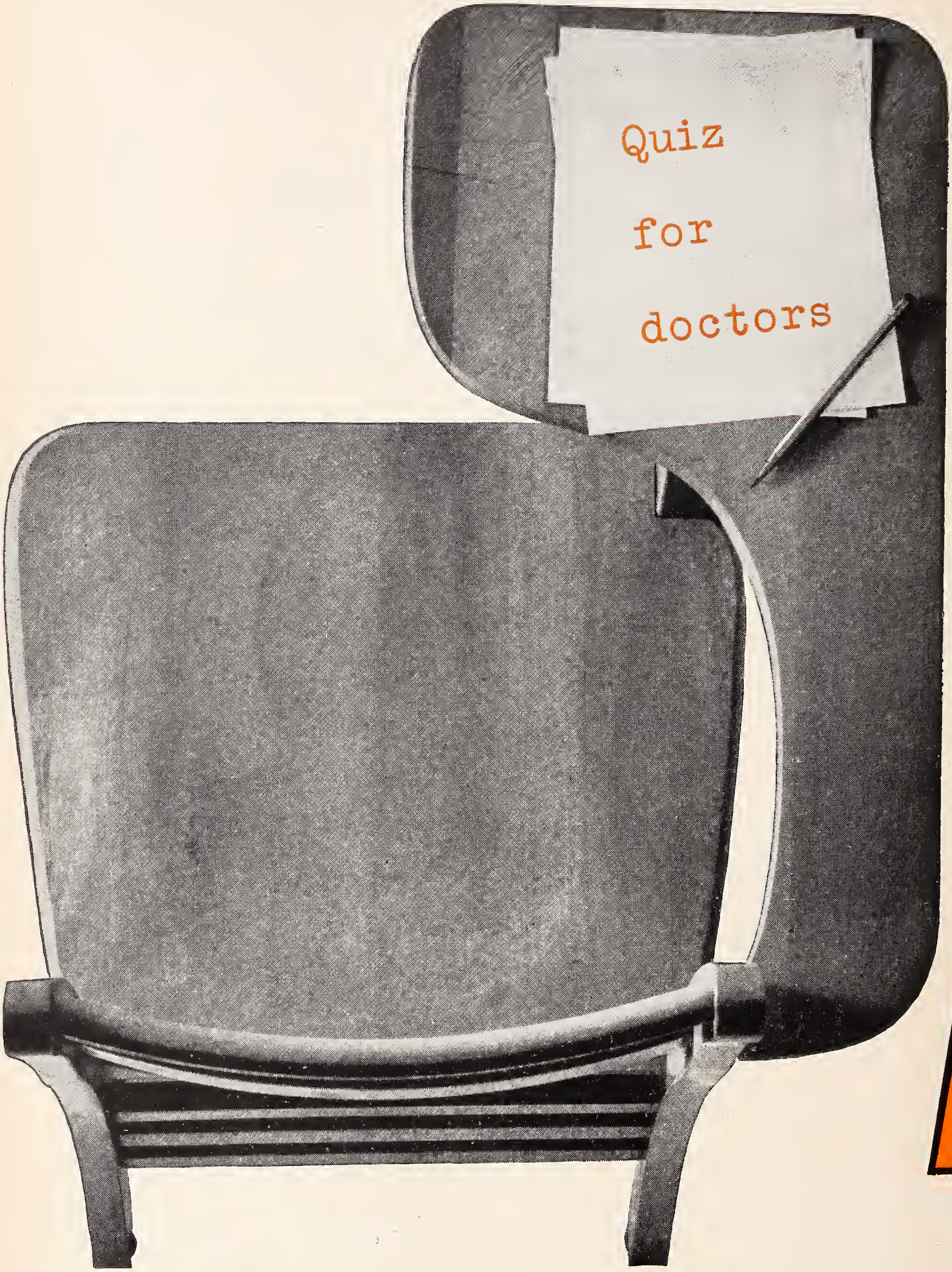
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The importance of heredity as a factor in establishing the lipoprotein pattern is emphasized by results obtained on two strains of dwarf pigs, the so-called long-lean, and the short-fat as bred at the University of Minnesota. Table II shows representative serum cholesterol and lipoprotein values obtained on a pig of each strain. While both strains of pigs received the same diet which consisted of SMA formula*, corn and oats, the concentrations of cholesterol and lower density -S$_{15-25}$, $_{25-40}$, $_{40-70}$ and $_{70-400}$ components were greater in the short-fat animal than in the long-lean.

TABLE II. SERUM LIPOPROTEIN PATTERNS OF LONG-LEAN AND SHORT-FAT STRAINS OF DWARF PIGS.

	Age	Serum Lipoprotein mg. per 100 ml.						Cholesterol mg. per 100 ml.
		-S>400	70-400	40-70	25-40	15-25	1-15	
Long-lean Strain #6	4 mo.	++	7	0	56	33	212	111 } 5 hr. after feeding
Short-fat Strain #1	4 mo.	+++	30	9	82	52	235	166
Long-lean Strain #6	12 mo.	0	21	21	56	71	246	145 } 16 hr. after feeding
Short-fat Strain #1	12 mo.	0	49	35	114	106	198	188

TABLE III. SERUM LIPOPROTEINS OF FAMILY WITH HEREDITARY HYPERLIPEMIA, DETERMINED BY ULTRACENTRIFUGATION AT D 1.21 (NACL + KBR).

mg./100 ml. serum

	-S70-400	40-70	25-40	20-25	1-10	Chol.	Age
Father	223	42	82	70	136	151	72
Mother	177	132	>235	30	136	292	—
Son No. 1	382	82	110	63	176	333	37
Son No. 2	>300	62	98	43	115	248	27
Son No. 3	189	51	53	46	65	208	<27
Son No. 4	28	54	188	30	136	—	<27

Similarly importance of hereditary factors is evident in humans exhibiting hereditary hyperlipemia. Table III summarizes the serum lipoprotein data on such a family. Three of the four off-spring showed abnormally high concentration of -S$_{70-400}$ components, while all four showed values of -S$_{40-70}$ and $_{20-25}$ higher than normal for their age.

The lipoprotein pattern of a given normal individual varies relatively little over a period of years. In a group of seventy-seven studied yearly for three to five years those with high S$_{f\ 12-20}$ and $_{20-100}$ and cholesterol concentrations initially usually showed similar high values on subsequent studies and those with low values remained so. Table IV gives the results on three representative subjects.

TABLE IV. SERUM LIPOPROTEINS OF NORMAL INDIVIDUALS DETERMINED ULTRACENTRIFUGALLY AT D 1.063, AT YEARLY INTERVALS.

Subject No.	Date	Age at Initial Study	mg./100 ml. serum		
			Sf12-20	Sf20-100	Chol.
A	5-51	43	77	77	335
	5-52		84	88	335
	5-53		69	78	330
	5-54		62	146	348
B	3-51	51	33	50	—
	3-52		29	42	—
	3-53		43	67	257
	3-54		30	34	245
	3-55		20	64	252
C	6-51	48	20	12	—
	6-52		21	46	186
	6-53		19	45	179
	5-54		6	72	205
	5-55		6	31	196

While the lipoprotein pattern remains relatively stable when the animal is in good health and on a normal diet, rapid changes can be produced by disease or modification in diet. Recent studies carried out in collaboration with George Wilgram, University of Toronto, of lipoproteins of rats during choline deficiency showed a decreased concentration of the low density -S$_{70-400}$ and of the α_1-lipoproteins in male rats fed a high fat, choline-deficient diet. It is interesting that despite the decreased concentration of lipoproteins the animals developed cardiovascular disease, i.e., cardiac necrosis, coronary lipidosis and aortic sclerosis.

The serum lipoprotein pattern of rats is greatly modified by the production of the nephrotic syndrome following injection of nephrotoxic serum.[6] Increased concentration of all lipoprotein components, but chiefly in the lower density fractions, is observed. Less severe changes of similar type occur in rats within forty-eight hours following unilateral nephrectomy and ureter ligation. Little change in the lipoprotein pattern is observed in the rat following bilateral nephrectomy. Thus the changes in the lipoprotein pattern of rats with renal lesions depend on the nature of the renal damage.

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*We are indebted to Dr. George E. Farrar, Wyeth, Inc., for generous supplies of SMA.

In contrast with rats, in the rabbit relatively great increases in serum low density lipoproteins and cholesterol occur following bilateral nephrectomy. Similar changes and of similar magnitude have been observed in rabbits' sera following DCA, cortisone, or DCA and renin treatment. The lipoprotein shifts in the rabbit seem to be produced more easily and from more varied factors than in the rat.

Summary

In any consideration of lipoproteins, the method of study, type of experimental animal used, hereditary, dietary, climatic background and physiological status of the animals, must be carefully evaluated. These and many more factors undoubtedly are involved in the lipoprotein story.

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Discussion

Ancel Keys, University of Minnesota: Dr. Lewis' data on Navajo Indians are particularly interesting in view of her statement that these people live on a diet similar to that of Americans in general. If it is actually the case that the Navajo get something like 40 per cent of his calories from fats in the diet, then it is remarkable that the lipoprotein and cholesterol concentrations in their blood serum resembles those in serum from the Peruvian Indians whose diet is, as I can testify from personal experience, far lower in fats. It would seem to be most desirable to get detailed data on the diets of these Navajo Indians. In particular, it would be important to have information on the contribution of different types of fats to their total calories. For example, it is hardly possible that the adult Navajo has as high a consumption of butterfat as is customary here in Minnesota.

In Dr. Lewis' data it is notable that the Navajo Indians she studied are much older than the other groups. This means, of course, that their serum cholesterol and lipoprotein values are not comparable because in almost all population groups there is a great change from the values common to men of thirty years of age to those in men fifty-six years old. But this age difference, actually, only emphasizes the discrepancy with other data in the literature, including our own on two population groups of Spaniards, six of Italians, three of Swedes, two of Sardinians, nine of South Africans and three of Minnesotans.

So we should like to know the details of the Navajo diet, the relative obesity of the men, their habitual level of physical activity, and other items that may influence the picture.

The Monks studied by Dr. Lewis are reported to get about 35 per cent of their calories from fats in the diet. This is, of course, considerably less than the U. S. average and is in conformity with the reported values for their serum cholesterol.

In this connection it is of interest to note that Dr. J. Groen of Amsterdam has recently completed a study on a large number of Catholic Monks in two different orders in the Netherlands. When the averages of these are plotted for decades of age, both groups show the familiar rise of serum cholesterol with age until fifty or sixty years and some decline thereafter. But the serum cholesterol values for the Benedictines are much higher than for the Trappists at given ages. Further, it is interesting that Dr. Groen found many more very old Trappists than Benedictines, suggesting that there may be a real difference in longevity between them. In any case it will be noted that the Benedictines live, in regard to diet, much as do other Dutchmen. The Trappists, however, live on a very spartan diet.

COFFEE, CAFFEINE AND CHEMISTRY

Recent laboratory analyses indicate wide variations in the amount of caffeine extracted from various types of "regular" and instant coffee, according to Drs. Robert G. Martinek and Walter Wolman. In assays conducted in the chemical laboratories of the American Medical Association, the investigators found that although various brands of regular ground bean coffee did not vary significantly in caffeine-content, "Almost twice the amount of caffeine per cup can be obtained from the regular ground bean coffee as from the regular instant coffee; . . . Caffeine content per cup prepared from the regular ground coffee bean is roughly three times that of the regular decaffeinated coffee and about four to eight times that of instant decaffeinated coffee."—Martinek, R. G., and Wolman, W.: Xanthines, Tannins, and Sodium in Coffee, Tea, and Cocoa, *Journal of the American Medical Association,* 158:1030 (July 23) 1955.

Cholesterol Metabolism

IVAN D. FRANTZ, JR.
Minneapolis, Minnesota

THE importance of cholesterol metabolism as a field of knowledge in its own right is scarcely open to question. That this subject bears any direct relationship to human arteriosclerosis, or that it is entitled to a place on the program of a symposium such as this, is much more debatable. The general topic of cholesterol metabolism has formed the subject of two recent reviews.[1,2] It is appropriate, therefore, for me to confine my remarks to the question of its relationship to arteriosclerosis, either real or supposed. These remarks will necessarily be speculative, because actual knowledge is lacking.

It is well known that cholesterol is a principal constituent of the atherosclerotic plaque. Although the possibility has recently been raised that this cholesterol is synthesized in the arterial wall itself,[3] most observers are agreed that it is derived from the blood. Evidence is at hand to indicate that injury to the vessel wall can predispose to the deposition of cholesterol.[4,5] Elevation of the blood cholesterol, produced by experimental means in a variety of animals,[6,7,8,9] can also cause the formation of plaques. Since the blood cholesterol of man is higher than that of most other animals, it is understandable that this characteristic should have been given credit for the much higher incidence of atherosclerosis in man. Studies of patients with outspoken manifestations of atherosclerosis indicate that such patients have, on the average, an abnormally high blood cholesterol.[10,11,12] The correlation is so poor, however, that other important factors must also be involved. More or less successful attempts have been made to implicate the cholesterol to phospholipid ratio,[13] the distribution of cholesterol between alpha and beta lipoproteins,[14] and the size and composition of the lipoprotein molecules as measured by their rate of flotation in the ultracentrifuge.[15] In the discussion to follow, I am going to assume that, other things being equal, atherosclerosis develops more rapidly if the blood cholesterol is in a condition which favors its separation from its stable colloidal state, either due simply to a high concentration, or to the nature of the lipoprotein aggregate in which it is combined.

When a disease is characterized by the excessive accumulation of a normal bodily constituent, investigations are usually undertaken to determine whether the cause is overproduction or underutilization. It is possible that the high blood cholesterol in man will eventually be traced to one of these mechanisms. Personally, I doubt that either will prove to be the primary cause. This doubt is based on the extreme lability of hepatic cholesterol synthesis, and its strong dependence on external stimuli. Several laboratories have confirmed the observation of Gould and Taylor[16,17] that cholesterol synthesis is depressed by cholesterol feeding.[18,19,20] If a rat weighing 150 grams is allowed to eat a single dose of 100 mg. of cholesterol along with its normal food, its cholesterol synthesis the following morning, as judged by the liver slice technique, is depressed ten-fold. In interpreting these results, one must be wary of the possibility that the apparent depression is due, at least in part, to decreased specific activity of the carbon-labeled precursors of cholesterol (c.f. Medes, Spirtes, and Weinhouse[21]). Recent experiments by Loud, however, in which incorporation of carbon from acetate and of hydrogen from water were followed simultaneously, provide additional evidence that the depression is genuine.[22] We have also studied two stimuli (biliary obstruction and intravenous injection of a surface-active agent) which appear to produce a sudden, large increase in the rate of cholesterol synthesis.[23,24] This lability may be regarded as an important factor in the homeostatic regulation of the cholesterol content of the body. An increase in the cholesterol content of the diet or a change in the rate of excretion or destruction of cholesterol might be expected

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 7, 1955.

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to be nullified in large measure by an immediate change in the rate of cholesterol synthesis. It seems likely to me, therefore, that we must probe beyond the rates of synthesis, destruction, absorption, and excretion for the primary cause of the elevated blood cholesterol in man.

I should now like to suggest a point of view which may help to account for a number of the facts mentioned above. The central idea in this viewpoint is that the living organism is to be regarded as a multiphasic system in which the thermodynamic activity or "escaping tendency" of cholesterol tends to be equal in each of the several compartments and phases. This system is undoubtedly not in a condition of equilibrium with regard to cholesterol, because of the continual removal and addition of cholesterol at various sites; yet such changes are followed by secondary changes in the other compartments which tend to restore the equality of the cholesterol activity throughout. These secondary changes may include transfer of cholesterol from one compartment to another, as well as alterations in the rate of cholesterol synthesis.

As specific examples of the operation of the above process, let us consider the regulation of the rate of cholesterol synthesis in the rat. In this animal it appears proper to regard the serum and liver cholesterol as freely exchangeable.[25] Isotopically labeled material passes between these two compartments very readily.[24] The concentrations on the two sides of the partition, however, are quite different. In our experience, the concentration in the serum is about 70 mg. per 100 ml., and in the liver about 250 mg. per 100 grams wet weight.[20,23,24] Evidently the state of combination of cholesterol in the liver is such as to reduce greatly its escaping tendency. If cholesterol is added to the diet, most of it is promptly deposited in the liver. The serum cholesterol rises only if the feeding is continued to the point that the animal's defenses are overwhelmed. Coincident with the rise in the concentration in the liver, the rate of synthesis in that organ, as measured by various isotopic techniques, decreases dramatically.

If one ligates the rat's common bile duct, the serum cholesterol rises to a very high level, with little or no change in concentration in the liver. Although definite proof is lacking, we have explained this finding on the basis that the accumulation of bile acids has a stabilizing effect on the serum cholesterol. The activity of the latter is thereby reduced, and cholesterol is rapidly poured out of the liver to make up the deficit. This hypothesis, together with the apparent large and sudden increase in total body cholesterol, led us to predict that the rate of synthesis in the liver would be elevated. We confirmed this prediction by *in vivo* and *in vitro* techniques, with the use of both carbon-labeled acetate and tritium-labeled water. Similar studies on the rate of synthesis following intravenous administration of the surface-active agent Triton WR-1339 led to similar results.[24] This agent, as shown by others,[26,27] produces a rise in serum cholesterol which in our hands proved even more striking than that caused by biliary obstruction. The rise is sometimes as much as ten-fold in a twenty-four hour period. It is not surprising that the rate of synthesis should also be elevated.

We have previously emphasized the reciprocal relationship which exists between the rate of cholesterol synthesis after cholesterol feeding and the concentration of cholesterol existing in the liver. It occurred to us that if the notion is correct that it is the activity rather than the concentration which is the important factor in regulating the rate of synthesis, then it might be possible to upset the reciprocal relationship by a judicious combination of cholesterol feeding and ligation of the common bile duct. Such proved to be the case. Following biliary obstruction in the cholesterol-fed rat, the cholesterol accumulated in the liver is rapidly poured into the blood. At early time intervals after the ligation, however, the rate of synthesis is elevated above that, even, of normal controls, while the concentration in the liver has not yet fallen to normal. This finding is to be expected if we assume that the activity of cholesterol in the liver as well as in the serum is decreased by the accumulation of bile acids. The detergent Triton WR-1339 in combination with cholesterol feeding produced a somewhat different effect. As in biliary obstruction, the cholesterol accumulated in the liver was rapidly dissipated; but we were never able to attain conditions which led to a rate of synthesis greater than normal while the liver concentration remained elevated. This result would be expected if the effect of the foreign detergent is exerted entirely extracellularly, in contrast to that of the bile acids, which themselves originate within the liver cells.

The concept of the regulation of cholesterol metabolism presented above represents a thermodynamic approach, and includes nothing about the actual mechanism responsible for the control of cholesterol synthesis. Some tentative ideas about the latter may also be drawn from the data available. One thinks first of the possibility that the enzyme responsible for the final step in the synthesis may be inhibited competitively by the product. In Figure 1, a calculated curve based on this possibility is superimposed on our data for the rate of cholesterol synthesis in liver slices as a function of cholesterol concentration. The agreement can be made to appear fairly good because of the large number of constants which remain adjustable for lack of experimental data. This agreement is not to be taken as evidence in favor of control by product inhibition. It is merely indicative of the fact that that mechanism cannot be eliminated on the basis of information now at hand.

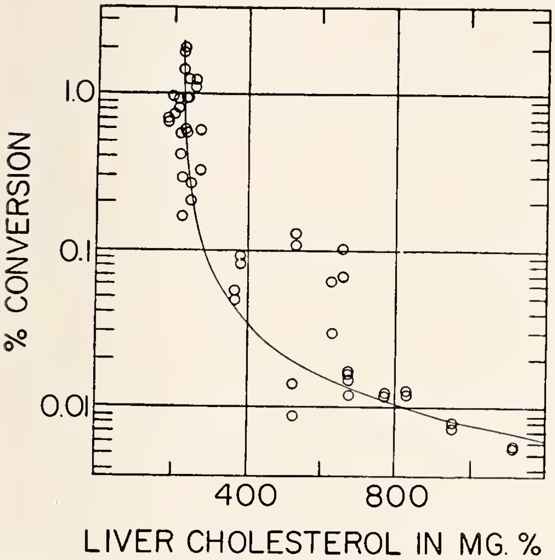

Fig. 1. Ability of liver slices to convert acetate to cholesterol, as a function of the concentration of cholesterol in the liver. The ordinate is the percentage of the radioactive acetate added which was converted to cholesterol in a 3-hour period. The curve was plotted from the equation for competitive inhibition,

$$v = \frac{V \frac{K_i}{K_m} S}{K_i + \frac{K_i}{K_m} S + I}$$

where v = velocity of the reaction, V = maximum velocity (enzyme saturated with substrate), S = substrate concentration, I = inhibitor concentration, K_m = equilibrium constant for the combination of substrate with enzyme, and K_i = equilibrium constant for the combination of inhibitor with enzyme. It was assumed that below a concentration of 222 mg./100 gm., all of the cholesterol is tightly bound and incapable of acting as an inhibitor. The following values were assigned to the components of the equation: V = 2.20 per cent, S = 1 mg./100 gm. (molecular weight of substrate assumed to be the same as that of cholesterol), K_m = 1 mg. per cent, K_i = 2.75 mg. per cent.

This figure (without the superimposed curve) was taken from reference 20, and I wish to thank the Journal of Biological Chemistry for permission to reproduce it.

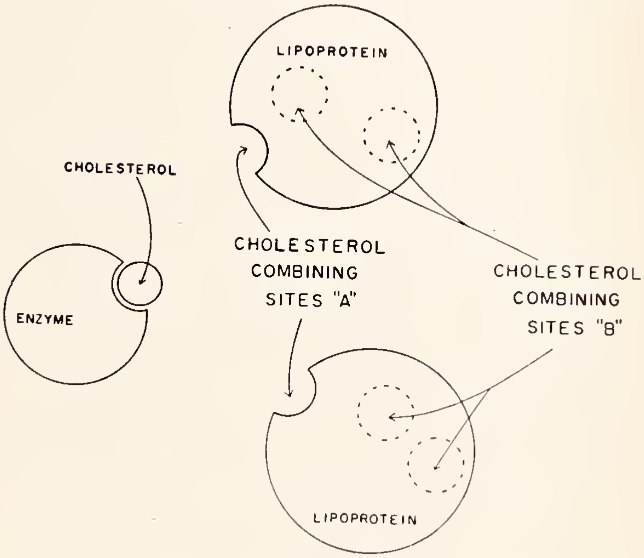
LIPOPROTEIN
CHOLESTEROL
ENZYME
CHOLESTEROL COMBINING SITES "A"
CHOLESTEROL COMBINING SITES "B"
LIPOPROTEIN

Fig. 2. Suggested scheme for the regulation of cholesterol synthesis. If sites "A," near the surface of the lipoprotein, are unoccupied, collisions of the lipoprotein with the enzyme will usually result in removal of the cholesterol from the enzyme and will permit the latter to begin the synthesis of a new molecule. Sites "B" are to be thought of as more numerous but less readily accessible. After sites "A" are filled, collisions become progressively less effective in removing cholesterol. If the "A" sites and most of the "B" sites as well are filled as a result of cholesterol feeding, collisions are rarely effective and synthesis drops to a very low level.

It seems improper to me to think of cholesterol synthesis in the terms used in the preceding paragraph, because of the extremely low water solubility of cholesterol unassociated with protein and phospholipid. A more promising viewpoint is to assume that a cholesterol molecule, once it is formed on the surface of the enzyme, must remain there until it is removed by collision with a lipoprotein molecule capable of accepting it. The enzyme would remain unable to initiate the synthesis of another molecule until such an encounter has taken place. Lipoproteins presumably contain a number of sites which can be filled by cholesterol. A few of these are probably near the surface, so that they can be filled very readily in the event of a collision with an enzyme carrying a cholesterol molecule. Most of the sites are probably in the interior or "core" of the lipoprotein aggregate, and the likelihood of their being filled is small, even in the event of a collision. If the animal's store of cholesterol is small, most of the sites are empty, including those easily filled. Collisions with enzyme molecules carrying cholesterol usually result in removal of the cholesterol, and the rate of synthesis is very high. As the sites are progressively filled, the rate of synthesis falls. Presumably there is a spectrum of cholesterol sites, with respect to accessibility, and this spectrum accounts for the observed relation between cholesterol concentration and rate of synthesis. Figure 2 is an attempt to clarify this notion.

The effect of surface-active agents on cholesterol synthesis is also understandable from the viewpoint suggested above. Intravenous injection of these agents probably produces an enormous increase in the number of readily accessible sites for cholesterol. Finished product is removed from the enzyme with great efficiency, and the rate of synthesis rises.

It seems possible that mechanisms akin to those outlined here may be responsible for the homeostatic control of the rate of synthesis of many bodily constituents.

We are now in a position to reexamine the relation of cholesterol metabolism to atherosclerosis. The high blood cholesterol in man may be due to an unusually large number of combining sites for cholesterol in human plasma lipoprotein. The primary cause thus becomes a species variation in protein, a chemical entity well known to vary widely from species to species. These sites are kept filled, as previously described. To return to the thermodynamic viewpoint, the activity of cholesterol is maintained at about the same level in man as in other animals, but the concentration in the blood is higher nonetheless. This picture is in accord with the well known fact that tissue concentrations of cholesterol do not show the striking species differences seen in the blood.

The interpretation presented here is only one of several which are more or less compatible with the currently available data. The possibility must also be considered that the elevated concentration of cholesterol in human serum is related to deficiencies in the mechanisms for the transfer of lipid from one lipoprotein to another. According to this view, the steady-state concentrations of cholesterol in human serum and liver would be established at values further removed from true equilibrium than in the animals with low blood cholesterols. Another reasonable interpretation of the apparent depression in cholesterol synthesis with cholesterol feeding should also be considered. As mentioned above, it is still possible that this depression will prove to be an artefact of the experimental methods, related to endogenous dilution of cholesterol precursors. If further evidence does prove that the depression is real, the cause may be adaptive reduction in enzyme concentrations, rather than a direct effect of the elevated concentration of the product of the reactions.

One might suppose that if the activity of cholesterol is the same in human serum as in the blood of other animals, the tendency of cholesterol to deposit in the arterial wall would also be the same, despite the higher concentration. A hint concerning this point may be drawn from the work of Kellner, Correll, and Ladd.[28] They found that surface-active agents offered a measure of protection against atherosclerosis in the cholesterol-fed rabbit. They found, on the other hand, that atherosclerosis could be induced by prolonged administration of the surface-active agent, even without cholesterol feeding. Apparently processes occur within the arterial wall which result in the breakdown of lipoprotein molecules which in the blood are perfectly stable. Thus, the total concentration of cholesterol in the blood may be of importance equal to or perhaps greater than the stability of the complex in which the cholesterol is held.

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CORONARY HEART DISEASE

(Continued from Page 766)

the concentration of cholesterol, especially that in the beta lipoprotein fraction of the serum, and the development of atherosclerosis.

In any case, in the effect of the mode of life must be our hope for the future. I think we can be confident that this hope is well founded.

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Hypertension and Atherosclerosis

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NUMEROUS studies of the relationship between hypertension and atherosclerosis have been carried out. When all the evidence is considered, it would seem that arterial hypertension does not cause atherosclerosis but will definitely accelerate it if there exists a natural underlying tendency for the process to occur. I will attempt to bring out the evidence which leads to this conclusion. Much of it is to be found in the study of atherosclerosis in the human species.

In the first place, atherosclerosis in man tends to localize in areas under increased hydrostatic pressure. As the pulse wave travels toward the bifurcation of the aorta it becomes steeper and the systolic peak is higher. The erect posture adds the weight of a column of blood to the hydrostatic pressure in the lower extremeties. This increase in pressure in the arteries of the lower part of the body is related to a much greater incidence of severe atherosclerosis in these lower arteries, necessitating the amputation of countless legs for atherosclerotic gangrene while the sacrifice of an arm for this reason is a medical rarity.[1] While atheromata are frequent in the systemic arteries, they are normally almost never found in the pulmonary artery. This protection of the pulmonary artery from atherosclerosis is due to the low pressure normally found within it. However, in patients with pulmonary hypertension, severe typical atherosclerosis may develop in the pulmonary artery. This experiment of nature is striking evidence that increased hydrostatic pressure accelerates atherosclerosis. Atheromata are also found at other loci of increased lateral pressure. For example, the knob of the transverse aorta is the target for each systolic jet from the left ventricle. At this area there is often considerable atherosclerosis, much more than is usually seen in the ascending aorta. Similarly the part of the aorta just above a coarctation often shows advanced atherosclerosis. The ventricular side of the mitral valve may show atherosclerosis. The point in the right ventricular endocardium opposite an interventricular septal defect may have an atheroma. The spot in the pulmonary artery opposite a patent ductus arteriosus may be the seat of an atheroma. The same process may be seen in a vein at the point opposite an arteriovenous fistula. Even phlebosclerosis occurs in patients with long standing elevations of peripheral venous pressure.[2] Wilens has pointed out that even cerebral atherosclerosis fits into this pattern.[1]

The second important consideration in this problem is the relationship of arterial hypertension in man to atherosclerosis. In 90 per cent of cases this means essential hypertension. At the outset it should be pointed out that there are many cases of essential hypertension without significant atherosclerosis and that there are many cases of atherosclerosis with normal arterial pressure. However, there is a considerable body of evidence that atherosclerosis is accelerated in the presence of essential hypertension.

Rosenthal studied the fat content of the aorta and found that it closely paralleled the severity of atherosclerosis. He found that patients with essential hypertension had a definitely increased fat content of the aorta for a given age compared to the general population studied.[3] Faber has made the same observation for the cholesterol content of the aorta in hypertensive patients.[4] These studies indicate an increased amount of aortic atherosclerosis in essential hypertension.

It would also appear that accelerated atherosclerosis of the renal arteries is associated with essential hypertension. Blackman found that 86 per cent of hypertensive patients had atherosclerosis of the main renal arteries, while only 10 per cent of the normotensive patients did.[5] Lisa, Eckstein, and Soloman found atheromata

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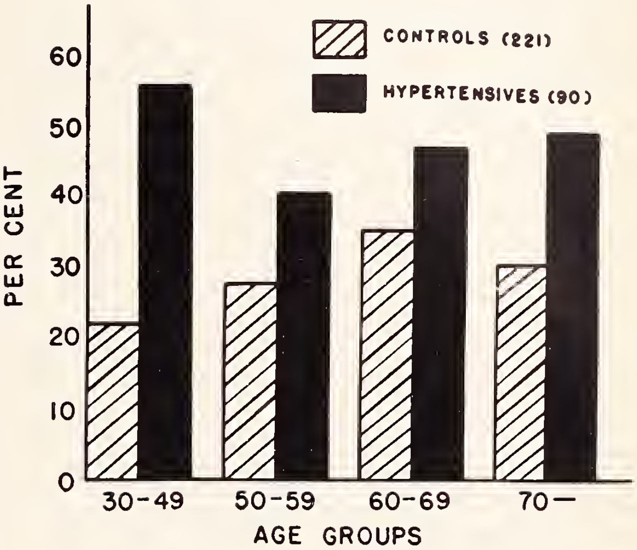
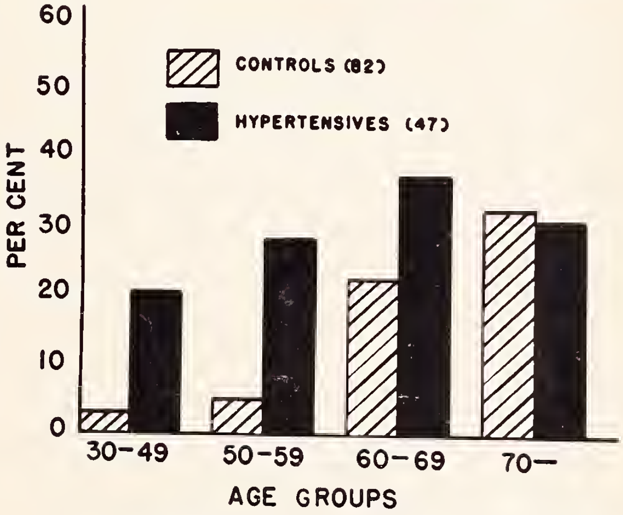
of the main renal arteries in 60 per cent of their hypertensive series and in only 43 per cent of their non-hypertensive group.[6] Richardson examined 145 consecutive autopsies; 78 per cent of the cases of essential hypertension had atheromata in the main renal arteries. Only 7 per cent of the normotensive controls had any atheromata at all in the main renal arteries, and only 3 per cent of the normotensive group had atheromatous involvement as severe as the least severe case in the hypertensive group.[7] These atheromata in the main renal artery do rarely cause hypertension in man similar to a Goldblatt experiment. In such instances there should be no arteriolosclerosis in the involved kidney. This is usually not the case in autopsy experience. In the vast majority of hypertensive patients the essential hypertension comes first and then accelerates the formation of atheromata in the main renal arteries. Usually these atheromata, even those of large size, do not significantly obstruct the lumen of the renal artery. Occasionally the atheroma is severe enough to obstruct the lumen, and in these cases there may be an aggravation of the pre-existing hypertension. However, it seems clear that hypertension does increase the frequency of atherosclerosis of the main renal arteries.

The most important problem is the influence of hypertension on coronary atherosclerosis. Bell and Clawson found severe sclerosis of the left coronary artery in 38 per cent of hypertensive patients, while only 4 per cent of a comparable normotensive control group showed similar lesions.[8]

Davis and Klainer made another well controlled autopsy study of essential hypertension and coronary sclerosis. They examined consecutive cases, utilized only blood pressure readings acquired prior to the terminal illness, and considered the factors of age and sex. They classified patients as hypertensive if the systolic pressure was consistently above 150 mm. Hg and the diastolic pressure was consistently above 90 mm. Hg. Forty-five per cent of all the hypertensive patients had severe coronary disease, compared to 27 per cent in the normotensive control patients. Figure 1 shows their comparison of hypertensives and normotensives in both sexes. In almost all age groups, the incidence of marked coronary disease is greater in the patients with hypertension. This is particularly so for patients under fifty. Hypertensive men under fifty had severe coronary disease two and one half times more frequently than their normotensive counterparts. The influence of hypertension, however, was the most striking in women under sixty. Hypertensive women had marked coronary disease six times more frequently than the normotensive women. The incidence of marked coronary disease in hypertensive men did not increase with advancing age in this series, while the usual increase with age was seen in the normotensive group. Even though the incidence of advanced sclerosis was considerable in hypertensive women, they had about the same amount of severe coronary disease seen in normotensive men. Davis and Klainer also noted that severe elevations of blood



—Marked coronary disease in males.

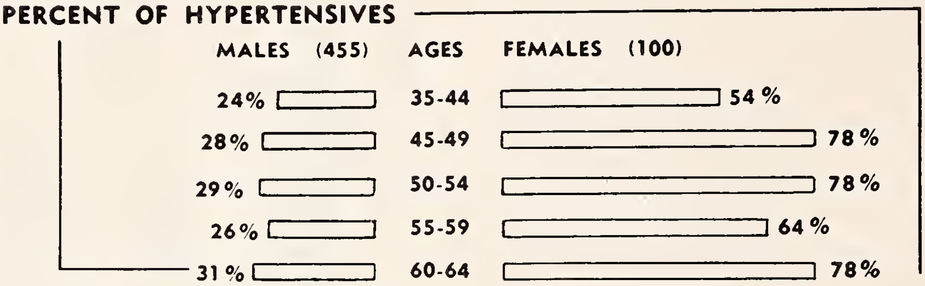


—Marked coronary disease in females.

Fig. 1.

pressure were not associated with more coronary disease than modest elevations of pressure.[9]

In 1953, Master completed a large study relating hypertension to acute coronary occlusion.[10] This study is very pertinent to the coronary sclerosis problem since nearly all cases of coronary occlusion are related to coronary atherosclerosis. In this study the general population was grouped as to sex and age, and a pressure level two standard deviations above the mean for a given group constituted hypertension. Since the average blood pressure of the population tends to rise with age, his lower limits of hypertension correspondingly rise with age. With these criteria, 2½ per cent of any group in the general population will have hypertension. A group of 600 cases with coronary occlusion was studied; 27 per cent of all the men with coronary occlusion had hypertension by these criteria, compared to 2½ per cent in the general male population. The per cent of hypertensives in the various age groups is shown in Figure 2. The incidence of hypertension in this male group is eleven times greater than the incidence in men of the general population. Hence hypertension definitely seems to predispose to coronary occlusion in men of all age groups. A man with hypertension is thirteen times more likely to have a myocardial infarction than a normotensive man. In women, however, the influence of hypertension in promoting coronary occlusion is even greater. Seventy-one per cent of all the women with coronary occlusion had hypertension, compared to 2½ per cent hypertension in the general female population (Fig. 2). It can be estimated from these data that a woman with hypertension is eighty-five times more likely to have a coronary occlusion than a woman with normal blood pressure.



—Frequency of hypertension in 455 cases of coronary occlusion (blood pressure readings taken before the onset of coronary occlusion).

Fig. 2.

In patients with coronary occlusion, hypertension did not seem to affect the recovery from the acute attack.

Yater et al, in analyzing myocardial infarction in army men under forty, also found hypertension a predisposing factor. In the group with coronary occlusion, 19 per cent had a diastolic pressure above 90 mm. Hg, while in the control group only 3.8 per cent had a diastolic pressure above 90. Hypertension was thus five times more frequent in the coronary cases than in the controls.[11] And these figures would be even more striking in a general unselected population, since men with obvious hypertension were excluded from the army in the first place.

When Gofman et al considered the diastolic pressure as well as the lipoprotein fractions, they improved their score in predicting the presence of coronary disease.[12]

The analyses of Hunter suggest that even a "normal" blood pressure predisposes to an excessive mortality when compared to blood pressures 20 mm. lower than normal.[13]

Experiments in various animals also throw light on the relation of hypertension to atherosclerosis.

Bronte-Stewart and Heptinstall produced renal hypertension in rabbits and then fed these rabbits cholesterol. After seventy-two days of cholesterol feeding, the rabbits with high blood pressure had an average of 47 per cent of the aorta involved with atheromata, while the control rabbits with normal blood pressure had only 11 per cent of the aorta involved. The rabbits in both groups fed cholesterol had comparable elevations of serum cholesterol. Hypertension did not lead to atherosclerosis if the rabbits were not fed cholesterol. The rabbits with mild hypertension had as much aortic atherosclerosis as the rabbits with severe hypertension. Even short periods of hypertension, such as four to five weeks markedly accelerated atheroma formation. Even in rabbits without kidney operations, increased lability of the blood pressure was associated with an increased severity of atherosclerosis.[14] Wilens noted that cholesterol fed rabbits forced to sit upright for five hours a day developed aortic atheromatosis sooner and much more extensively than control rabbits. This forced sitting produces a 40 per cent rise in arterial pressure, and the elevated pressure promptly reverts to normal when the rabbit is returned to his regular posture.[15] Evidently intermittent periods of mildly elevated blood pressure will accelerate atherosclerosis in rabbits.

The influence of DCA hypertension has also been demonstrated in cholesterol-induced atherosclerosis in chicks. Stamler, Pick and Katz found that 45 per cent of chicks receiving DCA, salt, and cholesterol had lesions in the thoracic aorta, while only 17 per cent of chicks receiving just the salt and cholesterol had thoracic atheromata.[16]

Wakerlin et al have also demonstrated an acceleration of atherosclerosis in hypertensive, hypothyroid dogs fed cholesterol. These dogs with bilateral renal artery clamps developed atherosclerosis sooner and to a much more severe degree than the normotensive control dogs.[17]

Considering all the foregoing evidence, there is little doubt that arterial hypertension accelerates atherosclerosis whenever the underlying conditions for forming atheromata are present. The mechanism of this acceleration is obscure. The most likely explanation is that the increased intraluminal pressure tends to hasten the filtration of serum lipids through the endothelial layer into the intimal space. It is also possible that the increased pressure creates an added vibration or produces some subtle mechanical damage to the artery wall with subsequent hastening of atheroma formation. More information will be needed to solve this problem.

Admitting the accelerated atherosclerosis in hypertension, what is its clinical importance? Patients with benign hypertension are endangered largely by the threat of heart failure, myocardial infarction, angina pectoris, and cerebro-vascular accidents. Myocardial infarction and angina are obviously related to coronary atherosclerosis. Moreover, angina appears in hypertensives with lesser degrees of coronary narrowing than are seen in normotensive patients with angina.[18] Moreover, congestive heart failure in hypertensive patients is often related to coronary sclerosis and thrombosis. Averbuck found that severe coronary atherosclerosis was present at necropsy in 85 per cent of patients with essential hypertension who had heart failure, but in only 10 per cent of hypertensive individuals without cardiac failure.[19] Coronary involvement, even of relatively slight degree, will diminish the very large coronary flows necessary in the hypertrophied heart of hypertensive patients. This reduction in flow retards maximum hypertrophy and promotes fibrous replacement of myocardial fibers, with the ultimate development of myocardial insufficiency. And of course gross myocardial infarction greatly hastens the onset of congestive failure. Without the accelerated atherosclerosis, essential hypertension would become a relatively benign disease.

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Hormonal Factors in the Pathogenesis of Atherosclerosis

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THE following discussion is focused upon effects of hormones on concentration and distribution of lipids in plasma. Its relevance to the pathogenesis of atherosclerosis is based on assumption that lipid composition of plasma exerts an influence on the rate of development of atherosclerosis and its complications.

Most of the discussion is centered about observations made by Miss Ella Russ, Dr. Howard Eder, and myself at the New York Hospital-Cornell University Medical Center. Chief emphasis is placed on the relative concentration of alpha and beta lipoproteins and the manner in which cholesterol is distributed between them. This relationship is stressed because previous experience in study of many conditions of varying degree of atherogenicity has indicated that it correlates more accurately than others with tendency to atherosclerosis.[1,2,3] For convenience of presentation this relationship is usually expressed as the percentage of total cholesterol combined with alpha lipoproteins.

Action of Gonadal Hormones

The most convincing evidence of hormonal influence on lipid composition of plasma has been obtained by study of action of estrogens and androgens.

Administration of estrogens to men who have survived one or more attacks of myocardial infarction accomplishes several predictable actions, with the result that the gross abnormalities in lipids usually found in these atherosclerotic men are almost completely corrected.[4] The most constant effect is an increase in concentration of alpha lipoproteins and a corresponding decrease in concentration of beta lipoproteins. Usually there is also a considerable reduction in concentration of cholesterol and almost always a sharp reduction in cholesterol-phospholipid ratios of plasma and of beta lipoproteins. Direction and extent of these changes are indicated in Table I.

TABLE I. EFFECT OF ESTROGENS ON CONCENTRATION AND DISTRIBUTION OF LIPIDS

Conditions	Total Cholesterol Mg. per 100 Ml.	Cholesterol in Form of Alpha Lipoproteins		Cholesterol in Form of Beta Lipoproteins Mg. per 100 Ml.	Cholesterol-Phospholipid Ratio by Weight	
		Mg. per 100 Ml.	% of Total		Plasma	Beta Lipoproteins
Before estrogen	282	32	11.3	250	1.05	1.32
After 9 weeks of estrogen	210	57	27.1	153	0.72	0.97
2 weeks after withdrawal	285	53	18.5	220		

More recent observations in our laboratory have shown that in women who have suffered myocardial infarction action of estrogen is similar in direction and extent to that observed in men. In the women a daily dose of 1.0 mg. of estinyl was maintained continuously with addition of 100 mg. of pranone for each of four days at the end of each month. Effects of treatment are indicated in Table II.

In cases of familial hypercholesterolemia and xanthomatosis with their extreme atherogenic tendency, effects of estrogens are similar in direction but seldom are sufficient to accomplish complete restoration of normal conditions. In the atherogenic state of nephrosis fragmentary and insufficient observations have offered little

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hope that estrogens will alter beneficially the extreme lipid abnormalities.[4]

In hypothyroidism which has often been regarded as atherogenic there is some evidence that estrogen may alter or correct abnormalities in lipid composition of plasma. Observations recorded in Table III were made on plasma of a forty-eight-year-old cretin whose basal metabolic rate during period of treatment with estrogen was -26.

TABLE II. EFFECT OF ESTROGEN THERAPY IN MYOCARDIAL INFARCTION OF WOMEN

Patient	Age	Conditions	Total Cholesterol Mg./100 Ml.	Percentage of Total Cholesterol in Alpha Lipoprotein	Cholesterol in Beta Lipoprotein Mg./100 Ml.	Cholesterol-Phospholipid Ratio	
						Plasma	Beta Lipoprotein
Bie	46	Before estrogen	342	12.4	297	1.03	1.26
		After 84 days 10,000 r.u. estinyl o.d.	211	36.6	132	0.67	0.94
Cha	59	Before estrogen	368	20.3	288	0.93	1.29
		After 41 days 10,000 r.u. estinyl o.d.	313	29.8	213	0.82	1.27
Coh	44	Before estrogen	358	10.2	316	1.19	1.34
		After 54 days 10,000 r.u. estinyl o.d.	313	22.7	234	0.91	1.33
Wol	48	Before estrogen	411	8.5	369		
		After 5 months 10,000 r.u. estinyl o.d.	337	19.8	265		
		2 months after withdrawal	441	9.8	362		

TABLE III. EFFECT OF ESTROGEN THERAPY IN CRETINISM

Name	Age	Conditions	Total Cholesterol Mg./100 Ml.	Percentage of Total Cholesterol in Alpha Lipoprotein	Cholesterol in Beta Lipoprotein Mg./100 Ml.	Cholesterol-Phospholipid Ratio	
						Plasma	Beta Lipoprotein
Laz	58	Before estrogen	374	11.3	330	1.10	1.40
		After 22 days 10,000 r.u. estinyl o.d.	301	21.3	232	0.91	1.20
		2 months after withdrawal	391	10.8	343	1.17	1.35
		After 27 days 10,000 r.u. estinyl o.d.	260	25.4	189	0.68	0.97

Effects of androgens on the lipid composition of plasma are in general the exact opposite to those following use of estrogens.[4] They exaggerate chemical pathology of survivors of myocardial infarction and produce abnormalities in plasma of individuals whose protein-lipid patterns previously have been normal. A constant effect of administration of methyl testosterone is relative increase in concentration of beta lipoproteins and a corresponding decrease in concentration of alpha lipoproteins. In some individuals there is also augmentation of concentration of cholesterol in unfractionated plasma with increase in cholesterol-phospholipid ratios of plasma and of beta lipoproteins. The changes in relative concentrations of alpha and beta lipoproteins expressed as percentage of cholesterol in alpha lipoproteins is presented in Table IV.

TABLE IV. EFFECTS OF METHYL TESTOSTERONE ON CONCENTRATION AND DISTRIBUTION OF LIPIDS

Conditions	Percentage of Total Cholesterol in Alpha Lipoprotein
Before testosterone	22.8
After testosterone	14.4
After withdrawal of testosterone	22.1

When estrogens and androgens are given simultaneously, effects of estrogens are modified or obliterated.[4] An example illustrating the extent of neutralization accomplished by methyl testosterone in daily doses of 50 mg. may be seen in Table V. Similar neutralization has been observed from use of 5 to 10 mg. methyl testosterone each day.

Observations on use of gonadal hormones show that protein-lipid patterns of plasma can be radically modified. They seem to have some relevance to the pathogenesis of atherosclerosis since they indicate a chemical reason for the relative immunity of young women to the complications of atherosclerosis and the greater susceptibility of young men to these accidents. They suggest that action of estrogenic substances in persons exhibiting the protein-lipid abnormalities of the type characteristic of atherogenesis might be beneficial and that use of methyl testosterone in such patients might be harmful.

TABLE V. INFLUENCE OF COMBINED ESTROGEN AND METHYL TESTOSTERONE

Conditions	Total Cholesterol Mg./100 Ml.	Percentage of Cholesterol in Form of Alpha Lipoproteins	Cholesterol in Form of Beta Lipoproteins Mg./100 Ml.
Before use of hormone	202	9.7	171
After 5 days 10,000 r.u. estinyl o.d.	173	29.6	120
After 69 days 5,000 r.u. estinyl o.d.	194	33.1	130
After 56 days 2,500 r.u. estinyl o.d.	217	31.2	148
After 28 days combined dosage 2,500 r.u. estinyl & 50 mg. methyl testosterone o.d.	351	3.2	338
After 50 days 2,500 r.u. estinyl o.d. without methyl testosterone	226	27.6	161

Action of Thyroid Hormones

Effects of thyroid hormones on concentration and distribution of lipoproteins and on pathogenesis of atherosclerosis are of special interest because of the well-known influence of thyroid activity on the concentration of cholesterol and phospholipids.[5] The question may be studied by examination of lipid composition of plasma in states of hyper- and hypothyroidism or during alteration of pathologic conditions by therapy. Table VI contains observations on plasma of a few patients with Graves' disease.

In this small group of patients ranging in age from thirty-four to sixty-nine, the low normal concentration of cholesterol is apparent. The average of 145 mg. per 100 ml. may be compared with a normal of 190 for young men and women, and of 245 for presumably normal older persons. Emphasis on this aspect is unnecessary, however, since there is in the literature abundant evidence that hyperthyroidism tends to reduce the level of plasma cholesterol. Attention is called especially to the percentages of total cholesterol in alpha lipoproteins where the values reveal no trend. It is perhaps noteworthy that two of the patients, one of whom is only thirty-six years of age, show distribution of cholesterol possibly indicative of an atherogenic tendency.

TABLE VI. CONCENTRATION AND DISTRIBUTION OF CHOLESTEROL IN THYROTOXICOSIS

Sex	Age	Total Cholesterol Mg./100 Ml.	Percentage Total Cholesterol in Alpha Lipoproteins
M	34	158	21.1
M	36	168	16.0
M	53	125	38.0
M	48	153	27.8
M	59	165	32.3
M	63	135	17.9
M	69	111	34.9
F	66	150	31.1
F	44	146	27.5
F	54	142	24.4

In Table VII results of treatment in Graves' disease and myxedema are presented. In each of the patients with Graves' disease treatment with antithyroid drugs or radioactive iodin resulted in marked increase in the level of plasma cholesterol. In two of them there was also a considerable reduction in the percentage of total cholesterol in the form of alpha lipoproteins. In the other three the percentage in alpha lipoprotein was actually increased. Again, no trend is apparent. In the cases of myxedema there was in each instance with treatment an increase in basal metabolic rate or a reduction in concentration of plasma cholesterol or both. On the other hand, the changes in the percentage of total cholesterol in the form of alpha lipoprotein were trivial and not always in the same direction. It is also of some interest that those patients whose percentage in alpha lipoprotein was initially low tended to maintain a low presumably atherogenic level after treatment.

Failure to demonstrate effect of thyroid hormones on distribution of cholesterol between alpha and beta lipoprotein is also apparent in the observation on the action of intravenously-injected tri-iodo-thyronine which is presented in Table VIII.

Although both basal metabolic rate and cholesterol concentrations were considerably modified by the action of the hormones, changes in the percentage of total cholesterol in alpha lipoproteins were trivial.

TABLE VII. EFFECT OF THYROID ACTIVITY ON CONCENTRATION AND DISTRIBUTION OF LIPIDS

Diagnosis	Sex	Age	Conditions	Basal Metabolic Rate	Cholesterol Mg./100 Ml.	Percentage of Total Cholesterol Alpha Lipoprotein
Graves' Disease	M	34	Before treatment	+47	158	21.1
			After 5 months	+39	375	10.6
Graves' Disease	M	69	Before treatment	+36	111	34.9
			After 3 months	−24	260	23.7
Graves' Disease	M	36	Before treatment	+23	168	16.0
			After 3 weeks	−10	281	17.9
Graves' Disease	M	48	Before treatment	+22	153	27.8
			After 2½ months		264	31.4
Graves' Disease	F	66	Before treatment	+16	150	31.1
			After 2 weeks	+ 1	218	36.9
Myxedema	M	60	Before treatment	−31	391	12.6
			After 6 weeks	−18	191	16.6
Myxedema	M	50	Before treatment	−35	346	12.8
			After 6 months	−24	360	11.3
Myxedema	F	37	Before treatment	−41	380	13.3
			After 3 months	−19	300	11.5
Myxedema	F	46	Before treatment	−26	272	23.7
			After 6 months	+ 9	133	21.6

These observations are of some interest in relation to clinical thought concerning the relationship of thyroid activity to the pathogenesis of atherosclerosis. Although there is abundant evidence that thyrotoxic individuals tend to have relatively low concentrations of cholesterol, there is no anatomic evidence that such patients have less than usual tendency to atheromatous deposits in arteries. Myxedema has often been listed as one of the diseases in which atherosclerosis develops extensively and prematurely. This impression has received some support from an apparently high incidence of myocardial infarction in hypothyroid patients and because of a long-prevalent idea that patients with higher than normal concentrations of cholesterol have a greater than usual tendency to develop coronary disease. There is no sound anatomic evidence that myxedematous patients have more atheromatous deposit than others of the same age in the general population. Indeed, Blumgart's study[6] of patients subjected to complete thyroidectomy revealed no greater than ordinary atheromatous deposits in large vessels.

Action of Cortisone

Extensive observations by Adlersberg and his associates[7,8] have indicated that cortisone administration in a variety of clinical states is followed by increased concentrations of serum cholesterol. Although this is not invariable it has averaged about 20 per cent. In some patients the augmentation of serum cholesterol became apparent in two weeks, while in others it required two or three months. The effect was both greater and prompter in patients with initially high serum cholesterol.

TABLE VIII. EFFECT OF TRI-IODO-THYRONINE DISTRIBUTION AND CONCENTRATION OF LIPIDS

Day	Basal Metabolic Rate	Cholesterol Mg./100 Ml.	Percentage Total Cholesterol in Alpha Lipoproteins
May 11	−38	456	6.9
May 12	−38		
10 a.m. 1.0 mg. Tri-iodo-thyronine i.v.			
May 13	−14	388	9.2
May 18	−16	338	8.4
May 27	−35		
June 1		357	12.8

Our own studies have been limited and are mentioned only because the Cohn method of fractionation gives some insight concerning the concentration of alpha and beta lipoproteins and the distribution of cholesterol between them. In Table IX observations on six normal men and four normal women are presented. Blood was drawn for microfractionation before treatment was started; again, after several days' cortisone administration.

Changes are not impressive. No increase in concentration of cholesterol is apparent. Slight increase in the percentage of cholesterol in alpha lipoproteins, notable in six of ten cases, does not obviously depend on size of dose of cortisone.

Some observations on use of cortisone in pathologic states are presented in Table X. They have been selected as illustrative of the variability of results and of difficulty in evaluating hormonal effects in the changing background of active disease. In the girl with asthma, increased cholesterol concentration apparent after seven days and persistent at the end of two weeks of treatment is accompanied by significant increase in percentage of cholesterol in alpha lipoprotein. A menopausal, dystrophic woman of fifty-five exhibits after ten days a sharp reduction in cholesterol concentration with slight change in distribution of cholesterol. The young woman with nephrotic syndrome shows progressive increase in cholesterol concentration with no considerable change in distribution of cholesterol. The nephrotic young man on 500 mg. of cortisone for five days shows no significant change in concentration or distribution of cholesterol. Later, a dose of 400 mg. three times each week is followed after fifty days by complete chemical remission. In each of two cases of hypopituitarism in elderly men, cholesterol concentration increases without significant change in distribution.

TABLE IX. EFFECT OF CORTISONE NORMAL MEN AND WOMEN

Subject	Sex	Age	Daily Dose Mg.	Conditions	Cholesterol Mg./100 Ml.	Percentage Total Cholesterol in Alpha Lipoproteins
Sc.	M	25	25	Before	206	17.4
				After 5 days	208	16.8
Eh.	M	40	50	Before	205	21.2
				After 5 days	184	28.2
Ho.	M	28	100	Before	249	15.7
				After 5 days	257	17.8
Ed.	M	37	100	Before	178	18.3
				After 7 days	188	24.4
Ba.	M	64	100	Before	233	26.2
				After 7 days	211	36.3
Li.	M	30	150	Before	238	22.4
				After 5 days	212	20.7
Ca.	F	40	100	Before	201	21.2
				After 5 days	184	28.2
Vo.	F	28	100	Before	179	36.2
				After 5 days	185	34.4
Da.	F	28	100	Before	239	35.1
				After 5 days	228	34.2
Ru.	F	38	100	Before	156	24.5
				After 14 days	137	31.4
Average				Before	208	23.8
				After	199	27.2

TABLE X. EFFECT OF CORTISONE IN DISEASE

Sex	Age	Clinical State	Daily Dose Mg.	Duration of Treatment	Cholesterol Mg./100 Ml.		Per Cent Cholesterol Alpha Lipoprotein	
					Before	After	Before	After
F	13	Asthma		14 days	135	181	36.5	37.5
				28 days		194		43.2
F	55	Muscular Dystrophy	100	10 days	193	116	28.8	31.4
F	19	Nephrotic Syndrome	400	14 days	394	472	8.8	7.9
				34 days		537		7.9
				54 days		624		9.4
M	25	Nephrotic Syndrome	500	5 days	711	737	2.5	3.7
			400	10 days	398	513	8.8	9.8
				25 days		438		9.8
				44 days		294		13.4
				50 days		247		17.7
M	61	Hypopituitarism	100	14 days	194	233	21.3	23.5
M	64	Hypopituitarism	100	14 days	249	324	26.1	26.1

Variable and inconstant effects of cortisone are in sharp contrast to predictable changes observed following administration of gonadal hormones.

Relationship of adrenal cortical hormones to development of atherosclerosis depends on somewhat questionable circumstantial evidence. On basis of clinical impression, it has been assumed that Cushing's syndrome and adrenal hyperfunction exert an atherogenic influence. Similarly without statistical proof it has been stated that patients with Addison's disease have a less than usual degree of atherosclerosis. The most direct evidence concerning the influence of adrenal cortical hormones on the development of lipid deposit in vessels comes from a study of Etheridge and Hoch-Ligeti,[9] who found that children between the ages of one and ten who had received large amounts of cortisone or ACTH had a greater degree of lipid deposit in vessels than did untreated patients with similar disease. This interesting study was somewhat less than decisive because of the necessity of the use of other therapeutic agents in the patients under consideration. Observations by Adlersberg and his associates[10] and by Gordon, Kobernick, McMillan, and Duff[11] indicate that use of cortisone in rabbits fed large amounts of cholesterol actually diminishes the degree of atherosclerosis.

Summary

Our observations indicate that the use of gonadal hormones both estrogenic and androgenic exert predictable effect on relative concentration of alpha and beta lipoproteins and on distribution of cholesterol between them. Lack of thyroid hormone permits accumulation of cholesterol, while excess of the hormone leads to lowering of concentration of cholesterol in plasma. Neither lack nor excess of thyroid hormone causes predictable changes in the relative concentration of alpha and beta lipoproteins. Administration of cortisone is followed by variable changes in concentration of cholesterol. It is not established that use of cortisone leads to predictable alteration in relative concentrations of alpha and beta lipoproteins.

The changes observed following use of estrogens and androgens appear to have some possible correlation with pathogenesis of atherosclerosis. A similar relationship has not been demonstrated for thyroid or adrenal hormones.

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Coronary Atherogenesis—An Endocrine Problem?

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IT is well recognized that there is a remarkable sex difference in the incidence of coronary disease. A recent analysis of 1,000 consecutive cases attending the Edinburgh Royal Infirmary, all of whom had electrocardiographic evidence of myocardial infarction or ischemia, indicated that the clinical manifestations of coronary disease were nineteen times more common in men than in women under the age of thirty-five, and fifteen times more common under the age of forty (Table I). This striking sex difference in the incidence of the overt disease is probably not true for atheromatous lesions of the coronary arteries (Lober, 1953), and this suggests that women are protected in some way from the development of the clinical manifestations of coronary disease so long as their reproductive physiology is maintained. The increase in the incidence of the disease after a natural menopause, and prematurely following an artificial menopause, presumes that its development may be accelerated by changes in the hormonal equilibrium. The continuation of this sex difference into the seventh decade suggests that middle-aged men may be exposed more than middle-aged women to pathogenetic factors which favour rapid evolution of the disease.

In contrast, there has been no significant difference between the sexes in the increase in the morbidity and mortality rates either from coronary disease or from cerebral vascular disease during the last two decades (Fig. 1). The morbidity figures represent the number of patients diagnosed annually as suffering from coronary disease and from cerebral vascular disease in the medical wards of the Edinburgh Royal Infirmary; the total admissions to these wards for all causes averaged 6,000 annually. The mortality figures represent the number of subjects certified annually as dying from coronary disease and from cerebral vascular disease in Scotland; the total number of deaths from all causes averaged 64,000 annually. When the morbidity and mortality rates were each expressed as a percentage rise over the 1937 figures, there was no significant difference between them and thus for this analysis they have been combined. Since 1937 the incidence of coronary disease has increased five times compared with an increase in the incidence of cerebral vascular disease of less than twice. A similar increase in coronary disease has been reported from many civilized countries and, despite the better diagnosis, the aging population and the greater interest of the last twenty years, there has probably been a real rise in the incidence of the disease.

TABLE I. SEX AND AGE DISTRIBUTION OF 1000 CONSECUTIVE CASES OF CORONARY DISEASE ATTENDING EDINBURGH ROYAL INFIRMARY.

	Numbers of Cases		Ratio of Men to Women
	Men	Women	
Younger than 35	19	1	19:1
35-39	44	3	15:1
40-49	188	26	7:1
50-59	256	53	5:1
60-69	195	86	2:1
Older than 70	68	61	1:1

The distinction between the incidence of the clinical manifestations of coronary disease and those of cerebral vascular disease is interesting. Whether thrombosis or a disturbance of lipid metabolism is of greater importance in the genesis of atherosclerosis the selective involvement of the coronary arteries must be explained. The dynamic and morphological characteristics of the coronary circulation may alone be sufficiently different from those of the cerebral circulation to account for this selectivity, but it may also be significant that no abnormalities in the circulating lipids and lipoproteins have yet been

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established in cerebral vascular disease when it is dissociated, which is often difficult, from coronary disease. In contrast, clinical coronary disease is commonly associated with abnormal circulating lipids and lipoproteins. In Britain a controlled investigation of subjects with coronary disease has indicated that there is elevation of the plasma total cholesterol, of the plasma total-cholesterol : phospholipid ratio (the C/P ratio) and of the concentration of cholesterol attached to the β lipoprotein fraction, as determined by paper electrophoresis, and thus a low $\alpha:\beta$ lipoprotein ratio (Oliver and Boyd, 1953b, 1955b). These abnormalities are comparable to those observed in the United States (Gertler et al, 1950; Barr et al, 1951; Steiner et al, 1952; Rosenberg et al, 1954).

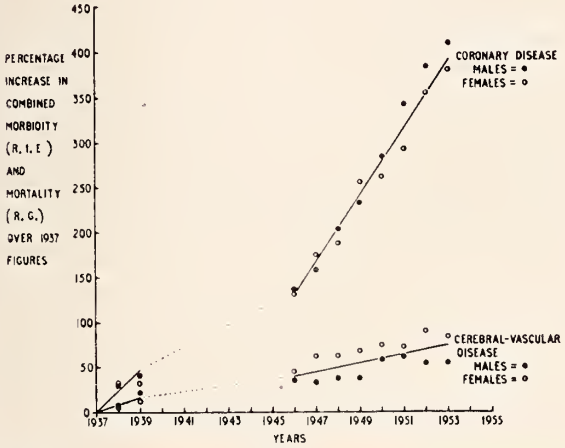

Fig. 1. The percentage increase in the combined morbidity and mortality rates for coronary disease and for cerebral vascular disease over the 1937 figures. The morbidity rates were obtained from hospital admissions (Royal Infirmary, Edinburgh) and the mortality rates from certification in Scotland (Registrar-General's Report). The dotted lines represent the war years during which accurate analyses of morbidity and mortality rates was not possible as part of the hospital was occupied by troops and the wartime population of Scotland was not static or comparable to the peacetime population.

The apparent protection of premenopausal women from clinical coronary disease and the particular association of coronary disease with abnormal circulating lipids and lipoproteins both emphasize the importance of hormonal and metabolic factors in the etiology of the disease. Study of the circulating lipids and lipoproteins during the menstrual cycle and pregnancy, the two physiological states peculiar to premenopausal women, has focussed attention on the endocrine aspects of coronary atherogenesis.

Menstrual Cycle

The plasma total cholesterol and the C/P ratio were both depressed at ovulation in each of twelve young nulliparous women whose circulating lipids were estimated during their menstrual cycles (Oliver and Boyd, 1953a). No such cyclical changes were observed in a group of twelve young men in whom analyses were made twice weekly for five weeks. In another group of twelve normal young women the distribution of cholesterol between the α and β lipoprotein fractions varied during the menstrual cycle (Oliver and Boyd, 1956b). There was less cholesterol attached to the β lipoprotein fraction, and thus a higher $\alpha:\beta$ ratio, at ovulation than at any other time during the cycle (Fig. 2). The relative concentrations of the ovarian hormones during the menstrual cycle are of interest in view of these cyclical changes. The secretion of the estrogenic hormones is maximal immediately before ovulation and remains high throughout the luteal phase (Smith and Smith, 1936; Brown, 1955); the peak coincides with the time when the plasma cholesterol, the C/P ratio and the concentration of cholesterol on the β lipoprotein fraction are all lowest. These values, therefore, may undergo regular variation in women during their reproductive years partly as a result of the endogenous secretion of estrogenic hormones at the midpoint and during the luteal phase of the cycle. These cyclical changes should be taken into consideration when making comparative studies of the circulating lipids and lipoproteins in young women, and the sex differences in lipid and lipoprotein con-

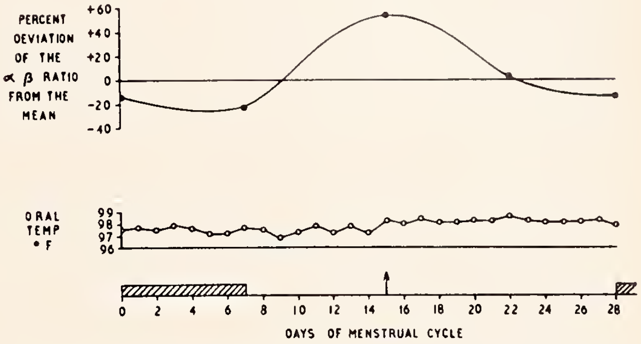

Fig. 2. The percentage deviation of the $\alpha:\beta$ lipoprotein ratio from its mean during the menstrual cycle of a typical healthy nulliparous woman, aged 30 years. Ovulation was determined by a rise at the mid-point of the cycle in the early morning oral temperature.

TABLE II. CHANGES IN THE CIRCULATING LIPIDS AND LIPOPROTEINS AFTER SIX MONTHS STUDY OF 40 MEN WITH CORONARY DISEASE.

	Plasma Total Cholesterol mg%.	Plasma C/P Ratio	Serum $\alpha:\beta$ Ratio
Ethinyl Estradiol (20 men)			
(a) Mean control value.	251	1.01	9:91
(b) 6 months later.	223	0.74	18:82
Inert Tablets (20 men)			
(a) Mean control value.	236	0.95	10:90
(b) 6 months later.	230	0.93	9:91

centrations, which have been reported in young subjects (Russ et al, 1951; Jones et al, 1951; Nikkila, 1953; Antonini, 1953), may partly result from the physiological variation within a menstrual cycle.

Pregnancy and the Puerperium

The circulating lipid and lipoprotein concentrations have been studied serially throughout pregnancy in twelve young healthy women (Oliver and Boyd, 1955a). As pregnancy advanced there was a gradual rise in the plasma cholesterol, the C/P ratio and the concentration of cholesterol on the β lipoprotein fraction, and by the thirty-second week the levels and distribution of the lipids and lipoproteins in each woman resembled those seen in the presence of clinical coronary disease; subsequently they regressed to return almost to the resting values by the fifth postpartum month. The development of a "coronary pattern" is fascinating particularly as clinical coronary disease is so rare during pregnancy. The relationship of multiple pregnancies to the subsequent development of clinical coronary disease is being studied, and preliminary observations from 500 postmenopausal women with coronary disease and 500 healthy women of comparable age indicates that the number of pregnancies in the former group is just significantly greater than in the healthy group. This investigation may help to assess the importance of episodes of hypercholesterolemia in the genesis of coronary atherosclerosis. The hypercholesterolemia of pregnancy is difficult to understand as there is an enormous increase in the concentration of circulating estrogens, increased thyroid and also increased adrenal activity—all of which are associated with a hypocholesterolemic response in men with coronary disease.

Estrogens

Correlation of the cyclical changes in the circulating lipids and lipoproteins during the menstrual cycle with endogenous estrogen secretion suggested that the administration of estrogens might influence the abnormal circulating lipids and lipoproteins of clinical coronary disease. A small pilot study indicated that a daily dose of 200 μg. or more of ethinyl estradiol caused depression of the plasma cholesterol, the C/P ratio and the concentration of cholesterol on the β lipoprotein fraction (Oliver and Boyd, 1954). Subsequently, ethinyl estradiol has been administered to more than 100 men with electrocardiographic evidence of myocardial infarction or ischemia and every man has shown some depression of these values (Oliver and Boyd, 1956a). Comparative studies of the effect of different strengths of ethinyl estradiol suggested that a dose larger than 200 μg. was not more effective in depressing the circulating lipids and might be a disadvantage in that it was more readily associated with feminizing side effects. Generally it is our experience that the higher the plasma cholesterol, the C/P ratio and the concentration of cholesterol attached to β lipoprotein fraction, the greater is the depressant action of ethinyl estradiol; it is upon the cholesterol increment present in coronary disease that ethinyl estradiol is most effective. Ethinyl estradiol was the only estrogen which caused highly significant depression of these values, although hexestrol and estradiol produced a similar trend. Estrone and estriol failed to produce any significant alteration in these values when administered in doses of equivalent estrogenic potency.

It is fundamental to determine whether control of the abnormal circulating lipid and lipoprotein patterns will inhibit or even cause regression of atherosclerotic lesions. Retardation of the atherosclerotic process once a myocardial infarct has developed may not necessarily prolong life. It is, therefore, essential to assess the effect of estrogens in terms of morbidity and mortality and thus a long-term study on men recovering from their first myocardial infarct was started in Edinburgh two years ago. Alternate cases have received either 200 μg. of ethinyl estradiol daily or identical inert tablets; there are now thirty-five men in each group. It is yet too early to make any significant observation on improvement or worsening of the functional capacity, on the morbidity rate or on the mortality rate of the two groups, but the change after six months in the lipids and lipoproteins in the first forty men is shown in Table II. Ethinyl estradiol at a dose of 200 µg. daily has been remarkably well tolerated; the majority of men developed gynecomastia and experienced depression or loss of libido, but these feminizing effects have never been severe enough to require withdrawal of the estrogen and are not regarded as major obstacles to the administration of ethinyl estradiol *at this dose level.*

Androgens

Methyl testosterone 50 mg. daily for ten days resulted in a significant increase in the concentration of cholesterol on the β lipoprotein fraction in six men who had had a myocardial infarct; there was no change in the effort tolerance of these men.

In an attempt to overcome some of the feminizing effects of estrogen therapy, methyl testosterone was administered simultaneously to two groups of hypercholesterolemic men with coronary disease. At the levels of 10 and 20 mg. daily, methyl testosterone caused partial inhibition of the depressant action of ethinyl estradiol on the circulating lipids and lipoproteins and failed to ameliorate any of the feminizing effects. The action of methyl testosterone on the circulating lipids and lipoproteins was most striking when ethinyl estradiol had been administered and its effect was being antagonized.

Our experiences with estrogens and androgens, which will shortly be reported in detail (Oliver and Boyd, 1956a), are very much in agreement with those recently published by Russ, Eder and Barr (1955).

Progestins

The intramuscular administration of pure progesterone 100 mg. daily for five days produced no significant change in the circulating lipids and lipoproteins of six hypercholesterolemic men with coronary disease.

A further attempt to eliminate the feminizing effects of estrogen therapy was made by the simultaneous administration of a progesterone analogue to twelve hypercholesterolemic men with coronary disease. This analogue, ethinyl testosterone, did not relieve the feminizing effects and did not have any significant action on the circulating lipids and lipoproteins when administered at a level of 60 mg. daily.

TABLE III. CHANGES IN THE CIRCULATING LIPIDS AND LIPOPROTEINS IN 6 HYPERCHOLESTEROLEMIC MEN WITH CORONARY DISEASE WHEN ACTH WAS ADMINISTERED.

	Plasma Total Cholesterol mg%.	Plasma C/P Ratio	Serum $\alpha:\beta$ Ratio
Before ACTH	268	1.00	9:91
After ACTH	224	0.84	17:83
10 weeks later	260	1.03	6:94

TABLE IV. CHANGES IN THE CIRCULATING LIPIDS AND LIPOPROTEINS IN 6 HYPERCHOLESTEROLEMIC MEN WITH CORONARY DISEASE WHEN CORTISONE ACETATE WAS ADMINISTERED.

	Plasma Total Cholesterol mg%.	Plasma C/P Ratio	Serum $\alpha:\beta$ Ratio
Before Cortisone	282	1.05	11:89
After Cortisone	223	0.89	16:84
10 weeks later	249	0.96	12:88

Pituitary-Adrenal Axis

Certain clinical endocrine disorders, such as diabetes, myxedema and Cushing's disease, may be associated with the complications of atherosclerosis, and, as most metabolic processes are affected by several hormones, it is unlikely that the circulating lipids and lipoproteins are influenced only by the sex hormones. It is interesting, therefore, to consider some preliminary observations concerning the effects of other hormones on the circulating lipids and lipoproteins of hypercholesterolemic men with a previous myocardial infarct.

There have been few and conflicting reports concerning the effect of ACTH and cortisone on the circulating lipids and lipoproteins in the human (Conn et al, 1950; Adlersberg et al, 1951; Mann and White, 1953).

The intramuscular administration of ACTH-Gel 100 mg. daily for five days to six men resulted in significant depression of plasma total cholesterol, the C/P ratio and the concentration of cholesterol attached to the β lipoprotein fraction (Table III). The administration of cortisone acetate 100 mg. daily for ten days to six other men also resulted in significant depression of these values (Table IV). There was no change in the functional capacity or the

TABLE V. CHANGES IN THE CIRCULATING LIPIDS AND LIPOPROTEINS IN 6 HYPERCHOLESTEROLEMIC MEN WITH CORONARY DISEASE WHEN L-THYROXINE SODIUM WAS ADMINISTERED.

	Plasma Total Cholesterol mg%.	Plasma C/P Ratio	Serum $\alpha:\beta$ Ratio
Before Thyroxine	274	1.03	12:88
After Thyroxine	200	0.85	12:88
6 weeks later	270	0.96	12:88

frequency of anginal attacks in any of these men during or after treatment.

The intramuscular administration of desoxycortone acetate 15 mg. daily for five days to three men did not result in any significant change in the circulating lipids and lipoproteins.

Pituitary-Thyroid Axis

Thyroid stimulating hormone has not yet been administered to hypercholesterolemic men with coronary disease. The intramuscular administration of TSH 10 units daily for four days to three subjects with primary myxedema did not result in any significant alteration in the circulating lipids and lipoproteins or in the uptake of radioactive iodine; it seems unlikely, therefore, that TSH has any action on the lipids and lipoproteins in the absence of functioning thyroid tissue.

The administration of l-thyroxine sodium 600 μg. daily for fourteen days to six hypercholesterolemic men with coronary disease resulted in significant depression of plasma total cholesterol and the C/P ratio (Table V). The lipoprotein ratio did not alter during the fourteen days therapy but there was a decrease in the concentration of cholesterol on the β lipoprotein fraction (Swahn, 1953).

Pituitary-Pancreatic Axis

Our investigations of the effect of growth hormone and glucagon on the circulating lipids and lipoproteins in the human are not yet definitive. In the rat, only growth hormone causes stimulation of hepatic cholesterol biosynthesis after it had been inhibited by hypophysectomy (Boyd and McGuire, 1955).

During the course of the administration of insulin 50 units daily to three men with depressive states there was no significant change in plasma total cholesterol, but the circulating phospholipids were elevated and the concentration of cholesterol on the β lipoprotein fraction was increased.

Discussion

Any theory of atherogenesis must explain the remarkable increase in the incidence of the clinical manifestations of coronary sclerosis. This increase might be attributed either to an increase in the factors suspected of contributing to the formation of atheroma and thrombosis, or to a decrease in the critical level at which the disease becomes manifest, or both; in either event, an equally fundamental change has occured in the natural history of the disease. An increase in the susceptibility of arterial and myocardial tissue to the factors responsible for atheroma and thrombosis might be analagous to changes which are thought to occur as a conditioning effect of stress (Selye and Heuser, 1954). Our environment is almost certainly stressful to some individuals, and it is surely significant that the remarkable social, economic and dietetic changes of this century have coincided with the evolution of clinical coronary disease.

It is considered that the circulating lipids and lipoproteins undergo physiological variation during the menstrual cycle and pregnancy as a result of endogenous hormonal influences, and it has been demonstrated that these lipids and lipoproteins can be influenced by the administration of certain hormones. At present their modes of action are obscure, but, as the level of circulating cholesterol is largely the net result of biosynthesis and catabolism of cholesterol in the liver, these hormones probably influence lipid metabolism through the liver. The maintenance of normal metabolism depends on an intact endocrine system, the secretions of which are continually adjusted to the requirements of the organism. Certain endocrine diseases result from hormonal insufficiency or excess and it is traditional to associate each of them with one particular disturbance of metabolism, such as the association of diabetes with a disturbance of carbohydrate metabolism; but carbohydrate, protein and lipid metabolism should really be regarded as a single integrated mechanism which is profoundly disturbed when its hormonal control is altered. Variations in the hormonal control of metabolism might result in changes in the circulating lipids and lipoproteins, and, without consideration of cause and effect, it is generally agreed that coronary sclerosis is associated with abnormal circulating lipids and lipoproteins; one asks, therefore, if it is also associated with hormonal imbalance. In fact, is coronary atherogenesis an endocrine problem? Is it possible that the changing environment of this century might be injurious to certain susceptible individuals who develop hormonal imbalance, and also develop decreased tissue resistance to the factors responsible for the production of atheroma and thrombosis? It is likely that investigation into these aspects of coronary atherogenesis will be rewarding.

In summary, then, many hormones influence the circulating lipids and lipoproteins. Ethinyl estradiol is effective in improving the abnormalities in the circulating lipids and lipoproteins which accompany clinical coronary disease. The question is raised whether coronary atherogenesis is partly an endocrine problem. It is thought that clarification of the complex interrelationships of the endocrine system may advance the understanding of the metabolic defects present in coronary sclerosis, and possibly lead to a therapeutic approach.

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Urinary Estrogen and Serum Protein-bound Iodine Levels

In a Group of Post-Menopausal Women With and Without Myocardial Infarction

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It is generally recognized that myocardial infarction occurs with considerably less frequency in cyclic women than it does in men of a comparable age group. Following the menopause, however, women become increasingly susceptible to this condition. Certain clinical and experimental evidence suggests that estrogen may be responsible for the "protection" of the premenopausal woman.[1,2,3] The postmenopausal woman continues to excrete a small quantity of estrogen in the urine which presumably arises from adrenal or residual ovarian activity. If estrogen is protective, it might be expected that postmenopausal women who have developed myocardial infarction excrete less estrogen than a comparable age group of female subjects who are free of this condition. An investigation was undertaken in order to determine if this is the case.

In the selection of cases, special emphasis was placed on subjects between the third and fifth postmenopausal years, although women within the ages of forty-seven to seventy-one were included. Individuals with diabetes, hepatic or renal diseases, as established by the usual laboratory procedures were excluded from this study as were those with clinical evidence of myxedema. Individuals with myocardial infarction were so classified only if there was unquestioned evidence of its occurrence by electrocardiographic and other clinical studies. The control group consisted of ambulatory patients without any evidence of cardiovascular disease as established by the usual clinical and laboratory criteria. In order to determine the effect of long-term debility, a "sick" control group was selected from hospitalized cases who had various long-term dyscrasias and were moderately to severely debilitated therefrom. These included lymphosarcoma, multiple sclerosis, severe secondary anemia, et cetera.

Urinary estrogen was determined biologically using the method of Migeon and Gardner with the exception that the extractions were made with ether.[4] The results were calculated for twenty-four-hour specimens and expressed as microgram equivalents of estradiol-17β.

Urinary estrogen was determined on the specimens from 23 postmenopausal women with myocardial infarction, nineteen "well" controls and eight "sick" controls. Fifty-five per cent of the women in the control groups excreted 0.50 micrograms of estradiol-17β equivalents or greater as compared to 22 per cent of the infarcted group. Preliminary study of the distributions of the estrogen values in the various groups showed them clearly to be skewed and that an exponential transformation normalized them with regularity. Statistical computations of these data were therefore made using the logarithms of the original values. Differences between the means of the well and sick controls were not significant so the two control groups were combined, giving a total of twenty-seven controls. The mean log value of this group was -.2886 (antilog .516 microgram equivalents of estradiol-17β. The mean log value for the myocardial infarct group was -.6127 (antilog .244 microgram equivalents of estradiol-17β). This difference was highly significant. ($p = < .01$).

As part of this investigation, the serum protein-

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The Coronaries

The Coronaries through the Ages

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ALTHOUGH coronary heart disease has doubtless existed since the time of Adam, and may even have been responsible for his death, we have no recognition of it as a possible cause of morbidity or mortality until the year 1700. Inasmuch as current investigations strongly suggest that it is in large part a disease of civilized man, it may well have been rare in the stone age, when our ancestors dwelt in caves, and uncommon even in the Dark and Middle Ages. It has been an interesting though rather frustrating experience to try to trace its course from the earliest historical records to the present day.

The extraordinarily slow recognition of coronary heart disease through the ages is matched only by our ignorance of its etiology today, in September, 1955. It is fascinating to compare the current theories as to its causation. Some blame the stress and strain of modern life, others heavy labor and accidents, some tobacco, more still the various constituents of the diet, and some even today regard it fatalistically as God's will. A recent book has stressed the psychosomatic factors, opening with the admonition that "the fear of the Lord is the beginning of wisdom" and ending with the hope that "those who labor and are heavy laden" should seek spiritual aid for their health's sake. Probably we need both, a good psychosomatic attitude and good health habits. It may well be that, much as the padre in the Second World War s quoted as having said, "Praise the Lord *but* pass the ammunition," we should say, "Fear he Lord but do something else too in the way of preventive medicine."

Before Herrick

In the civilizations before Christ there is little or no note of illness or death that might now be interpreted as the result of coronary heart disease. Several Bible students have been unable to recall such instances and the historians of Egypt, Babylon, and Syria have not apparently uncovered reports of disease that might be interpreted as the result of defective coronary circulation. Prominent personages not infrequently died suddenly or after very short illnesses but almost invariably these deaths are to be ascribed to accidents, war, assassination, poisoning, and infections. It is possible once in a great while to blame the heart for certain troubles recounted by historians of ancient days but this is only by inference. To be sure, calcification has been noted in the arteries of Egyptian mummies but there is no record of such calcification of the coronary arteries so far as I know, quite possibly because of the lack of preservation of the hearts themselves.

There is perhaps a little circumstantial evidence in the history of Israel. For example, the High Priest Eli, as recorded in the first book of Samuel in the Old Testament, greatly disturbed by the news of the slaying of his sons and the seizure of the Ark of the Covenant by the Philistines, fell from his chair, perhaps as a result of a cardiovascular accident, and broke his neck. It is intimated that the fracture killed him rather than a heart attack, which may, however, have caused him to swoon. Incidentally it is of interest that his sons who were priests of poor repute had sinned against God in breaking the commandment not to eat fat and they were punished for that and doubtless other reasons too. They had partaken of morsels of fat from the sacrificial offerings. In the book of Leviticus, 7th Chapter, 22nd to 24th verses, the children of Israel had been ordered by Moses at God's command to avoid eating any fat from the ox, the sheep, or the goat. Whether

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this was wholly a matter of sacrificial rite or whether it was already recognized as a measure of preventive medicine, like many other commandments, we have no way of knowing. It is quite possible that within a few years we may need to advise the American people to do with their diet what Moses advised the children of Israel to do some three thousand years ago.

Another reference in the first book of Samuel, Chapter 25, is of some interest. A prosperous land owner, named Nabal, obviously with considerable concern about his flocks and unsympathetic in his dealings with David, after a controversy with his wife and a feast at which he got quite intoxicated, suffered from an acute illness which prostrated him so that he lay like a stone for ten days before he died. He was, of course, "struck down by God"; but in all probability he suffered either from a cerebral vascular accident or a profound coronary heart attack, perhaps a punishment for his prosperity, as in modern days. As one reads through the Books of Kings and the Chronicles in the Bible, looking for other possible examples of cardiovascular disease, one is struck by the numerous instances of assassination or death in war. When there is recorded what appears to have been natural deaths as in the case of David and Solomon, who ruled for forty years, it is said simply that they slept with their fathers.

So far as I am aware, we have no knowledge from records in the East in the writings of India and China of the existence of coronary heart disease. The average lifetime in Asia has always been short, being terminated early as a rule by accident, war, or infectious disease. However, three years ago I myself saw in consultation in New Delhi, India, a young native, twenty-nine years old, with typical myocardial infarction.

Historians of Greece and Rome have little more to add. Socrates died of hemlock poisoning. Alexander died at an early age of dysentery. Caesar was assassinated. In the Dark and Middle Ages, the duration of life was still grossly limited by wars, starvation, and pestilence. Although the coronary arteries themselves had been recognized by the Alexandrian anatomists, Erasistratus and Herophilus, and more fully described by Vesalius and Vieussens more than a thousand years later in the sixteenth century, disease thereof was not reported.

Unable myself to recall the names of prominent personages who may have suffered from coronary heart disease in ancient times in the Dark and Middle Ages, in the Renaissance, and in the writings of the great authors like Shakespeare, I have asked students of those eras and literary works to help me. Professor Gilmore, chairman of the Department of History at Harvard University, has written to me that he believes very few celebrated persons at the time of the Middle Ages and the Renaissance in Europe died from anything that could be diagnosed as heart disease. Also he stated that longevity was apparently quite rare; for example, Emperor Charles V considered that he was an old man when he resigned the crown at the age of fifty-five.

In more modern times coronary victims (probable or certain) include Matthew Arnold, Charles Dickens, Nathaniel Hawthorne, the Earl of Clarendon, Charles Bell, Sir James Mackenzie, Sir Thomas Lewis, Richard Cabot, President Harding, and Wendell Wilkie.

It was Bonetus who, in the second edition of his *Sepulcretum* published in 1700, reported the first association of a seriously pathological condition of the coronary vessels with rapid death. A fat poet, after a hearty meal and much orating, climbed a flight of stairs, was seized by great discomfort in his chest, and died within a few minutes. At autopsy he was found to have such narrowed coronary arteries that it was impossible even to insert the end of a needle into them. We do not know whether he had pain or not. The description was that of discomfort which might have been due to or associated with some difficulty in breathing.

In 1728, Lancisi wrote on Sudden Death, namely *De Subitaneis Mortibus,* in order clearly to demonstrate natural causes of sudden death. Although a number of interesting illustrations were given, including cerebral hemorrhage and aortic rupture, there was no clear instance of coronary occlusion. Nor did Senac or Morgagni do any better; the latter in 1760 did, however, wonder what symptoms might be produced by coronary narrowing. He thought that palpitation could perhaps be so induced. Eight years later in 1768 Heberden gave his classical lecture on angina pectoris which was not published until 1772, but he did not know or guess that angina pectoris was due to coronary insufficiency. Within a few years, however, actually in the early 1770's, Jenner of smallpox fame noted calcification of the coronary arteries in the autopsy of a patient who died of angina pectoris. But since his close friend, John Hunter, had begun to have angina pectoris at about that time or shortly before, Jenner did not announce this discovery of his, confirmed by later autopsies, until 1799 when a famous letter of his was published in Parry's book entitled *Syncope Anginosa*. Parry cited several cases in that book. Fothergill (1776) and Black in 1794 published reports of cases of angina pectoris who showed postmortem ossified coronary arteries. Fothergill also described myocardial scars.

Thus, at the end of the eighteenth and the beginning of the nineteenth centuries, there was the clear recognition by a few individuals of this association of coronary artery sclerosis with morbidity and mortality. Despite this recognition a hundred and fifty years ago, the medical world was very slow in paying attention to its importance. A few scattered papers appeared here and there at the beginning of the century, such as that by John Warren on page 1 of Number 1 of Volume 1 of the *New England Journal of Medicine,* published in Boston in 1812.

When the *New England Journal of Medicine* changed its name to the *Boston Medical and Surgical Journal* in the late 1820's, it was the custom to record the deaths in Boston week by week. I have selected the year 1829 to peruse the causes of death of Bostonians and have found the following. Among the total deaths of 1,221, the most common cause was consumption, that is, tuberculosis, with 203 cases. Many other infections played their role also. Pneumonia was second with ninety deaths and measles third with seventy-two. In searching for cardiovascular deaths, I discovered that "old age" was cited sixty-five times; it is almost certain that a good many of that group died from diseases of the heart and arteries. Apoplexy was specifically mentioned twelve times in addition and twelve others had dropsy. Interestingly enough, the diagnosis in only seven individuals was noted as that of "sudden death." Rare cases died of "enlargement of the heart," of "disease of the heart," of "palpitation of the heart," of "complaint of the heart," and of "ossification of the heart." Rheumatic fever was recorded in very few individuals as a cause of death. Although angina pectoris had been described and clearly recognized during several earlier decades, and although it was the title of the very first paper published in the *New England Journal of Medicine,* there was not a single diagnosis of angina pectoris as a cause of death in Boston in that year 1829. There were 160 persons who died of "diseases unknown"—this miscellany may well have included some "cardiacs" and coronary cases as well as diabetics, who were not noted at all in the table, and individuals who had obscure cancer or blood diseases.

It has been of further interest to determine the average ages at death of the male and female Bostonians in the year 1829. Almost exactly half of 1,221 persons who died that year were tabulated by sex and age. There were 303 males and 305 females, a total of 608. The males averaged only 23.5 years at death, the females 26.8 years, and both sexes together 25.2 years. This is in contrast to Bostonian males and females in 1953 who averaged 67 and 72 years respectively with total average of 69.5 years. In the year 1829, one quarter of the deaths (154 or 25.3 per cent) occurred in infants aged two years or younger, and only forty-six (7.6 per cent) lived to the age of 70 years or more. In 1953, only 4.7 per cent died under the age of five years, while 45 per cent died at 70 and over. The death rate in Boston in 1829 was 1,221 out of 61,392 inhabitants, which was almost exactly 2 per cent. The Boston death rate in 1953 was 1.2 per cent. From all these figures, it is quite evident that one important reason why there was undoubtedly less coronary heart disease in 1829 in Boston than now was the fact that the average Bostonian lived too short a life to acquire it. Other important reasons were, of course, failure to look for it and inadequate techniques. Just how great an increase there actually has been no one knows.

Medical writers continued to pay scant attention to coronary heart disease in the middle of the century and even towards the end of it. Austin Flint stated in his writings in the 1870's that he had rarely seen angina pectoris in his practice in New York City; he said, in fact, that an interval of five years had passed without his having had such a patient, a statement which has seemed incredible. This observation of his meant that he was either not recognizing cases of the sort, at least the milder ones, perhaps on the basis of inadequate history taking, or that the disease was actually rare in one of the large cities in this country. Quite possibly both factors are to be given equal credit. On the other hand, pathologists were quite aware of coronary sclerosis during the last few decades of the century; among them were Weigert in 1880, Cohnheim in 1881, Ziegler in 1881, Huber in 1882, and Leiden in 1884. Marie in 1896 published a thesis on myocardial infarction. A few internists, in particular Osler in 1910 did lecture on angina pectoris.

This brings us well into the twentieth century. My own teachers at the Harvard Medical School, almost all of whom were in active practice, were cognizant of angina pectoris and its serious significance, but hardly mentioned it in their teachings, absorbed as they were by infectious diseases. These teachers of mine included Frederick Shattuck, Henry Jackson, Henry Christian, and Richard Cabot. Later when I was an intern at the Massachusetts General Hospital, my attention was rarely called to this disease. We were still swamped with the problems of infection which included in particular dysentery, typhoid, syphilis, the so-called contagious diseases, and tuberculosis. The age range of the patients in the medical wards was far lower than it is today. There was no room for middle-aged or older patients with which we are now overburdened, patients with cancer, arthritis, and cardiovascular diseases.

While I was an intern in the hospital in 1912, James Herrick of Chicago established his immortality in medical history by his recognition and description of the clinical entity of coronary thrombosis, but, strange to say, although he presented his paper before learned colleagues at a national society meeting, little or no attention was paid to it for the next ten years. This was doubtless in part due to the First World War, but whatever the cause, it is another example of the slowness with which important scientific contributions were recognized in former days.

My own early experience at the Massachusetts General Hospital for the ten years from 1912 to 1922 was interwoven with a year of study under Thomas Lewis in London in 1914, a few months in the British Expeditionary Forces back of the Somme in 1916, a year and a half in a General Hospital in the A.E.F. in France from 1917 to 1919, and six months on a relief mission to Greece in Macedonia in 1919. In none of these experiences or in the clinics at the Massachusetts General Hospital was I more than faintly aware of the existence of angina pectoris and certainly I did not recognize coronary thrombosis. I recall that in 1920 I was asked to see in consultation a prominent surgeon from another state who was visiting a New England colleague at his summer place. This surgeon had been afflicted by what he thought himself, and to which his medical and surgical friends agreed, was an attack of cholecystitis complicated by pericarditis. I was asked to bring along a needle and syringe for a pericardial tap but I found this unnecessary since the pericarditis was quite dry. There was a very slow recovery from this illness, and shortly after his return home to Cincinnati there was evidence of heart involvement. An electrocardiogram was taken and sent to us. As I recall it now, there was a marked abnormality of the T waves which we noted but did not recognize as pathognomonic of any particular condition. Shortly after this, however, that is, by 1921, most of us had begun to be aware of the clinical condition of coronary thrombosis and its electrocardiographic manifestations.

There were a few exceptional individuals who recognized the disease earlier, two of whom were Samuel Levine of Boston, who in 1918 wrote an article entitled "Infarction of the Heart Simulating Acute Surgical Abdominal Conditions," and L. Whittington Gorham of Albany, who in 1920 published a paper entitled "The Significance of Transient Localized Pericardial Friction in Coronary Thrombosis (Pericarditis Epistenocardica)." Levine has told me that at the time he prepared his paper he was unaware of Herrick's important contribution of six years earlier. Incidentally, Levine published an important monograph on coronary thrombosis in 1929.

My Own Generation

Thus, the present generation has slowly and painfully acquired knowledge about the coronary circulation and its diseases, which, of course, are almost wholly summed up in the one word "atherosclerosis." I would like to interpolate at this point a brief observation, namely that we as internists have been even slower in the recognition of the importance of cerebral atherosclerosis. We have left this problem largely to be dealt with by our neurological colleagues, but we should certainly share the responsibility with them.

My own private practice started in 1920, and I have recorded the diagnoses of all my private patients from the beginning — etiologically, structurally, and functionally. During the first year of my practice in 1920 I did not make a single diagnosis of coronary thrombosis. The first case clearly so recorded was that of a man aged seventy-one, whom I saw in consultation with my father on January 13, 1921. He died thirteen days later in pulmonary edema. During the years 1921 and 1922 I made the definite diagnosis of coronary thrombosis in nine cases and questionably in five more. Meanwhile a diagnosis of "cardiosclerosis" or of "arteriosclerotic heart disease" with or without angina pectoris was not at all rare. The most common occurrence of so-called arteriosclerotic heart disease or cardiosclerosis (these terms were used interchangeably) was in the case of older individuals, that is, those more than fifty years old who had any arrhythmia or cardiac enlargement. Such was the custom in diagnosis that we now have happily long outgrown since we know that arrhythmias can occur at any age without any underlying structural heart disease and that hypertension is a common cause of cardiac enlargement.

To continue with my own personal experience, I have looked up the cases which I diagnosed definitely as coronary thrombosis in the years 1925, 1935, and 1945. During those years my practice remained much the same in character, consisting almost entirely of patients with cardiovascular signs or symptoms. During the earlier ten years of my practice I was seeing more patients annually than later but the percentages are of interest and importance.

In 1925 out of 501 new cases in my own practice, I diagnosed coronary thrombosis definitely thirty-two times, in twenty-five males and seven females. This is 6 per cent. I would add that there was one male under the age of forty and that two of the females had diabetes. During that same year there were six questionable male cases and two questionable female cases.

In 1935 out of 357 new cases of my own, I saw a total of forty-eight patients in whom I diagnosed definitely coronary thrombosis; forty-five of these were male and three were female. There was one male under forty. Thus, during that year the percentage of coronary cases in my practice increased to 13 per cent. In 1935 there were in addition six questionable male and four questionable female cases of coronary thrombosis.

In 1945, out of 351 new cases seen in private practice, there were seventy-one cases definitely diagnosed by myself as coronary thrombosis, which gives a percentage of 20; fifty-seven of these cases were male and fourteen female. There were three males under the age of forty, and two of the females had had myxedema. During that same year there were thirteen males questionably affected by coronary thrombosis, two of whom were under forty, and there were three questionable females. Thus, there was a steady rise in those three years at decade intervals from 6 per cent to 20 per cent.

I hasten to add that I am not including in this category all the cases of coronary heart disease, inasmuch as there were a good many patients with definite angina pectoris without myocardial infarction, actually more than I was seeing with coronary thrombosis and myocardial infarction. Nor have I included the relatively few cases of the Adams-Stokes syndrome.

I would like at this point to acknowledge my indebtedness, and that of some of my cardiological colleagues in Boston, to Timothy Leary who demonstrated to us in those early years the microscopic pictures of coronary artery atherosclerotic lesions and occlusion and myocardial scars too. During those early days it was not always the rule for pathologists in medical schools or hospitals to examine the coronary arteries in great detail. Also not infrequently, as I myself discovered, small myocardial infarcts, both old and new, were being overlooked. I remember one case with a small posterior myocardial scar visible under the posterior cusp of the mitral valve, once the chordae tendinae had been cut, and with a very small patch of adherent pericardium posteriorly, which had been overlooked by the pathologist but which clinically we had diagnosed. Thus it is that some of the increased frequency of coronary heart disease is more apparent than real but how much no one knows. Some authorities have, however, stated that, on searching back in old hearts preserved from former years in pathological museums and pathological protocols, coronary heart disease was not uncommon and that there has not been so great an increase in its frequency noted by pathologists as by practicing physicians, but it hardly seems possible from our general clinical experience that coronary heart disease even at the same ages was as common a generation ago as it is now.

There is one other important contribution that I would like to pay tribute to at this time and that is the fundamental work by Schlessinger and Blumgart concerning the ramifications of the coronary circulation, both normal and abnormal. They demonstrated clearly the relationship of the site of the coronary thrombosis to the location of the resulting myocardial infarction and most important of all they made the discovery of the vital importance of the development of a collateral coronary circulation in the restoration of improved myocardial oxygenation and restoration of health, often with complete clinical recovery.

I would like now to end this part of my story with a few of the more important conclusions concerning the clinical aspects of coronary heart disease resulting from this experience of my own. I feel quite sure that many of my colleagues who also have been in clinical practice for a good many years will have had the same experience.

1. It is very important to recognize the degree of trouble. One may have the least possible amount of coronary heart disease to diagnose or one may have it so severely that death comes at once from coronary insufficiency or within a few hours from a fulminating attack of myocardial infarction. In between these two extremes there are all grades and manifestations of the disease. There is no strictly average case, although there are a good many who are close to the middle line. I often tell my patients who have a slight degree of angina pectoris on effort that they are very little different from their colleagues and friends of the same age who are free of symptoms. Doubtless some of these individuals who seem to be free of symptoms could have angina pectoris or even die if they should run very hard for a train or engage in very unusual strenuous exercise carrying as they do a considerable amount of so-called silent coronary atherosclerosis. Thus there may be only a few per cent difference between the middle-aged man who has angina pectoris and the middle-aged man who hasn't. In summary, one may have, just as in the case of many other conditions, such as respiratory infections or accidents, a touch of trouble or a fatal amount.

2. In addition to the variation of the degree of trouble there is often a considerable variation in the symptomatology thereof. Some individuals are hypersensitive and are aware of very little actual disease. Others are so insensitive that they may be almost mortally ill before they are aware of it. In my own experience with patients I have found that the heavy use of alcohol tends to favor the suppression of symptoms which may be important. One must, therefore, judge the person himself as well as the disease in diagnosis, prognosis, and treatment.

It is quite probable that a failure to take into consideration the two points I have just mentioned may account for at least some of the apparent increase of coronary heart disease as diagnosed today in comparison with that of twenty-five to fifty years ago.

3. There has been often a good deal of difficulty about nomenclature and a variety of terms have come into usage which are sometimes confusing. I myself was guilty of some of that years ago when I separated off in the first two editions of my book angina pectoris as a functional entity from coronary heart disease. It is true that there are a few cases of angina pectoris due to coronary insufficiency without any coronary artery disease but they are uncommon. Such cases include patients with syphilitic aortitis causing coronary mouth narrowing, or severe valvular stenosis, either aortic or mitral, with inadequate cardiac output and, therefore, inadequate coronary blood supply, and a few other causes, not commonly operating in the same way, however.

To my mind it is simplest to speak of coronary heart disease as an abbreviation of coronary atherosclerotic heart disease with its various subdivisions as shown by simple coronary insufficiency (usually angina pectoris) on effort, coronary thrombosis with or without infarction, and heart block, bundle branch or a-v, with or without the Adams-Stokes syndrome.

4. At the same time that during the last generation a correction has been made in changing the diagnosis of acute indigestion in many patients to coronary heart disease, especially to acute coronary thrombosis, and adding cases to the coronary category who had been underdiagnosed because of the mildness of their attack or of their long survival, or because of lack of electrocardiographic evidence, there has developed an opposite tendency to overdiagnose coronary heart disease in some cases in whom indigestion, in particular cardiospasm, really does exist as a cause of symptoms, or in whom pericarditis may be confused with acute myocardial infarction. Happily the old custom still too prevalent in a good many places of labeling any older patient with arrhythmia or cardiac enlargement as having "arteriosclerotic heart disease" or "cardiosclerosis" is on its way out. Many persons seventy or eighty years old who may have premature beats or paroxysmal tachycardia or atrial fibrillation have no important heart disease, coronary or otherwise, and some of them have had arrhythmias of the sort ever since they were twenty years old. Also a good many of the persons who have enlargement of the heart of unknown cause or due to unrecognized hypertension in the past or present have been wrongly labeled arteriosclerotic heart cases.

5. One of the most dramatic changes that has come in our understanding of coronary heart disease during the last generation is that which concerns prognosis. It was customary in the early 1920's to regard the fate of individuals with either angina pectoris or coronary thrombosis, but more particularly the latter, as very dark. It was generally believed by the medical profession as well as by the laity that life would extend for only a few years at best and that it was likely to be quite an invalid life at the same time. Hence, I well remember hearing many persons say that since their life would be a short one, it might as well be a merry one, and all caution was thrown to the winds. A strenuous and gay life was carried on, and this, to be sure, did not infrequently end abruptly in a relatively short time. Their motto was: a short life, therefore a gay one. As time passed, however, many patients clearly diagnosed as having angina pectoris or coronary thrombosis, or both, were found not only to live along year after year but actually to recover fair health if not indeed in some cases good or excellent health. Many patients got perfectly well again and have continued so for many years.

One of the cases that I saw myself in consultation during a rather severe attack of acute coronary thrombosis in 1927 when he was fifty-one years old is perfectly well today after twenty-eight years. Now, seventy-nine years old, he is able to play golf without symptoms and yet with a moderately large scar in his heart.

It is quite obvious now that one of the reasons why coronary heart disease was underdiagnosed in the 1920's and possibly in the 1930's too was that if there was recovery it was commonly said that if the patient recovered or lived more than a few years, the original diagnosis must have been wrong. At the present time, the average duration of life after the first symptom of coronary heart disease is somewhat more than ten years and this includes those who die quickly as well as those who survive twenty-five years or better. This change in viewpoint naturally affected greatly our attitude toward the disease and has influenced our opinions about treatment.

6. Thus, following up what I have just said, the prognosis and treatment of coronary heart disease must be very carefully individualized. Although there are a few simple general rules, no two patients are exactly alike and therefor the therapy must be made to fit the individual case. Most often no very special treatment at all is necessary except for a full discussion with the patient of what we know and what we don't know about the disease and the application of common sense measures. Very few medicines are important, although specifically we do use a good deal of nitrite therapy in a prophylactic way as well as in the treatment of the symptom of angina pectoris. We employ rest and often anticoagulants at the time of an attack of coronary thrombosis with myocardial infarction and we do prescribe a low calorie and especially a low fat diet for individuals who are either candidates for coronary heart disease or who have had evidence thereof, especially if they are overweight and their serum

cholesterol content is high. More important, however, than all such therapy is our current intensive study of coronary heart disease with a view to prevention.

The Future

It is quite obvious from what I have already said and from the extensive program of the present symposium that coronary heart disease is tremendously in the limelight in this country today. It is also quite obvious that research with a view to its prevention is of prime importance. The laboratory study of animals, the clinical study of patients, and, of at least equal importance, the epidemiological studies throughout the world, well conducted by international teams, must receive full support. It should be possible within the next decade to learn so much from all these researches that we can give useful advice to the candidates for early coronary heart disease to prolong their lives and years of usefulness and happiness. Not only does this apply to the citizens of this country but to the people in every nation in the world who are inclined, as a result of their increasing prosperity, to live the way most Americans live today.

RESEARCH IN ARTERIOSCLEROSIS

(Continued from Page 745)

one-sided and uninhibited claims of success. Physicians and scientists must preserve their heritage of criticism. Without it they will not build a tower of knowledge for too many unsound struts will have been introduced to support it.

So whether we think arteriosclerosis is due to smoking, drinking, eating too much of everything or just too much fat, too much or too little exercise, too much or too little vitamins, too much or too little protein, too much or too little thyroid, let us remember that there are differences among people and among peoples. There are those who might care to become Bantus, there are those who might care to go back to Methuselah and I only hope that there are few of us who, for the sake of a placque in our arteries, would care to abolish those small or large differences which make us men and women. Investigators must find a better solution to the problem than that.

URINARY ESTROGEN AND SERUM PROTEIN-BOUND IODINE LEVELS

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bound iodine was determined in thirty-eight postmenopausal women with myocardial infarction and forty-seven control subjects. A modified Barker procedure was used.[5] Forty-two per cent of the patients with myocardial infarction had PBI values below 4.6 micrograms per 100 cc. of serum, whereas only 11 per cent of the control group were in this category. The mean PBI value for the former was 5.10; the latter 5.81. A statistically significant difference was found. ($p = .05$).

These observations suggest that diminished estrogen and thyroxine production may play an important role in the occurrence of myocardial infarction in postmenopausal women.

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Case Presentation

DIABETES INSIPIDUS AND PREGNANCY

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THE coexistence of diabetes insipidus and pregnancy is a rather infrequent condition. This is attested by the fact that Williams stated that the incidence was nineteen cases per 100,000 admissions.

According to Lake,[1] Frank in 1794 was the first person to describe adequately a case of diabetes insipidus. Before this it had been confused with diabetes mellitus. Lake[1] also states that Von Velden and Farini independently commented on the efficacy of pituitary extract in controlling the polydipsia and polyuria. Diabetes insipidus is apparently due to a deficiency of an antidiuretic factor normally produced in the posterior lobe of the pituitary gland. According to Ranson and associates (quoted by Carfagno et al[2]), there are nuclei located in the hypothalamus which control the functions of the posterior lobe of the hypophysis. About 63 per cent of diabetes insipidus cases are due to tumor, 10 per cent to trauma, and the rest due to infections (lues, encephalitis) and idiopathic.[1] The dominant symptoms and signs are of polydipsia, polyuria, and urine of consistently low specific gravity. According to Von Hann (quoted by Carfagno et al[2]) and Richter,[3] the anterior lobe of the pituitary must be intact in order for diabetes insipidus to develop. Patients with Simmonds' or Sheehan's disease do not have diabetes insipidus. Carfagno and colleagues[2] believe that the anterior lobe of the hypophysis exerts its diuretic effect through the adrenal cortex, and that this effect is delicately balanced by the antidiuretic effect of the posterior lobe. They also feel that the thyrotropic hormone of the anterior lobe may play a part, and that the placenta itself may secrete a diuretic factor. Therefore, quoting Carfagno again, there are several factors which increase the diuretic effect in pregnancy itself: The stimulation of the adrenal cortex, the stimulation of the thyroid, the placental factor, and the diuretic effect of increased nitrogenous wastes of pregnancy.

Soule[4] in 1937 found thirty-six cases of diabetes insipidus associated with pregnancy and added one of his own. At that time he made some observations on the previous cases: (1) The consistent lack of demonstrable posterior pituitary lesions (as determined by skull x-rays), (2) the infrequent accompaniment of a positive Wassermann reaction, (3) the frequent occurrence of premature labor, and (4) the uniform response to posterior pituitary extract.

It seems that the most consistent finding in diabetes insipidus with pregnancy is the remarkable variety. The disease may be aggravated by pregnancy, or may be unaffected or even ameliorated during pregnancy. Diabetes insipidus may be transitory during pregnancy, may have its onset during pregnancy and persist afterwards, or may appear transiently following delivery. Novak (quoted by Blotner and Kunkel[5]) reported a patient in whom diabetes insipidus developed during her first pregnancy and disappeared post-partum, the same sequence of events recurring in her ten subsequent pregnancies. Weil (also quoted by Blotner and Kunkel[5]) and his son studied five generations of a family of 220 members, headed by a man with diabetes insipidus, of whom thirty-five developed the disease.

Case Report

The patient was eighteen years old when first seen in 1949. She was followed through a normal pregnancy which terminated in a normal delivery under saddle block anesthesia in that same year. Her post-partum course was uneventful. In February, 1951, she had German measles (rubella). Following this she developed intense headache and general malaise. In retrospect, it is possible this may have represented a post-rubella encephalitis. In the early part of July, 1951, she developed polydipsia, polyuria, anorexia, ataxia, dizziness, etc. She was seen by an internist who found a twenty-four-hour urine output of 10,100 cc., with a specific gravity of from 1.001 to 1.005. She was given

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From the Asbury Methodist and Abbott Hospitals, Minneapolis, Minnesota.

This report was presented at Medical Grand Rounds of Asbury Methodist Hospital, Minneapolis, Minnesota, May 5, 1955.

TABLE I. RESUME OF TWELVE RECENT CASES OF DIABETES INSIPIDUS AND PREGNANCY

Author and Year	Etiology	Unusual Symptoms	Treatment	Course of Pregnancy	Type Labor	Baby	Later Course
Greene and Gibson (1938)[7]	?	Diabetes mellitus, infrequent menses	Nasal insufflation of pituitary	Normal pregnancy diabetes insipidus better	Normal	Died on second day of brain injury	Intranasal pituitary continued post-partum
Carter (1940)[8]	?	Sterility 5 years Scanty menses, poorly developed secondary sex characteristics	Procedures for sterility ineffective till pituitrin used	Continued with pituitrin till 7½ months	Normal except for cephalopelvic disproportion and head did not engage	Stillborn by Cesarian section 9 lbs.	No pituitrin after pregnancy, had output of 4-5 liters
McLaren and McLeod (1942)[9]	Fell in 22nd week of pregnancy. Hit side of head x-ray ok. Traumatic?	None	No pituitary extract. Disease began in pregnancy and ended post-partum	Diabetes insipidus developed at 34 weeks. Abruptio and hepatitis at 36 weeks	Induced by amnionotomy. Normal contractions	Died during labor 6 lbs. 2 oz.	Pitressin daily post-partum. Hemorrhage on third day. No recurrence of diabetes insipidus
Blotner and Kunkel (1942)[5] 2 cases	? Had it for 9 years before pregnancy	Dysmenorrhea caused by the pituitrin injections	Used pituitary insufflation throughout pregnancy	Disease worse Output increased up to 7 months then decreased till term	Normal	Normal 8¼ lbs.	Output continued at 8 liters. Pituitrin was continued afterwards
	Had it during a previous pregnancy	None	None	Diabetes insipidus reappeared in fifth month of present pregnancy. Became worse	Repeat Cesarian section done 18 days before term	Twins normal	Symptoms disappeared post-partum and were absent for at least 3 months
Maranon (1947)[10] 1 patient with diabetes insipidus in 3 pregnancies	?	None	Treated for 12 years before pregnancy. Continued treatment during pregnancy with pituitrin	Diabetes insipidus unchanged during pregnancy.	Labor began, then stopped	Stillborn	See next pregnancy
	Had 3 month abortion						
			Treated with pituitrin	Went to term	Inertia was treated with pituitrin. Normal delivery	Normal	See next pregnancy
			Same treatment	Went to term	Inertia. No pituitrin, 3 day labor	Stillborn	At 43 years of age still had 11 liters urine output
Lake (1947)[1]	Fever at 17 years age. Encephalitis?	Weakness weight gain scanty menses absence of libido—hirsutism	Pitressin tannate	Had diabetes insipidus in a previous pregnancy, continued in this one	Inertia. Forceps	Normal	Disappeared post-partum. Became pregnant third time Diabetes insipidus recurred and she was aborted
Tannenbaum, Bertling, and Burwell (1952)[11]	?	None	Pitressin tannate	Needed less pitressin as pregnancy went on. None after 6 months	Normal—low forceps	Normal, average size	48 hours after delivery resumed pitressin
Carfagno, Durant, Shuman (1953)[2]	?	None	Developed diabetes insipidus in a previous pregnancy, continued pitressin in this one	Used pitressin till onset of labor	Normal. No pituitrin. Spontaneous delivery	Normal 7 lbs.	Diabetes insipidus disappeared post-partum
Hickey, Keette (1954)[12]	?	None	Pitressin before pregnancy continued	Pitressin used all through pregnancy till 24 hours before induction	Normal Spontaneous delivery	Normal	No mention
McKenzie, Swain (present report)	Post-rubella encephalitis	Irregular menses Sterility till diabetes insipidus under control	Pitressin tannate before and during pregnancy	Required less pitressin as pregnancy progressed. None at all last 10 weeks	Normal	Normal 6 lbs. 14¾ oz.	No return of diabetes insipidus post-partum

pitressin in the office but she developed carpopedal spasm and generalized cramps and was hospitalized. General physical and neurological examinations were essentially negative. An x-ray of the skull showed the sella turcica to be normal. The Wassermann reaction was negative, the blood urea nitrogen 7 milligrams per cent, the blood sugar 83.5 milligrams per cent. She was placed on pitressin tannate in oil 1 cc., intramuscularly twice daily. With the pitressin injections, both intake, output, and specific gravity became normal. She was sent home after two weeks in good condition. She continued on the medication twice a day for a while, then once a day in late 1951. In 1952 it was used once every other day, and in 1953 once a week and finally, once a month. During the first part of 1954 it was also necessary once a month. The patient's menses were grossly irregular from the time of the rubella (February, 1951) till April or May of 1954.

Her last menses occurred on June 8, 1954. She continued taking pitressin during the first part of her pregnancy. For a while she required injections oftener, from two to seven times monthly depending on symptoms. The need for them gradually decreased so that during the last ten weeks she needed none at all. The membranes ruptured spontaneously at term on March 14, 1955, and labor began three and one-half hours later. The first stage lasted eight hours, the second was terminated by low forceps after fifteen minutes, and the placenta was delivered five minutes later. The uterine contractions were normal throughout. The patient received no pitocin or pituitrin but did get the routine ergotrate on delivery of the head. The blood loss was estimated at 50 cc. The baby was quite normal and weighed 6 pounds 14¾ ounces. There was no return of the polydipsia or polyuria during the hospital stay, and the specific gravity of the urine during this period ranged from 1.009 to 1.032. When the patient returned for her six-week post-partum examination on April 22, 1955, she stated she still had no return of symptoms. Pelvic examination was entirely normal. She had not had any pitressin since December, 1954.

Since Soule's report (1937) of the thirty-seven cases of diabetes insipidus in pregnancy, we have been able to find eleven additional cases reported. The case herewith reported makes a total of forty-nine.

Discussion

In this portion we shall be concerned with the eleven recent cases of diabetes insipidus and pregnancy we have culled from the literature, and our own case.

Of these twelve, only one could possibly have developed diabetes insipidus as a result of trauma and this is doubtful. There were two patients in whom the disease may have occurred as a result of encephalitis. There were none in whom tumor or lues could be proved as a cause. Four of the patients had irregular or scanty menses; this was accompanied by decreased fertility in two of the four.

In ten of these twelve patients the disease began before pregnancy, the remaining two had the onset of diabetes insipidus during pregnancy. In four of the patients the disease was less severe or disappeared during pregnancy, in three it became worse and in the remaining five it was unchanged. The disease disappeared after pregnancy in five patients, persisted in six, and in the other there was no mention of the post-partum course. We concur that the outstanding feature of diabetes insipidus in connection with pregnancy is its great variety!

Two of Soule's observations can be borne out by our review: There was no x-ray evidence of a pituitary lesion in any of the patients, and there was no relationship between the disease and syphilis. His other two observations are more controversial. He felt that premature labors were more common; this may have been the result of using pituitrin injections or nasal insufflations of pituitary extract instead of pitressin injections. The only premature labor in this series was induced by amnionotomy because of abruptio placenta, and this patient received no pituitary preparations prior to this procedure. Soule also stated that there was uniform good response to posterior pituitary therapy. In one of the present group the author stated that pitressin failed to control the polyuria. In two others the disease was described as becoming worse despite treatment.

Treatment of these patients was divided between pituitary nasal insufflations, pituitrin and pitressin injections. In some, whole posterior pituitary, whether given intranasally or by injection, seemed to cause annoying uterine contractions whereas the pitressin did not. Two of the patients developed uterine inertia during labor, one of these had inertia in three successive pregnancies. However, in the light of the work of Fisher et al[6] one might expect a higher incidence of inertia. These authors were able to demonstrate the occurrence of uterine inertia in cats and monkeys when the stalk of the hypophysis was cut.

Summary and Conclusions

1. The theory of the pathologic physiology of diabetes insipidus has been briefly reviewed as have the various etiologies.
2. The incidence and significance of its coincidence with pregnancy have been discussed.
3. Eleven recent cases of coexistent diabetes insipidus and pregnancy are reviewed and one of our own has been reported.

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Laboratory Aids

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George G. Stilwell, Editor

EXFOLIATIVE CYTOLOGY: A REALISTIC APPRAISAL

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Rochester, Minnesota

It is now almost thirty years since the cytologic examination of exfoliated cells was developed as a diagnostic test for cancer. As with many new methods, immediate acceptance of the technique was not widespread. During the past ten years, the method has been tried with regard to every conceivable bodily orifice and secretion, with different degrees of success. The reaction to this technique has varied not only among individuals but among groups of specialists. Some groups have been most enthusiastic, whereas others have been frankly skeptical. For example, many gynecologists have accepted the method as one that eventually may eliminate carcinoma of the cervix as a cause of death. Gynecologists are of the opinion that cytologic study of cervical smears should be done at regular intervals (six months to one year) on all women more than thirty years of age on the assumption that this procedure would lead to the recognition of carcinoma of the cervix in its preinvasive stage and that the treatment of this lesion would eliminate subsequent infiltrative carcinoma of the cervix. So enthusiastic have some gynecologists become that a number of them make pathologic studies of and interpret cervical smears and even smears from other material. A national organization for exfoliative cytology has been formed largely through the efforts of certain gynecologists.

By contrast, pathologists have been skeptical toward exfoliative cytology. Their reasons have been valid in most instances. Pathologists maintain an inborn cautiousness toward the acceptance of an untried technique. Furthermore, the successful performance of this technique can be accomplished only by the generous use of the pathologist's time, a scarce commodity; thus, many pathologists have concluded that the procedure is neither economical nor practical. Also, many pathologists have feared that positive reports as the result of studies on such smears would be accepted by clinicians as proof of the presence of carcinoma, with no further efforts being made to perform biopsy. This, of course, could lead to unfortunate treatment.

The mails recently have been flooded with literature concerning the Cancer Cytology Foundation of America, Inc., whose president is Dr. J. Ernest Ayre. This is an organization to provide a mail-order service in cytology on a nationwide basis. It does not appear that this approach to the practical application of exfoliative cytology is the one that is best fitted for the sound practice of medicine.

The greatest field for usefulness of exfoliative cytology apparently lies in the field of cervical cytology. As this test is now done, the cells that are examined are not exfoliated but are living cells scraped from the cervix; hence, the term "exfoliative" is not strictly accurate. The cervical smear is at its best in the diagnosis of carcinoma *in situ* of the cervix. Fairly good evidence exists that at times certain instances of cervical carcinoma *in situ* later have become infiltrative. However, considerable disagreement is present among pathologists as to what constitutes cervical carcinoma *in situ*. Siegler[1] sent to twenty-five pathologists a group of twenty slides that represented difficult and borderline problems in the histologic diagnosis of cervical carcinoma *in situ*. Complete diagnostic agreement was not obtained on any of these slides. Every individual slide was believed to represent a malignant lesion by at least one observer.

Less useful fields for exfoliative cytology include the lung and pleural and peritoneal fluids. However, carcinoma of the lung only occasionally is diagnosed solely on the basis of cytologic ex-

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From the Section of Surgical Pathology, Mayo Clinic, Rochester, Minnesota.

amination. Evidence of a pulmonary lesion in the lung usually is present in thoracic roentgenograms, a fact that, by itself, renders an operation imperative; also, "positive" results of bronchoscopic biopsy are more accurate than are "positive" results of cytologic studies. "Positive" results of the cytologic examination of ascitic fluid are falsely positive in at least 5 per cent of cases, so this technique is not completely reliable. It is highly questionable whether much value can be attached to routine cytologic examination of lesions of the skin, oral cavity, rectum or breast. Examination of gastric material, regardless of whether or not the Ayre "rotating gastric brush" or similar instruments are used, is of questionable practical value.

It is commendable that the American Board of Pathology has included exfoliative cytology in its examinations. The American Society of Clinical Pathologists has included this topic in one of its annual workshops. It appears that pathologists should take a more active interest in cytology to see that these techniques are done properly and ethically. In this regard, the pathologist is best suited for the performance of the test by virtue of his training in the interpretation of pathologic changes in tissues and his familiarity with laboratory organization and procedures.

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This is the thirty-third in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between laboratory physicians and their colleagues. This month's report complements one by the same author that was printed in this section last month. The reader also is referred to the ninth report in this series, which concerned cervical cytology and which appeared in MINNESOTA MEDICINE in October, 1953.

Several weeks ago, a total of 183,000 physicians received pamphlets describing the Cancer Cytology Foundation of America, Inc., which was mentioned by Dr. McDonald. This brochure was accompanied by a copy of the pamphlet called *Spotlight,* published by the Committee for Constitutional Government, Inc. This entire issue of the *Spotlight* was devoted to a discussion of exfoliative cytology under the title "Cancer Cytology is the Discovery of the Century." It is rather curious to see a medical diagnostic procedure being promoted by such an organization.

The American Cancer Society is evaluating Dr. Ayre's Foundation. They point out that pathology specialty groups have been opposed to the establishment of regional centers for cytology because the test is rapidly becoming available to patients and physicians desiring it and because it is important that the closest possible professional relationships be maintained among the patient, the clinician and the pathologists endeavoring to interpret test material. The Inter-Society Cytology Council has gone on record as not favoring the aforementioned Cytology Foundation.

GEORGE G. STILLWELL, M.D.
Chairman, Editorial Committee

DIABETES INSIPIDUS AND PREGNANCY

(Continued from Page 811)

4. In conclusion, diabetes insipidus is a rather infrequent complication of pregnancy, and the disease itself seems usually of obscure etiology.

5. The course of diabetes insipidus during and following pregnancy is rather unpredictable.

6. At least during pregnancy, the preferred treatment seems to be pitressin injections, because of the absence of oxytocic effect.

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Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

PHYSICAL MEDICINE

Physical medicine, in a large measure, is concerned with the treatment of chronic diseases or disabilities in which recovery may be long delayed. As such, it has frequently been neglected by the general practitioner who is busy with acutely ill patients. Yet, it has been estimated that up to 40 per cent of clinical visits are for conditions which need treatment by physical therapy.

Physical therapy to traumatic injuries, arthritis, neurological disabilities and other conditions, when they are first seen, accelerates recovery and helps to prevent the development of a chronic disability. The methods which can be used in an office for treatment of these conditions should be a part of the armamentarium of every general physician.

Approximately one-fifth of the patients in general hospitals need treatment by physical medicine. Patients who remain in bed for any length of time show progressive deterioration of the cardiovascular system, progressive weakening of the muscular system, demineralization of the skeletal system, development of contractures, and loss of motivation to be independent. Of all these, loss of motivation is the most serious, for without motivation to be independent no patient can be successfully rehabilitated. When patients must remain in bed for prolonged periods, a program of activity should be planned commensurate with their capacities. Occupation therapy provides medically supervised activities at any energy level to maintain the patient physically and mentally. The nurses should maintain the range of motion of all of the joints and supervise posture to insure that contractures do not develop. Physical therapy with all of its forms of treatment hastens recovery and prevents deterioration of the patient. The patient should be taught and required to do for himself the activities of which he is capable. These include independence in moving in bed, eating, getting out of bed, moving from chair to chair, dressing, standing and walking. The longer the patient remains dependent the more difficult is it for him to re-establish independence.

The goal of medical practice is to restore all patients to health as completely and quickly as possible. Physical medicine provides some of the tools necessary to achieve this goal.

FREDERIC J. KOTTKE, M.D.

PSYCHIATRIC CONSULTATION IN SOCIAL AGENCIES

Social agencies give many tangible services to families and individuals, such as financial assistance, homemaker service and child placement. But if there is severe stress in family relationships or if any family member is seriously at odds with others or with himself, both the need and the service required are less tangible.

Whenever such a situation comes to the attention of a social agency, the social worker seeks to determine whether or not the person or family in question has enough social adequacy and personality strengths to use counselling, a child guidance approach, or such other forms of a helping relationship as may be appropriate. Much of the skill of the social worker has been developed in this area of relationships. If it is found that the client cannot enter into a meaningful and constructive relationship with the social worker, questions as to the precise nature of his emotional problems and personality structure demand thorough-going attention. It may be that the client is mentally ill or severely neurotic or has a deep-seated personality problem. With a clearer understanding of the behavior of such a client and of his family relationships, the social worker may be able to give the correct kind of support and guidance to him and to his family. Or, referral to a private psychiatrist, a mental hygiene clinic or a hospital providing psychiatric care and treatment may be the only service the social agency is equipped to offer.

To clarify these problem situations, social agencies have developed the practice of employing a psychiatrist, usually on a part-time basis, to give consultation to members of its staff, as need arises. The social worker presents the problem case in detail to the psychiatric consultant,

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Third in a series of editorials on Family Service.

and the handling of it to date is discussed. Ordinarily, the psychiatrist does not see clients. He evaluates the worker's experience with the case and contributes his understanding of psychiatric elements appearing in the total situation as it is presented to him. He may support a continuance of the existing plan of social treatment or suggest changes. He may point out that, from the social history, it appears likely that a certain member of the family is mentally ill and recommend a psychiatric examination. In all cases presented to him, the psychiatrist acting as consultant provides certain safeguards, professional guidance and rich learning opportunities for the staff of the social agency in directions extending beyond their proven areas of competence.

KATHERINE H. TINKER, *Supervisor*
Family Service of Saint Paul

SEPARATE FUND-RAISING

The established policy of the volunteer Board of Trustees of the National Foundation specifies that a separate March of Dimes is the only public appeal for funds to finance the work of the organization. The 3,100 local chapters of the National Foundation are required by charter to adhere to this policy of separate fund-raising and may not participate in any joint, united or federated fund-raising activities, or accept any funds raised by this method of solicitation.

Joint, united or federated fund-raising is interpreted by the National Foundation as any single solicitation of funds within a community, plant, institution or organization for the benefit of more than one organization.

There are six basic reasons for this National Foundation policy of separate fund-raising:

1. *Public Awareness.*—The recent tremendous advances in the field of polio prevention would not have been possible without a separate March of Dimes. Better than anything else, the March of Dimes tells the story of polio to the American people, helps make them aware of polio and educates them to fight the disease. In any joint type of fund-raising, the polio problem might lose the attention it deserves and the conquest of the disease might be postponed for years.

2. *Fluidity of Funds.*—A separate March of Dimes is essential because conditions all over the country have to be met as they arise and this requires a complete mobility of funds, equipment and personnel. In any joint fund-raising, local committees—unfamiliar with the complex nation-wide activities of the National Foundation—would pass upon its annual budget. Any arbitrary regulation of expenditures — which would be inevitable under a federated fund-raising program—would hamper continuation of the National Foundation's long-term research and professional education programs.

3. *Opportunity.*—A separate March of Dimes offers an individual a chance to be a part of a great humanitarian program at a very small cost. A dime or a dollar is a trifling sum in itself but, given a nation-wide drive, this same dime or dollar helps sponsor research that may mean the end of a crippling disease. Multiple drives offer an individual a variety of opportunities where his small sum can do a great good. If a person does not choose to take the opportunity to do good, he need not give.

4. *The American Way.*—A separate March of Dimes is based on the American tradition of freedom of choice. Each January, Americans have the right to endorse or withhold their support from the March of Dimes on the basis of their approval or disapproval of its program. Last year, about 75,000,000 Americans chose to contribute to the fight against polio. It also is in keeping with the American tradition that each organization should be free to decide whether it will join any federated or group fund-raising program.

5. *Volunteers.* — Each separate drive draws upon civic-minded citizens for volunteers; a separate March of Dimes gives an individual a worthwhile activity in which he can participate along with neighbors. This is perhaps the most effective picture we have of democracy at work at the grass-roots level. Many March of Dimes volunteers serve throughout the year, aiding families and communities hit by polio. Volunteers from the Mothers' March on Polio helped make the Vaccine Validity Trials of 1954 successful by aiding doctors, nurses, teachers and health officers. Joint fund-raising narrows community participation in welfare, reduces opportunities for volunteers and closes valuable avenues of leadership.

6. *Success.*—In 1938, the public gave the first separate March of Dimes a gross of over $1,500,000 to fight polio. In 1954, it gave a separate March of Dimes a gross of $55,000,000 for a comparable drive. This is an unprecedented accomplishment for a truly voluntary health agency—not because of the amount raised, but because of the vital work that this money made possible. The National Foundation does not exist to raise money—it raises money to exist so that it may carry on its work. The National Foundation, now on the threshold of conquering a crippling disease, cannot abandon this successful method of separate financing.

DESIGN AND DECORATION FOR THE DOCTOR'S OFFICE

With a few pleasant exceptions which, happily, are increasing in number, doctors' offices have never been noted for relaxing comfort and good taste in interior design, color and furnishings. One might almost say that these elements are out-dated only by the magazines on the reception room table.

Because most people who go to doctors' offices are disturbed and would rather be some place else, it would seem that making them as comfortable and happy as possible while there would be to show not only decent consideration but also good business sense. Most would rather build themselves up for a visit with an Albert Schweitzer than a Torquemada.

The doctor's office is a problem in design and decoration which involves skilled diagnosis and treatment by a professionally trained person. Turning it over to your receptionist, your office assistant, or even your wife—because it needs the well-known "woman's touch"—is tantamount, generally speaking, to turning over a lobotomy to the pharmacist at the hospital.

The doctor's office is not "home" to anyone; it is a semi-public, professional premise and it should be treated as such in matters pertaining to style and color. There is a firm and significant trend towards what is known as "contemporary" design in the decoration of business and professional offices. The fact that you and most of your patients may live in houses that you fondly imagine are "period" or "traditional" in style may be all the more reason for "going modern" in your office. Contemporary design need not be mechanistic, impersonal, sterile or coldly clinical—epithets which have been leveled at it by the nonunderstanding. It *can* be warm, inviting, stimulating—and good advertising, as well. (Advertising, being by definition a four-letter word, how did it get in here? Let's just say it's nice if people notice your pleasant office the way they notice your "bedside manner" and tell other people about it.)

Color, whether used for purely decorative appeal or, more purposefully, as a very functional tool, has assumed tremendously increasing importance in recent years. Although medical literature contains many references to color therapy, these are perhaps to be heavily discounted. But as to its psychological and emotional effects on people, enough is known to allow us to put it to good use in business and industrial situations. One would be reactionary, indeed, to ignore its functional possibilities and continue to treat it merely as pretty to look at. A well-integrated interior design job in the doctor's office calls for a working knowledge of the effects of color on people psychologically, as well as aesthetically.

The successful doctor is not the one who hung up his diploma five years ago, or twenty, and then forgot about studying and learning any more. It's competition, as well as a thirst for improvement that keeps him reading, attending lectures, going to conventions and constantly striving to improve his techniques. Many will hesitate to admit the existence of competition in the professions, but it's there. Some businesses—banks, for example—used to feel it undignified to go after business aggressively. Now, they're merchandising to the limit the services they offer. It may not be amiss for the doctor to divest himself of the "better mousetrap" psychology and begin doing a little merchandising.

One way he can, within the framework of professional ethics, is to make his place of business look like a good place to come to and feel like a nice place to be.

Clarence Deutsch
Color Counselor

ANCILLA, -AE, *f. dim.*

What with all the energy expended to make meanings clear, or to slant words, phrases, sentences, and whole books, we were pleased to have cognizance taken of the misuse of *ancillary** in a current issue of *The Journal of the Medical Society of New Jersey. Ancilla* is a diminutive of a Latin noun meaning servant, specifically, maid-servant. In its evolution into our word *ancillary,* all that might be pejorative has survived in most current usages, and none of the faithfulness or other desirable characteristics of a good maid-servant remains. The tag *ancillary services* thus becomes tinged with servility, if not the scullery.

Decades ago someone relegated the practice of anesthesiology, pathology and radiology to this scullery with the inept complaint that the "faculties of the physician were being subordinated to the ancillary services. . . ." Perhaps he was wroth and used ancillary to express his meaning precisely, but the phrase stuck, despite the physical removal of laboratories from the cellars and attics of medical installations to central locations, and the development of these specialties into necessary parts of the modern practice of medicine. Unfortunately, these specialties have remained ancillary in the thinking of some poorly informed folks; e.g., in the current dispute between Iowa physicians and Iowa hospitals, a Dr. Letourneau has tried to uphold the hospital's proprietary interests in the practice of anesthesiology, pathology and radiology by testifying that pathology and radiology are not the practice of medicine, but are only fact-gathering methods (one of the bitterest jokes in teaching x-ray departments the country over is the widespread impression among laymen that x-ray examinations take all the guess-work out of diagnosis)!

Of necessity, various specialties in medicine must play secondary roles when circumstances require it. The patient consults *his* physician and from thenceforth, all other physicians consulted are auxiliaries, though not subsidiaries. The patient's physician is the one who integrates the diagnostic and therapeutic parts other physicians play in the solution of that patient's problem. Such a procedure is simply the day-in and day-out cooperation for the welfare of the patient at the foundation of medical ethics. However, a secondary role whenever the circumstances require it, and the rendition of only ancillary (we remind you, ANCILLA, -ae, *f.*: Maid-servant) services are vastly different. And to be labeled thusly by confreres is doubly derogative.

You are not affected, you think; but if any group of physicians gets lost in the welter of non-physicians' trying to dominate the practice of medicine, we all lose. We could do no better than to end this lament the same way Dr. Demy ended his erudite comments, with the Devotion of John Donne:

> No man is an Iland, intire of itselfe; every man is a peece of the Continent, a part of the maine; if a Clod bee washed away by the Sea, Europe is the lesse, as well as if a Promontorie were, as well as if a Mannor of thy friends or thine owne were; any man's death diminishes me, because I am involved in all Mankinde; and therefore never send to know for whom the bell tolls; it tolls for thee.

H.G.M.

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*Demy, Nicholas G.: The Ancillae, J. Med. Soc. of New Jersey, 52:419, Aug., 1955.

In Memoriam

JACK FRIEDMAN

Dr. Jack Friedman, Minneapolis radiologist, died September 29, 1955. He was clinical assistant professor of radiology at the University of Minnesota and a radiologist at Mount Sinai Hospital in Minneapolis. He was forty-four years old.

Dr. Friedman attended the University of Illinois College of Medicine and interned at Lutheran Deaconess hospital in Chicago. He took postgraduate work in radiology at the University of Minnesota.

A member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association, Dr. Friedman also belonged to the American College of Radiology, the Radiological Society of North America, the American Roentgen Ray Society, and Sigma Xi. He was a member of Temple Israel.

He is survived by his wife, Dorothy, and his daughter, Elizabeth, Minneapolis; his parents, Mr. and Mrs. Louis Friedman, Chicago; two brothers, Irving and Leon, and a sister, Adele, all of Chicago.

President's Letter

SOMETHING GOOD

At this time of year, when America's bounty should make us very thankful, I feel it appropriate to quote something said by one of our elder statesmen, Herbert Hoover, on his eighty-first birthday:

> "During the last score of years our American way of life has been deluged with criticism. It comes from our own people who deplore our faults and genuinely wish to remedy them . . .
>
> "Criticism comes daily from the habit of our political parties to deplore the opposition. It arises from the forthright refusal of the American people to wash their dirty linen in secret. It comes from our love of sensational incidents where villainy is pursued by law and virtue triumphs . . .
>
> "Altogether, if we look at the criticism alone, we seem to be in a very, very bad way and engaged in our decline and fall.
>
> "But we should lift our eyes unto the hills from whence cometh our help. We should occasionally mention something good about ourselves.
>
> "We could point out that our American way of life has perfected the greatest productivity of any nation on earth; . . . We could point to our constantly improving physical health and lengthening span of life. We could point out that the mechanical genius of our people has, by millions of labor-saving machines, taken the sweat from the backs of most of our people.
>
> "In the governmental field, we could suggest that our supposedly decadent people still rely upon the ballot and the legislative hall to settle their differences without a secret police with slave camps.
>
> "In the cultural field, we could point out that, with only 6 per cent of the world's population, we have more youth in our institutions of higher learning than all the rest of the world put together. We could probably enumerate more libraries and more printed serious words than all other 94 per cent of the earth put together.
>
> "On the moral and spiritual side, we have more hospitals and charitable institutions than all of them (the nations). And we could suggest that we alone, of all nations, fought in two world wars and asked no indemnities, no acquisition of territory, no domination over other nations.
>
> "We could point to our advancement of the spirit of compassion. We could prove it by the billions of dollars we have made as gifts to save millions from famine and governments from collapse.
>
> "Much as I feel deeply the lag in giving an equal chance to our Negro population, yet I cannot refrain from mentioning that our 14 million American Negroes own more automobiles than all the 200 million Russians or the 200 million Negroes in Africa.
>
> "All of which is not boasting, but just fact. And we could say a good deal more.
>
> "What does all this mean? It means that freedom of mind, of spirit and of initiative still lives in America. It means that our people are strong in religious faith. Here alone are the open windows through which pours the sunlight of the human spirit. Here alone, even with all its defects, is human dignity not a dream but an accomplishment.
>
> "These ideals of freedom and religious faith guarantee there will be no decline and fall of American civilization."

Yes, we have much for which to be thankful.

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

SURVEY SHOWS PUBLIC LIKES M.D.'S

The public generally likes Doctors of Medicine and understands the problems facing the profession today. This was brought out recently by a public opinion survey sponsored by the Los Angeles County Medical Association. The poll was conducted this summer.

The survey showed that doctors as a group enjoy a position of confidence and respect. According to a recent report in *The Bulletin of the Los Angeles County Medical Association,* "this finding differs markedly from the results of other recent opinion samplings carried out in scattered areas of the nation which indicated widespread 'public hostility' toward the medical profession." The survey was based on interviews with 360 adults in Los Angeles County.

The Bulletin article points out, however, that there are many areas in which the public has insufficient information and others in which medical public relations could be improved. But, one compelling fact emerges from all the data, the report states: The public is *not* dissatisfied with physicians and their services today!

The Bulletin quotes one interviewee as saying:

> ". . . doctors didn't invent sickness and misery. I think they are doing their best to do a job that has to be done. They are in a tough spot to have good public relations. Their 'customers' just don't *want* to buy what they are selling at any price. Even if doctors could fix broken legs for a dime a dozen, no one would want to have one. Nobody likes to be sick. And many people—when they get sick—get sore and take it out on the doctor."

Other important findings were:

1. 75.1 per cent had a regular or family doctor.

2. 91 per cent stated that the last time they had seen a physician he had made entirely clear all that they wanted to know about their illness.

3. 95.2 per cent felt that the M.D. had given them all the time they needed for diagnosis and treatment.

4. 85.5 per cent called the doctor's attitude toward them, "kindly, sympathetic," and definitely not calloused or impersonal.

5. More than half, 54.3 per cent, had changed physicians because of dissatisfaction. And the principal reasons for dissatisfaction were, in order of their frequency: "Not thorough;" "Didn't like his medication;" "Not a specialist;" "Charged too much."

6. 77.5 per cent believed that an osteopath is a medical doctor who has specialized.

7. 83 per cent of the public confused internists with internes.

8. 35 per cent had undergone surgery in a Los Angeles hospital.

9. 76.5 per cent believed that all doctors would make night or Sunday calls if humanly possible.

10. Only 15 per cent of those who called a physician in Los Angeles found he couldn't take the case. And 61.3 per cent of this group were of the opinion that the doctor was doing his best but was just too busy.

11. When patients were referred to doctors other than their own in emergencies, 70.8 per cent were entirely pleased with the substitute and 26.1 per cent stayed on with the new physician.

12. 86.5 per cent were positive that physicians never undertook procedures they were not qualified to perform but always referred patients to appropriate specialists.

13. The public generally was of the opinion that "all" doctors were more competent to diagnose different diseases than to perform different surgical procedures.

14. 64.2 per cent rely on the recommendations of a friend in selecting a physician.

15. Most persons were lenient about waiting for the doctor to keep his appointment. 82.8 per cent felt the M.D. was justified in running behind schedule in his appointments while 10.5 per cent thought this was "unreasonable." 22.6 minutes was the mean amount of time the respondent expected to wait. 4.3 per cent considered an hour or more "usual."

16. Based upon their own personal experience, 88 per cent were entirely satisfied with physicians' fees. However, 30.2 per cent were of the opinion

that medical fees "generally" are "unreasonable." The reason most often given for this was, "The average working person can't afford the fee."

17. 74.3 per cent were more interested in having their hospital bill covered by insurance than in having the doctor's fees covered. This percentage rose to 86.4 per cent with community leaders and influence groups.

18. 74.8 per cent had the doctor's professional education pegged right—eight or more years of study.

19. 76.4 per cent were sure that they had far more leisure than an M.D. 73.4 per cent felt that doctors worked harder than they did.

20. 50.2 per cent are of the opinion that doctors send their patients to particular pharmacies. And 33.9 per cent think that the physician gets a "rebate" from the pharmacy.

21. 83.7 per cent praised the medical profession for informing the public regarding "new methods, treatments and medicines."

22. 82.1 per cent wanted no part of compulsory health insurance. The percentage went up to 91.5 per cent with the influence group.

23. 59.8 per cent preferred "service type" insurance. 28.8 per cent wanted "indemnity." 7.4 per cent like "closed panel" coverage.

24. 70.1 per cent want itemized bills and 77.8 per cent prefer a letter to a phone call as a reminder for a delinquent account. 37.9 per cent considered it completely unjustified to have delinquent accounts turned over to a collection agency.

The questions in the survey were asked of two groups—the general public (309) and the influence group (fifty-one). The influence group was selected arbitrarily to represent as nearly as possible a cross section of those persons whose position put them in a category of leadership. They included representatives of labor, social groups, teachers, clergy, business and industry, State assemblymen, State senators, city councilmen, and the Los Angeles Board of Supervisors.

AMA STUDIES MEDICAL CARE ECONOMICS

A recent issue of *The Journal of the American Medical Association* reports on a review of the economic position of medical care over the past quarter century made by the Bureau of Medical Economic Research.

According to an AMA editorial, "The findings emphasize the 'middle ground' position of the medical care 'industry' during that period, 1929-1953, when gross national product rose from 104 billion to 365 billion dollars and personal consumer expenditures rose from 79 billion to 230 billion dollars." The editorial continues:

> "While dollars expended for medical care followed the over-all pattern of expansion, the Bureau notes that since 1930 the percentage of personal consumer expenditures going for medical care varied only slightly, from 3.99 to 4.35. Since World War II, however, the percentage has risen at a more rapid rate, from 4.14 to 4.35."

Effect of Birth Rate

One factor that increased personal consumer expenditures for medical care, in dollars and as a percentage of the total, the editorial says, was the tremendous upswing since World War II in the number of births and the birth rate. The increased number of babies and young children has affected not only hospital and obstetric expenditures but has increased the demand for other medical services, since this youngest age group normally requires the most frequent medical attention. The editorial notes this effect:

> "In the long run, of course, this imbalance will be corrected as today's toddlers become tomorrow's productive adults. Moreover, the reduction of the maternal mortality rate to a very low level has contributed to the high birth rate by reducing the mortal fear of childbearing almost to the vanishing point. The economic effect of the 1,200,000 increase in births from 1945 to 1953 has been obscured by statistics that do not show a division of medical expenditures into those for childbearing, disease, and accidents. Without such a breakdown, it is easy to misinterpret the increase in medical expenditures by ascribing it to a decline in the health of the American people or a disproportionate rise in medical care costs."

Second Factor Isolated

The editorial goes on to say that the Bureau study has isolated as a second factor responsible for the increase in expenditures the item "medical care and hospitalization expense." These expenditures represent premiums less claims paid; this difference is primarily overhead cost. "The rapid growth in voluntary health insurance has resulted in an increase of 779 million dollars in this insurance overhead item from 1929 to 1953; this amount represents one-ninth of the total 7,064 million dollar increase for all medical care items. The increase in overhead costs is, of course, a price that must be paid for the rapid extension of voluntary health insurance."

Prices Studied

The index for medical care and drug prices was studied for this period. In 1954 it stood at 173.9, the period of 1935-1939 equalling 100. This is well below the 191.9 for the entire index, based on the U. S. Bureau of Labor Statistics' Consumer Price Index. And, the editorial continues, the item indexes for physicians' and dentists' fees were still lower—160.3 and 174.5. "The only component of the total medical care and drug index that showed a marked rise, the editorial states, "was hospital room rates, which was 322.1 in 1954. If the upward effect of this component were eliminated, the over-all medical care and drug index would be still lower."

Reporting on the physician's share in the medical care dollar, the editorial says that a dollar spent for medical care in 1953 was not divided among the suppliers of these services and commodities in the same way as was a 1929 dollar. It states:

> "The 33 cents that the physician received in 1929 dwindled to 28 cents by 1953; the share of the dentist and that for drugs and sundries also declined. The compensatory increases were for all other medical care (which includes the rapidly growing insurance overhead item) from 17 to 20 cents; and for hospitals, from 14 to 26 cents. If these trends continue, the hospitals' share will exceed the physicians' share by 1960."

Rise in Productivity

Output per physician has risen, the editorial notes, but this has counterbalanced the lag in physicians' fees and the decline in the physicians' share of the medical care dollar. Thus, the effective supply of physicians' services has increased much more rapidly than the number of physicians, the editorial emphasizes. "Also, seeing a decreasing percentage of patients in their homes has resulted in a saving of the physician's time. Use of more auxiliary personnel in his office and in the hospital and use of more effective drugs have also been major factors in increasing the technological efficiency of the physician. As a result, the average income of physicians rose at almost exactly the same rate as the average earnings of all wage and salary workers over the period. . . . The incomes of physicians and their patients continue to keep pace.

Efficient Industry

Noting these over-all trends and their effect on the over-all medical care picture, the editorial concludes:

> "The various providers of services and commodities in the medical care 'industry' can well be proud of a quarter century record that included great medical advances—the increased life expectancy at birth, the declines in infant and maternal mortality, and the development of new drugs and treatments—while little change occurred in the industry's relative economic position. Possibly this is our most efficient industry!"

AMERICA CALLED MEDICAL MAGNET

A recent news release from the American Medical Association states that the United States has become a "medical magnet" for physicians in Europe, Asia, Africa, and Latin America.

"More than 5,000 foreign physicians came to this country during the year 1954-55 for study," the release says. This information was brought out in a survey by the Institute of International Education and the American Medical Association.

The survey notes that the physicians came from eighty-three different countries for internship and residency training at hospitals in forty-two states, the District of Columbia, Hawaii, Puerto Rico, and the Canal Zone. Those countries sending the most doctors to America were the Philippines, Canada, Mexico, Germany and Turkey.

Resources Checked

The survey reported that more than 2,000 of these physicians were in the United States on their own resources. Others were sponsored by at least sixty-seven different agencies, including their own or the United States government, the United Nations, and religious, educational or philanthropic organizations. Many were sponsored by the hospitals in which they were training.

The survey also notes:

> "In addition to the large number of physicians in hospital internship-residency training, others visited this country as observers, professors, or guest participants in research. They represented 21.5 per cent of all foreign educators who visited the country during the year.
>
> "In comparison, only 3.6 per cent of all American educators visiting other parts of the world in 1954-55 were listed under medicine."

The Dean's Page

THE FALL SEMESTER BEGINS

The Medical School has started upon another year's activities. One hundred and twenty-eight new students have enrolled as freshmen and four new students have been accepted into the junior class as transfers from other medical schools.

One half day at the opening of the year was devoted to a program of Welcome and Orientation for the freshman students. Talks about the Medical School and about the opportunities and obligations of medical students were given by the Dean and by the Assistant Dean, William Maloney; Dr. Cecil Watson, Professor of Medicine, spoke on "The Relation of the Basic Sciences to Clinical Medicine"; Dr. Donald Hastings, Professor of Psychiatry, on "The Personal Relationship of Student, Physician, and Patient"; senior student Ralph Swanson discussed medical student activities and the Medical Student Advisory Council; and senior student Duane Flogstad spoke on "The Student American Medical Association."

After a break for coffee and sociability, the freshman students reassembled in the Mayo Memorial Auditorium with the students of all other classes, members of the faculty and of the Minnesota Medical Foundation. Dr. Wesley Spink, President of the Minnesota Medical Foundation, presided at this assembly and presented scholarships to six freshmen, five sophomores, seven juniors, and three seniors. These scholarships were provided by the Minnesota Medical Foundation, and were awarded on the basis of financial need and of promise as a successful medical student and physician. Until a few years ago, the University of Minnesota offered no scholarships for medical students. Today, these scholarships are proving invaluable in enabling a group of well-qualified young men and women not only to study medicine, but also to profit by the opportunities for a medical education which are available to them.

Following the presentation of scholarships, the Dean spoke on "Health as an Instrument of International Policy." Immediately following the adjournment, the Minnesota Medical Foundation held its annual meeting at a luncheon in the Junior Ballroom of the Coffman Memorial Union.

The orientation program for freshmen and the general Medical School Assembly constituted a splendid and thrilling beginning for another year of effort and achievement on the part of students, faculty, and friends of the Medical School. The construction of the Mayo Memorial Auditorium makes possible these assemblies which are of inestimable value in fostering student-faculty team work and esprit de corps in the Medical School. I only regret that all of the physicians of the state could not have shared this splendid occasion with us.

Harold S. Diehl
Dean, The Medical School
University of Minnesota

Letters ▲ ▲ ▲

Dear Editor:

The other day I ran into an abstract of an original paper in MINNESOTA MEDICINE, 37:862, 1954, by Dr. R. E. Semsch. The paper recommends a universal antidote for children, the preparation consisting of charcoal, two parts, magnesium oxide, one part, tannic acid, one part. I would like to call your attention to two matters with regard to this formula which should not be given such widespread publicity without considering the scientific facts.

Charcoal varies enormously in its adsorptive properties. Perhaps in the full paper, the author describes the type of charcoal to be used, otherwise the statement "charcoal" is meaningless. The second or major part of my criticism is that the author compounds two incompatibles, magnesium oxide and tannic acid. It is elementary chemistry to know that these two compounds react promptly to form magnesium tannate and I have not taken the trouble to determine the amount of magnesium oxide which would be left over after the interaction of these two chemicals, but it is obvious that there would be no tannic acid. Instead, the author's mixture would contain charcoal, magnesium tannate and magnesium oxide, and therefore it is entirely improper for him to state that the tannic acid will precipitate alkaloids, glucosides and some heavy metals. Perhaps magnesium tannate will do so, but this would have to be proved in the laboratory.

The only reason I am writing this letter is in the interest of scientific accuracy, for the type of loose statement used in the paper is not in accordance with established facts in chemistry. After all, when one mixes hydrochloric acid and sodium hydroxide, one loses entirely the properties of one or the other compound, or both, depending upon which is in excess. The chemical reaction produces common salt, which is of course entirely unlike the chemicals from which it is formed.

Very truly yours,

HERMAN H. KAHLENBERG, PH.D.

Sarasota, Florida
October 3, 1955

* * *

Dear Editor:

Summer vacations offer the physician in practice a chance to muse about events during the year preceding, and no event will be mused upon by more physicians than the coming of the Salk vaccine against poliomyelitis. Beclouded though the subject is at the moment, there is ample reason to believe that the doubts will be settled and the questions will be answered, and within a reasonable time the new tool will take its place in routine practice.

The raising of such doubts and questions in the first place, as they have been raised in the press and in the minds of the public leads the muse a little further, though, and the doctor finds himself assuming a posture of distressed dissatisfaction with some parts of the situation. Furthermore, he senses that his posture may easily be misunderstood by the same American public which at first demanded the vaccine in a hurry, and then began to telephone family doctors to find out if the vaccine was safe. It would take only what the press agents call a small "switcheroo" to make the doctor the fly in the public's ointment at the present time. One's comments about such a "hot" subject had better be circumspect if they are likely to be quoted from one patient to another. Now, how is it that this situation came to be?

To come to the point quickly, this is the first time that public demand for a medicament has ever been created by such a large and careful campaign of press releases. The campaign was started before the medicament was manufactured, before it was tested for safety, before scientific information concerning it was published in professional journals. But these are really minor breaches of good medical policy. The doctor wonders if it is right to advertise any remedy. It never has worked well for the public interest in the past. Should there have been any campaign of press releases in the first place? Doesn't such a campaign remove the element of professional judgment from the administration of the injections? The answer is "yes" of course, and we still do not know enough about the material to know whether a small grain of professional judgment ought not enter the decision to use the vaccine. A highly intelligent cancerophobe asked the writer whether Salk vaccine sensitized one to monkey kidney. If it did, he wanted none, for future cancer therapy (for his as-yet nonexistent cancer) might thereby be proscribed for him.

The only justification for such advertising of a medicament that occurs to one readily is that it is required in order to get a testing situation set up which will contain the needed number of individuals. This is not the case here, for the testing supposedly is finished, and was going on when the campaign started. One must conclude that the press releases were not required by the scientific part of the situation.

One muses along a bit farther to the realization that the National Foundation for Infantile Paralysis conducted its publicity campaign in order to raise money which has been used for polio care and for polio research. Both are blameless causes. Both have been helped by advertising a not-yet-ready vaccine. But does this justify advertising a medicament? Bear in mind that the free advertising via press releases was voluntarily sought out by the National Foundation. But remember also that this is not a single doctor and not a medical society, and therefore not governed as doctors and medical societies are governed by an ethical code which forbids advertising. There has been no ethical breach. The National Foundation merely has to follow its own rules and the laws under which it was incorporated; it is not subject to a code of ethics. It is not practicing medicine though there are those who now are questioning this point. Actually the conduct of a sizeable chunk of medical practice in 1955 has been determined by the policy of the National Foundation. The Foundation has created a situation in which thousands of doctors have injected millions of children with a substance to them unknown. No one need quibble about its being unknown; the unexpected cases of polio which followed its use proved that it was unknown.

The present events are not the first which have arisen as a result of the National Foundation for Infantile Paralysis. In 1953 the Foundation promoted gamma globulin with equal energy, enlisted co-operation of the Red Cross and health departments, and spent a large sum of money on a tool which we know now to be of little value. It could have been shown useless without trying it out on a national scale, and the money could have been saved. The same sense of urgency filled all the press releases in 1953 as have been noted in 1955. It may be rememberd by some that the Foundation has had previous experience with vaccine as well. It is an experienced agency as well as a large and rich agency. Its performance with Salk vaccine probably may be regarded as what one should expect from such a quasi-medical, quasi-public agency.

Thus one muses along some more and into the field of the relationship of the practicing doctor to such agencies. They exist in some numbers. They have large funds, some of them to be spent for what is called education. Other terms for education in this sense are "public relations," "lobbying," "public education," and the providing of "educational materials." We have before us now an example of the way such public education may go astray.

These medical foundations will continue to award research grants, and we now know, will take research results direct to government units for testing in the field as well as for distribution. We now know that they will drop responsibility into the lap of the Federal government if the going gets rough or if controversy arises. They are subject to technical medical guidance, usually of the most brilliant caliber. But what is the relation between the practicing doctor to these agencies? Is there a way for the practitioner to steer agencies like these away from pitfalls like premature advertising which he has known for generations to be a pitfall? Would the suggestions of the practitioner be welcome, and would they be heard?

Individual participation by thousands of doctors in hundreds of organizations, agencies and Foundations seems not to work, and a priori it is impractical. Opposition to the agencies is not only not in the public interest, but futile. They include in membership, great numbers of earnest, well-educated, serious people of the greatest integrity. The difficulty seems to be that they have a propensity for gigantic boo-boos which are also not in the public interest. These arise because agencies are not accustomed to medical practice, and their good works require medical practice to be applied.

Where does the answer lie, then? It must be admitted that the answer isn't new: the relation of agencies to practicing physicians must be a function of the American Medical Association, the State Medical Associations, and the County Medical Societies. The change which might make medicine's efforts more fruitful is to take the initiative. Make the county societies' programs more topical and discuss the policies of the local units of the agencies, as well as the national policies. Stir up the inactive State committees to go and deal with the Foundations that they were appointed to treat with. In the national AMA offices, raise more money from the membership so that more national officers can take a year away from practice and travel the country and counsel with and advise Foundations, agencies, and quasi-medical organizations. These groups are a part of the medical picture in the American community, and all the practicing doctors must learn where they fit, what they can do, and how they can be helped. If we are to avoid another premature release or other miscarriage of effort from this or another agency, we must take our money in dues and assessments and use it to carry the suggestions and advice of practicing physicians to the agencies.

Organized American medicine must share in the blame for the polio vaccine embarrassment. We in practice should have pressed our advice harder. If it seemed unwelcomed by the National Foundation for Infantile Paralysis officials, the same team should have taken the same time and money and brought the advice to the physicians instead, so that when the vaccine was announced the doctors who have to administer it would have known pretty well what they were to handle.

This is only the first episode of the sort on this scale. A good blood or urine test for cancer may turn up tomorrow. Will we be ready to show our investigative colleagues how it may be applied best for the good of man? More people fear cancer than fear polio.

Herbert Plass, M.D.

Minneapolis, Minnesota
August 27, 1955

* * *

Dear Editor:

We have read with interest your letter to Mr. Elder of our St. Paul District Office dated June 7, 1955, in which you comment as to the need for standardization of medical report forms being used by an increasing number of agencies.

Your observations in this matter are of special interest since we have been actively engaged in analyzing medical report forms with representatives of life insurance companies, State medical societies, and with a Medical Advisory Committee appointed to assist the Social Security Administration in the formulation of sound medical standards for the evaluation of disability. The focus of these deliberations has been toward a standardized form that will elicit a minimum of essential medical data with the least drain on the individual physician's time.

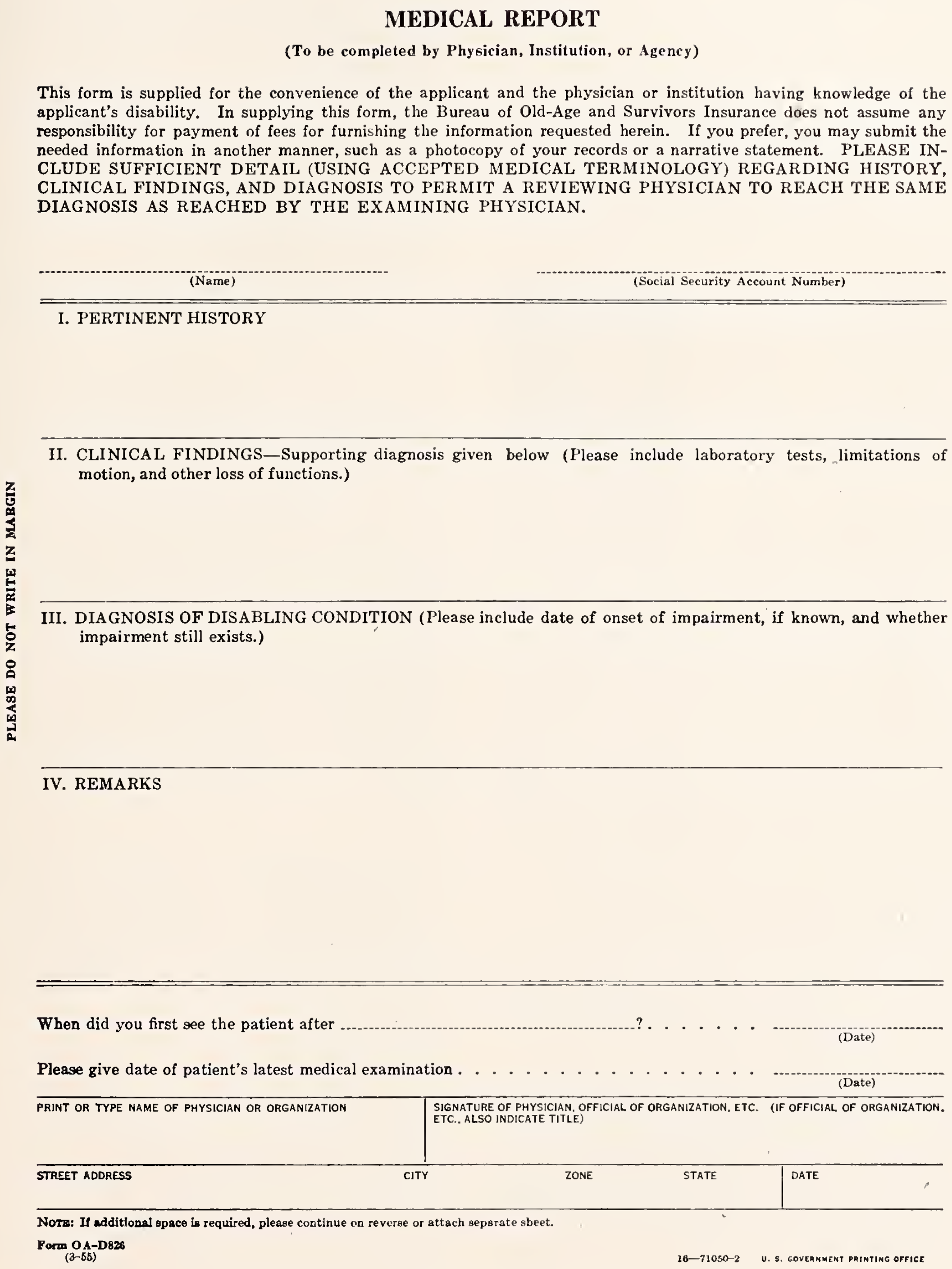
As our negotiations with these interested groups continue and as we gain further experience with the medical report form currently in use, the whole matter will come up for reappraisal by the Medical Advisory Committee in the fall. Because of your expressed interest in this area, we are enclosing a copy of the medical report form and earnestly solicit your comments and suggestions with respect to it. You will note that it has been kept brief but, if properly executed, should provide the necessary medical information.

Since the quality of response to our requests for medical data depends so much on the good will and co-operation of the medical profession, we are considerably heartened by your constructive interest in the program.

Sincerely yours,

Arthur E. Hess
Assistant Director
Department of Health,
Education and Welfare

Baltimore, Maryland
July 15, 1955



PLEASE DO NOT WRITE IN MARGIN

MEDICAL REPORT

(To be completed by Physician, Institution, or Agency)

This form is supplied for the convenience of the applicant and the physician or institution having knowledge of the applicant's disability. In supplying this form, the Bureau of Old-Age and Survivors Insurance does not assume any responsibility for payment of fees for furnishing the information requested herein. If you prefer, you may submit the needed information in another manner, such as a photocopy of your records or a narrative statement. PLEASE INCLUDE SUFFICIENT DETAIL (USING ACCEPTED MEDICAL TERMINOLOGY) REGARDING HISTORY, CLINICAL FINDINGS, AND DIAGNOSIS TO PERMIT A REVIEWING PHYSICIAN TO REACH THE SAME DIAGNOSIS AS REACHED BY THE EXAMINING PHYSICIAN.

(Name)

(Social Security Account Number)

I. PERTINENT HISTORY

II. CLINICAL FINDINGS—Supporting diagnosis given below (Please include laboratory tests, limitations of motion, and other loss of functions.)

III. DIAGNOSIS OF DISABLING CONDITION (Please include date of onset of impairment, if known, and whether impairment still exists.)

IV. REMARKS

When did you first see the patient after ....? . . . . (Date)

Please give date of patient's latest medical examination . . . . (Date)

PRINT OR TYPE NAME OF PHYSICIAN OR ORGANIZATION	SIGNATURE OF PHYSICIAN, OFFICIAL OF ORGANIZATION, ETC. (IF OFFICIAL OF ORGANIZATION, ETC., ALSO INDICATE TITLE)			
STREET ADDRESS	CITY	ZONE	STATE	DATE

Note: If additional space is required, please continue on reverse or attach separate sheet.

Form OA-D826
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(See letter from Arthur E. Hess on opposite page)

Reports and Announcements

MEDICAL MEETINGS

National

American Academy of Obstetrics and Gynecology, Conrad Hilton Hotel, Chicago, December 13, 1955.

American College of Surgeons, sectional meeting, Milwaukee, Wisconsin, February 27-29, 1956.

American Rheumatism Association, Interim Session, National Institutes of Health, Bethesda, Maryland, December 9, 1955.

Association for Research in Nervous and Mental Diseases, Hotel Roosevelt, New York, December 9-10, 1955.

Mediclinics of Minnesota, postgraduate course sponsored by Academy of General Practice of Broward County, Fort Lauderdale, Florida, March 5-14, 1956.

Radiological Society of North America, Chicago, Illinois, December 11-16, 1955.

MEDICLINIC OF MINNESOTA

The first postgraduate course provided by Mediclinics of Minnesota, a new organization, will be given at Fort Lauderdale, Florida, March 5 to 14, 1956. Presented during an eight-day period, the thirty-two-hour course will be sponsored by the Academy of General Practice of Broward County. Credit for the course will be given by the American Academy of General Practice.

Tuition for the course is $50, payable in advance. Reservations must be made early. Further information can be obtained from Mediclinics of Minnesota, 516 Medical Arts Building, Minneapolis 2, Minnesota.

INTERNATIONAL CONGRESS ON DISEASES OF CHEST

The Fourth International Congress on Diseases of the Chest, sponsored by the Council on International Affairs of the American College of Chest Physicians, will be held in Cologne, Germany, August 19 to 23, 1956.

The scientific program is now being organized, and physicians who have carried out original work in diseases of the chest (heart and lungs) are invited to send outlines of their studies to Dr. Andrew L. Banyai, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois. Further information about the congress may be obtained by writing to the same address.

SEMINAR IN OPHTHALMOLOGY AND OTOLARYNGOLOGY

The Tenth Annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach, Florida, January 16 to 21. Lectures on ophthalmology will be presented during the first three days, and on otolaryngology during the last three days of the session. All registrants will be invited to the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on January 18.

Among the lecturers at the seminar will be Dr. C. Wilbur Rucker and Dr. Frederick A. Figi, both of Rochester, Minnesota.

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The nineteenth annual meeting of the New Orleans Graduate Medical Assembly will be held February 27, 28, 29 and March 1, 1956, with headquarters at the Municipal Auditorium.

Eighteen guest speakers will participate, and the program will include fifty-four discussions on topics of current medical interest, in addition to clinicopathologic conferences, symposia, color television, medical motion pictures, round-table luncheons and technical exhibits.

The assembly has planned a postclinical tour to follow the meeting. On March 2, a party of doctors and their families will leave New Orleans for the West Indies and Central America via plane. The itinerary includes Puerto Rico, St. Thomas, Haiti, Jamaica, Panama and Guatemala, and arrangements have been made for medical programs in the places visited.

Details of the New Orleans meeting and the postclinical tour are available at the offices of the assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, Louisiana.

TAPE RECORDINGS ON HEART DISEASE

A collection of nine reels of normal and abnormal heart and breath sounds recorded on tape has been made available to the physicians in Minnesota. This device to assist in the teaching of clinical auscultation of the heart, complete with high fidelity playback equipment, has been placed in a number of hospitals for use of the medical and intern staffs.

The equipment is on loan from the U. S. Department of Health, Education and Welfare, Public Health Service, and is made available through the Minnesota Heart Association in co-operation with the Minnesota Department of Health. Any medical groups interested in availing themselves of the heart sounds equipment should contact either of the two state organizations.

CONTINUATION COURSE

The University of Minnesota will present a continuation course in *Obstetrics for General Physicians* at the Center for Continuation Study from January 5 to 7, 1956. The course will be presented under the direction of Dr. John L. McKelvey, professor and head, Department of Obstetrics and Gynecology.

General Interest

Dr. W. H. Halloran, Jackson, is the new president of the Southern Minnesota Medical Association. Elected as vice president of the organization at the group's annual meeting this fall was **Dr. Leo R. Prins,** Albert Lea. Named as secretary-treasurer was **Dr. G. R. Diessner,** Rochester.

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Dr. Merrill D. Chesler, Minneapolis, was the principal speaker at the eighth annual banquet of the Public Health Nursing Service for Cottonwood County, held at Windom on September 13. His talk on "Reconstructive Surgery for Congenital Deformities" was illustrated by color slides.

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Dr. and Mrs. C. L. Oppegaard, Crookston, left on October 8 for Chicago where Dr. Oppegaard attended a meeting of the American Academy of Ophthalmology and Otolaryngology. While in Chicago he represented the Council of the Minnesota State Medical Association at a meeting with the legal branch of the American Medical Association. During the week of October 16 Dr. and Mrs. Oppegaard flew to Mexico City for a tour of clinics and hospitals, and returned to Crookston on October 24.

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Dr. Frances Elizabeth Schaar, Minneapolis, was certified by examination as a diplomate of the American Board of Pediatrics in Chicago recently.

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Named as president of the Northern Minnesota Medical Association at its annual fall meeting in Bemidji was **Dr. L. F. Wasson** of Alexandria. He succeeded **Dr. W. W. Will** of Bertha in the office.

Dr. N. E. Leppo, Duluth, spoke on the "Handicapped Program of the World Health Organization" at a meeting of United Cerebral Palsy of Duluth on September 23.

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At the annual meeting of the American Association of Obstetricians and Gynecologists at Hot Springs, Virginia, in early September, **Dr. L. M. Randall,** chairman of the sections in obstetrics and gynecology at the Mayo Clinic, Rochester, was installed as president of the organization.

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Dr. Walter S. Neff, Virginia, was appointed medical director of the Range Rehabilitation Center in Virginia at a meeting of the governing commission on September 12. The steadily enlarging center now employs two physical therapists, a therapist-aide and a secretary.

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Five consultants have been added to the staff of the Mayo Clinic, Rochester. They are **Dr. Kenneth G. Berge** in the general section, **Dr. Robert J. Gores** in dentistry, **Dr. Stanley Karansky** in anesthesiology, **Dr. William G. Smith** in proctology and **Dr. Jack P. Whisnant** in neurology.

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Dr. Leo G. Rigler, professor in chief of the department of radiology at University Hospitals, has been appointed to the National Advisory Cancer Council by Dr. Leonard A. Scheele, surgeon general of the U. S. Public Health Service.

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Gustaf A. Hedberg

Dr. Gustaf Arvid Hedberg, superintendent and medical director of Nopeming Sanatorium, Nopeming, Minnesota, has been awarded the 1955 Dearholt Medal by the Mississippi Valley Conference on Tuberculosis.

The medal, highest honor the organization can bestow, was named after the late Dr. Hoyt E. Dearholt, of Milwaukee, and is presented for outstanding service in tuberculosis control in the Midwest.

The presentation was made by last year's recipient, W. M. Morgan, Alliance, Ohio, October 13, 1955, at the Conference's annual meeting in Des Moines, Iowa.

Dr. Hedberg is a graduate of the University of Minnesota, and was in private practice in that state until 1931, at which time he joined the staff of Nopeming Sanatorium.

He is an assistant professor of medicine, University of Minnesota, Mayo Foundation, and is area consultant in tuberculosis for the Veterans Administration.

Dr. Hedberg holds memberships in the National Tuberculosis Association, American and Mississippi Valley Trudeau Societies, and a number of other professional societies, and has served on the board of directors of the St. Louis County (Minnesota) Tuberculosis and Health Association.

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Dr. Carl Herzog, president of Duke Laboratories of Stamford, Connecticut, has donated $500.00 to the Division of Dermatology, University of Minnesota, in memory of the late Dr. Paul O'Leary of Rochester. The fund is to be used in any manner that Dr. Michelson, the Director, sees fit.

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Dr. Shirley C. Lyons, of New Orleans, Louisiana, president of the Alumni Association of the Mayo Foundation, was the presiding officer of the three-day annual reunion of that organization in Rochester, Minnesota, on October 27, 28 and 29, 1955.

A graduate of Tulane University of Louisiana, Dr. Lyons came to Rochester on May 15, 1921, as a fellow in surgery of the Mayo Foundation, which is a part of the Graduate School of the University of Minnesota.

He took graduate work in general medical and surgical diagnosis, postoperative care, surgical pathology and operative surgery. He was appointed a first assistant in surgery on July 1, 1924.

On November 11, 1924, he returned to the private practice of surgery in New Orleans, where he is now located.

Dr. Lyons was certified in 1939 as a specialist in surgery by the American Board of Surgery, Inc., and from 1930 to 1938 he was assistant professor of surgery in the Louisiana State University Medical Center. In World War II, he served in the U. S. Navy Medical Corps, advancing from the grade of lieutenant commander to that of captain.

Dr. Lyons is a fellow of the American College of Surgeons, and is a member of the American Medical Association, the Southern Medical Association, the Pan American Medical Association, the Alpha Kappa Kappa professional medical fraternity and the Phi Kappa Sigma academic fraternity.

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Dr. and Mrs. Rolf Hovde returned to Winthrop in September after a summer of travel which included several weeks in Europe. While in the Scandinavian countries Dr. Hovde visited several hospitals and attended an international medical meeting in Copenhagen.

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New president of the Minnesota Public Health Conference is **Dr. S. A. Whitson**, Albert Lea. Secretary of the organization is **Dr. Dean S. Fleming,** Minneapolis, director of disease prevention for the state health department.

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Dr. Allyn G. Bridge and **Dr. Roger I. Lienke** are now associated in the practice of pediatrics, with offices at 2908 Lowry Avenue North, Minneapolis.

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Dr. R. Gordon Douglas, chief of the New York Lying-In Hospital, was a house guest of **Dr. and Mrs. William B. Stromme**, Minneapolis, during the week of October 10.

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September was a travel-filled month for **Dr. H. W. Meyerding,** retired staff member of the Mayo Clinic, Rochester. First he attended the annual meeting of the Canadian-American section of the International College of Surgeons in Philadelphia, and as vice president presided over several sessions. He then flew to Miami where he presided as president at the convention of the American Fracture Association. Following that he flew to Havana, Cuba, for a joint meeting of the Cuban Orthopedic Society.

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Principal speaker at a meeting of the Rock Lake Farm Bureau at Balaton on September 27 was **Dr. L. J. Thill,** Balaton, who discussed cardiovascular diseases.

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Dr. T. M. Seery, Austin, attended a refresher course in internal medicine at Duke University, Durham, North Carolina, during the second week of October. The course was sponsored by the American College of Physicians.

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A joint meeting of the Minnesota Industrial Nurses and the Minnesota Academy of Occupational Medicine and Surgery was held at the Leamington Hotel, Minneapolis, on September 28. Principal speakers were **Dr. O. J. Campbell,** Minneapolis, who discussed immediate care of surgical emergencies, and **Dr. John F. Briggs,** St. Paul, who talked on immediate care of medical emergencies.

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Dr. Louis A. Buie, a member of the staff of the Mayo Clinic, Rochester, for thirty-six years, retired on October 1. Dr. Buie organized the section on proctology at the clinic and served as head of it from 1919 to 1953, when he became a senior consultant. He was one of the founders of the American Board of Proctology in 1934. He has written several books on the subject, and many proctologic instruments bear his name. He has always been highly active in both state and national medical affairs.

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Dr. Arnold Lazarow, chairman of the department of anatomy at the University of Minnesota, has been appointed a vice chairman of the Committee of Detection and Education of the American Diabetes Association.

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Dr. E. E. Novak, New Prague, left on September 26 for a vacation trip covering the Pacific Coast, Mexico and Honduras. Making the trip by plane, he planned to visit his daughter in Honduras.

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Dr. Robert B. Pierce has closed his office in Olivia and is now devoting full time to his practice at Renville. For several years he has maintained office hours at both cities.

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Dr. Evelyn E. Hartman and **Dr. Wilford E. Park** were married in Minneapolis on September 23. Dr. Hartman is director of maternal and child health in the Minneapolis Health Department, while Dr. Park is chief of the occupational health service of the department.

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A feature article about **Dr. M. S. Munson,** Barnum, which appeared in the *Duluth News Tribune* on September 4, pointed out the benefits of practicing in a small town. Dr. Munson has practiced at Barnum for six years.

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A three-day surgical symposium was held at the University of Minnesota September 22 to 24 in honor of **Dr. Owen H. Wangensteen,** head of the department of surgery at the university for the last twenty-five years. Sponsor of the event was the Owen H. Wangensteen Surgical Education Foundation, composed of former residents and assistant residents of Dr. Wangensteen at the University Hospitals.

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Peripheral and Cerebral Arteriosclerosis

Clinical Aspects of Occlusive Arteriosclerosis of the Extremities

A. WILBUR DURYEE
New York, New York

THE physician charged with the management of a patient with arteriosclerosis involving the peripheral vessels is faced with one of the most complex problems in medicine today. All too frequently he is inspired by the glowing claims of pharmaceutical houses, and once having made this diagnosis, he sits down and writes out a prescription for a vasodilating drug, only to be sadly disappointed with the results as the weeks and months roll by. On the other hand, many of us who have had twenty-five years or more experience in this field will often reach a point of frustration as far as therapy is concerned after spending hours in studying an individual problem. However, the last quarter of a century has developed an approach of study which in many cases helps outline a regime of management often producing satisfactory results. Therefore, it is my purpose to look back over the past twenty-five years and try to pick out from the vast amount of information in the literature and from our own personal experiences such findings that will be of value in studying the individual case and in arriving at a proper means of management and outlining such management.

Arteriosclerosis

Kellner has described this disease as an episodic and segmental disease. Its course is bizarre and unpredictable. The broad term "arteriosclerosis" embodies various pathologies of the arterial tree, frequently overlapping each other, but in each individual case one of the forms of the pathology predominates over the others. For example, in the gangrenous ulcers of the skin associated with hypertension, we find the pathology largely limited to the smaller arteries and arteriols supplying the skin. As Allen, Barker and Hines have pointed out, one can roughly subdivide arteriosclerosis into three types. First, there is medial-sclerosis, frequently referred to as Mönckeberg's sclerosis, where most of the pathology involves the medial coat of the artery and where calcinosis is common. The pathological changes associated with this type of lesion rarely involve the intima, and therefore the signs of arterial occlusion are not frequent in this condition. Although usually present in the latter decades of life, calcification of the arteries may occur from infancy on. It may be present for many years without symptoms. However, atheromotosis may complicate the pathology at any time and produce obstructive lesions leading to the production of symptoms of arterial insufficiency.

Atheromotosis or atherosclerosis is the pathological process which brings most of these patients to the physician. Recent observations during the Korean War where careful studies of peripheral vessels were made in young individuals would indicate that changes in the intima may occur in a much earlier age level than previously suspected. It has been thought for years that this disease starts in the fourth and fifth decades of life. However, it is now common knowledge that it is seen very frequently in the second and third decades of life. With advancing years it becomes much more common and probably is most active in the fourth and fifth decades. The intima becomes involved with the development of localized plaques filled with cholesterol and lipoid material and associated with thrombotic masses in the lumen of the vessel, either partially or completely occluding its lumen. This pathology is much more frequently found in the larger or medium sized arteries of the extremities than it is in the smaller arteries or arteriols. There

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 9, 1955.

is absolutely no set pattern for the development of the lesions which are usually segmental in location and may involve only small areas of one vessel or multiple areas in many vessels. The disease may progress slowly or rapidly and there may be long periods of quiescence.

Involvement of the smaller arteries in an extremity is most frequently associated with hypertensive vascular disease. We have relatively little knowledge as to the early changes in these vessels which are probably largely of a functional nature. As the hypertension persists, organic changes are recognized within these vessels with a tendency for an hypertrophy of the medial coat with secondary thickening of the intima and in some cases complete occlusion of the vessel.

Clinical Picture of Peripheral Arteriosclerosis

The patient who presents himself for medical care due to symptoms of occlusive arterial disease in the lower extremities does so because his symptoms are due to some alteration in the blood supply to the tissues in his extremities. The large majority of these individuals are males, most of them beyond the age of forty-five, and over 99 per cent of them have complaints referable to the lower extremities in contrast to the upper extremities. Their presenting symptoms are a result of a combination of factors which tend to reduce the blood supply to the tissues. These factors may be roughly divided into two main groups: the organic changes which have already been discussed, and the associated spastic or functional factors. Before one can approach the problem of therapy, a most careful analysis of each individual case must be carried out in order to determine not only the type of organic change and its location but also the rate of development. A knowledge of the hemotological and metabolic picture of the individual must be obtained. Associated anemia or polycythemia or diabetes superimposed on the vascular organic lesions will present other problems in management. In addition, an estimation of the secondary or associated vasospastic state is necessary in the over-all evaluation of the specific case. Various means have been developed to estimate the state of the vasomotor tone. Reflex vasodilatation tests using skin temperature as a guide of such vasodilatation, plethysmographic studies, and in this age of atomic energy radioactive materials and their rate of flow following various methods of producing vasodilatation may be used to evaluate each case. Even without such detailed studies, one can frequently determine whether vasospasm exists by simple observation of an extremity. Does it become warm and cold alternately? Does it sweat or become dry from one period of observation to another? Are the patient's complaints extremely variable from time to time?

In the early stages of a slowly progressive occlusive disease, this vasomotor state may make up a large element of the clinical picture and account for considerable reduction in blood flow. This is true particularly of the vessels in the skin. At this point it might be well to point out the so-called "borrowing-lending" phenomenon in the lower extremities. It is a well-recognized phenomenon that where vasospasm of the skin occurs, blood supply to the muscles tends to increase; and vice versa, where vasodilatation of the skin vessels is produced by any method, blood supply to the muscles is reduced. It has also been demonstrated that where a muscle is put to use, an opposing resting muscle is temporarily depleted of blood supply. Some of these observations are extremly important in arriving at the proper therapeutic management of these cases.

Symptomatology

I shall briefly review the outstanding complaints of a patient with arteriosclerosis of the vessels involving the lower extremities, pointing out their significance:

Coldness.—Although coldness may be simply a manifestation of vasospasm and not indicative of organic disease, it is more frequently found in individuals with a rapidly progressing organic disease and an associated vasospastic state. Unilateral coldness is of much more signficance than a bilateral coldness from a diagnostic point of view. Coldness which is persistent is of greater signficance than coldness which is recurrent. Reduced temperature in digits or in part of an extremity tends to point out localized arterial insufficiency. Often the subjective feeling of coldness is more important than the objective evidence of its existence. Coldness is an indication of changes in skin blood flow but gives one no idea of the blood supply to the muscles of an extremity.

Paresthesias.—Many patients in the early stages of occlusive arteriosclerosis will complain of numbness, tingling and burning. Such symptoms are much more common in those patients suffering from the vascular lesions associated with hypertensive disease than they are with the occlusive process of major vessels due to atherosclerosis. However, when atherosclerosis causes acute occlusion, paresthesias are predominant in the picture. Persistent paresthesias are more indicative of organic blockage while recurrent paresthesias would indicate a picture of recurrent vasospasm alternating with periods of vasodilatation. Again, let me point out that these neurological symptoms are the result of alteration of blood supply to the skin and not to the deeper tissues.

Intermittent Claudication.—Here we have a symptom which is directly related to impaired blood supply to muscle. It has nothing to do with the ischemia of skin or subcutaneous tissue. It is a distress frequently described as a tightening, band-like sensation with inability to use an extremity which develops as muscles are inadequately supplied with blood while they are in active use. There is almost immediate relief of this symptom once the muscle is allowed to rest. The symptom returns with the resumption of activity of that muscle. The recurrence of the symptom usually follows almost exactly the same amount of use of the muscle which produced it in the first place. Although some patients will tell you that after the first episode of intermittent claudication their walking distance will increase, it is my personal belief that this increased walking distance is due to the fact that these individuals tend to reduce their speed of walking after the symptom first presents itself and therefore walk a greater distance than they did when the rate of speed was greater.

Rest Pain.—This symptom is an indication of serious arterial impairment to the skin and rarely to the subcutaneous and muscular tissues. It indicates that there is insufficient blood to permit adequate nerve nutrition when the patient is at complete rest. It is most frequently observed during the sleeping hours when, with a reduction of blood flow due to a slow cardiac function, pain develops which awakens the patient from a sound sleep. With dependency of an extremity and perhaps a little physical activity to raise the blood pressure and pulse rate, there is temporary relief of the pain.

Weakness.—The complaint of weakness is one which usually develops after the disease has been present for a long period of time and muscle blood supply has been reduced to a point where atrophy takes place. It also occurs after a long period of disuse with atrophy and with associated osteoporosis.

Objective Findings

Trophic Changes.—On inspection of an extremity with arterial insufficiency due to arteriosclerosis, one frequently notes trophic changes in the skin, reduction in hair growth, transverse ridging of the nails, and actual muscle atrophy. These observations are indicative not only of occlusive arterial disease but may occur in all trophic and functional vascular pathology. However, they are more common where occlusive organic disease is present.

Color Changes.—The classical rubor on dependency and pallor on elevation are almost diagnostic of arterial occlusive disease. With arterial insufficiency to the skin, one has a delayed hyperaemia after blood has been expressed from a local capillary bed. In acute occlusion mottling and cyanosis and marbling of the skin may occur.

Temperature Changes.—Objectively as well as subjectively, one can note by palpation areas of decreased temperature associated with arterial insufficiency. It might be well to point out that frequently the level of occlusion in a main arterial supply is much higher than the temperature level change. In other words, an occlusion in the lower femoral artery frequently is manifested by a coldness extending from the mid-calf area downward. The sudden development of warmth in an extremity which was previously cold, particularly if it is a localized area of warmth, is very suggestive of secondary infection.

Determination of Vessel Pulsation.—This can usually be determined by simple palpation over the major vessels of the extremities, such as the femoral, popliteal, posterior tibial and the dorsalis pedis arteries. However, such a method of testing is unsatisfactory in certain individuals because of associated edema, mal-position of vessels, or obesity. In such individuals, the use of an oscillometer will frequently determine whether or not a pulsation is present. This instrument is not one of precision and should not be used to determine accurately minor changes between extremities or in the same extremity from time to time. However, it is of a great deal of assistance in determining whether there are gross changes and whether or not pulses are present. Examination for pulsations must be carried out in a warm environment with the patient relaxed. There are certain individuals in whom coldness or emotion will cause vasospasm of major vessels. In rare instances examination must be carried out following a paravertebral block or some other method of producing vasodilatation.

Gangrene and Ulceration.—In occlusive arterial disease due to arteriosclerosis, gangrene is almost universally a distal affair, occurring either in the toes or the heel and progressing proximally from these locations. In rare instances infarction of the skin may occur in any part of the extremity, usually in the leg and not in the thigh. Such lesions are extremely painful and are frequently multiple.

The development of gangrene usually depends on the rapidity of the development of the occlusive process, being much more frequent in the acute occlusion than in the chronic one because of the inability of collateral circulation to develop to replace the occluded main vessels. Ulceration of the skin in the lower legs, particularly laterally, may indicate a form of gangrene of the skin secondary to hypertensive disease with secondary changes in the arterioles and smaller arteries, the so-called hypertensive ulcers.

Edema.—Edema as the direct result of arterial occlusion does not occur. Indirectly it may occur on two bases—either secondary to venous or lymphatic occlusion as the result of impaired arterial supply and thrombosis or infection, or as the result of atony of the tissues and dependency of the extremities. In advanced arteriosclerotic disease of the extremities, many patients sit for hours with their feet in a dependent position in order to obtain some relief from the pain. Edema is common in these individuals.

Laboratory

Blood Studies.—As indicated previously, an accurate blood count including a hematocrit study is important to rule out abnormal concentrations or dilutions of the blood as well as to rule out other blood dyscrasias such as the leukemias. Prothrombin time studies are necessary where anticoagulants are to be used therapeutically. Otherwise they have no value. The estimation of the vitamin C level in the blood should be done in all cases, as many of these individuals have a poor intake of this vitamin with a tendency for increased capillary bleeding which in turn tends to complicate the skin lesions of the lower extremities. Blood sugar and blood lipoid determinations should be done on all patients. Associated diabetes should be ruled out. Until we know more about the relationship of lipoid metabolism to this disease, the acquisition of this information may not be of too much value. However, because of the close relationship of abnormal lipoid metabolism to atherosclerosis, each case should have a careful work-up of this type because in the future it may be of real value.

X-ray Studies.—Determination of calcification of vessels should be carried out in each case. This information is of no value in estimating the functional capacity of the vessels but does give one evidence of abnormal pathology in the wall of the vessel. X-rays are also helpful in determining blood supply to bone and indirectly measuring blood supply by determining the amount of osteoporosis present.

Visualization of the luminae of the arteries and collateral circulation by the injection of contrast media is a most important diagnostic procedure in these cases of occlusive disease. Since we now recognize the segmental distribution of many of these occlusive lesions, it is important to localize such areas if they are to be attacked from the surgical standpoint. I am sure that my successor on this program will point out the value of such a diagnostic procedure in studying the lesions of the terminal aorta. Such tests are equally important in studying the blood flow particularly through the femoral artery. Moreover, in those cases of arteriosclerosis complicated by aneurysms, such lesions may be readily picked up by this method.

Measurement of Cardiac Function.—Since the blood flow through the peripheral circulation is dependent on satisfactory cardiac function, it is necessary to carry out routine studies to determine the function of the heart in every patient. An electrocardiogram and fluoroscopic and clinical study of the heart action should be routine.

Management of Arteriosclerotic Peripheral Vascular Disease

I prefer the term "management" of arteriosclerotic peripheral vascular disease to the term "treatment" of arteriosclerotic peripheral vascular disease. We have no specific therapy for arteriosclerosis and at best we can only tend to direct or guide its course and to control the complications which arise. There are certain basic principles of management which we shall discuss. They are as follows:

1. To increase collateral circulation at a rate greater than the occlusive process develops.
2. To overcome vasospasm.
3. To prevent and to control infection.
4. To prevent and to control thrombosis.
5. To remove local areas of arterial obstruction.
6. To treat associated disease.

We shall discuss each of these approaches in detail.

To Increase Collateral Circulation.—In those cases where the predominant symptoms point to poor blood supply to muscle and where segmental blockage cannot be removed, I know of only one procedure which may help develop collateral circulation to the important muscles of a lower extremity. That is the proper use of these muscles. In a series of over a hundred cases treated privately and in our vascular clinic, we have followed a group of individuals with intermittent claudication as the only symptom and have limited their treatment largely to simple advice as to the use of their extremities. Considerable time has been spent with them from visit to visit, detailing an outline of physical activity where the leg muscles are used routinely but well within their tolerance for activity. In other words, these individuals are urged to walk frequently short distances but never under any circumstances to force their walking against any distress in the legs. As this paper was prepared, a rough review of these cases would indicate that over 75 per cent of them had increased walking distance at the end of a year and less than 3 per cent of them had serious complications of their pathology with loss of digits or in one case an extremity. At the end of three years, over 60 per cent still had increased walking distance and only 6 per cent had developed serious complications.

Overcoming Vasospasm or the Production of Vasodilatation.—In those individuals where collateral circulation to the skin must be developed, our second principle of management is applicable, and that is the overcoming of vasospasm. In this group of individuals, the complete abstinence from smoking is extremely important since tobacco causes a high degree of spasm of the skin blood vessels, particularly in the digits; and where such vasospastic ability is still present, the continued use of tobacco interferes with the development of collateral circulation and cuts down the available blood supply. This holds true for any other vasospastic drug such as ergot. Exposure to extremes of temperature should be eliminated. Emotional factors should be controlled as far as possible.

Many physicians have been sadly led astray by the claims of the commercial houses and by certain papers in the literature advocating vasodilating drug therapy. The use of so-called oral vasodilators or the use of these same substances parenterally or intravenously is contra-indicated in practically all cases of peripheral arteriosclerosis. Certainly in those individuals with intermittent claudication as the main symptom the use of these drugs does not increase the blood flow to the muscle. If they do increase blood flow to the skin they probably do so to the healthier parts of the body and to the skin over the healthier extremities rather than to those with impaired blood supply. Purely from a long-term observation of the use of these drugs in our clinic, I have been impressed with their failure to produce results when given as described above. Many of the vasodilating drugs have been put on the market following a short period of experimental use on animals or on normal humans. When applied to our problem which we are discussing today, they are often without value and occasionally detrimental. If one reads the literature supplied with these preparations and has some basic knowledge of the diseases for which they are being prescribed, one cannot help but be impressed by the fallacy of their application. For example, one of the newer drugs which has recently reached the market is claimed to have no effect on increasing skin circulation but marked effect on increasing muscle circulation. Literature from abroad is supplied with this drug by the contact man indicating that in normal human beings the blood supply to the muscle is markedly increased. Yet when this drug is given a clinical trial, there is no indication that it relieves symptoms of claudication. Moreover, in the advertising it is claimed to be an excellent drug for the treatment of a Raynaud's syndrome. In other words, the manufacturers contradict themselves in stating that it is not a skin vasodilator in one breath and then claiming that it will help Raynaud's disease which is a disease of vasospasm of the blood vessels to the skin. This particular drug has already received much publicity in many lay journals.

If vasodilating drugs are to be used, they should be directed toward the nerve control of the blood vessels in the involved extremity. There is some evidence that if they are injected intra-arterially, local vasodilatation can be achieved. However, in our own experience, this vasodilatation may occur to a maximal degree proximally in the skin of the involved extremity while the distal parts, where the need for vasodilatation is greatest, are robbed of blood. Therefore, the most logical method of increasing skin blood flow is by the elimination of vasospasm by the blocking of the sympathetic nerve supply or by its severance with a surgical sympathectomy. In those individuals with impaired skin blood supply to a point where serious complications of ulceration and gangrene may develop, sympathectomy is indicated. Moreover, it is my belief that sympathectomy does not increase blood supply to the muscles and is contra-indicated in the management of intermittent claudication. We have seen numerous examples of further reduction in walking distance following lumbar sympathectomy with a most satisfactory increase of blood supply to the skin. Relaxation of the entire nervous system with the use of sedatives and the so-called tranquilizing drugs and with alcohol sometimes seems to help increase skin circulation.

Control of Infection.—I am sure that most clinics can report a reduction in amputation rate in peripheral arteriosclerosis since the advent of the antibiotics. Certainly every individual with advanced arteriosclerosis should have everything done prophylactically to prevent infection and to treat it once it occurs. This means the elimination of all fungus invasion of the skin by bland fungicide therapy and the immediate treatment of any wound or infected area with systemic antibiotics. Because of the reduced blood supply to the tissues, adequate dosage is indicated to get the local concentration at a satisfactory level to combat the infection. The use of local antibiotics in this condition is of relatively little value, although small areas of ulceration may be partially cleared with some of the local preparations.

Prevention of Thrombosis.—One immediately thinks of the use of anticoagulants when one mentions thrombosis. However, clotting in the diseased arteries can be delayed or prevented by other methods than with the use of anticoagulants. By maintaining a good body metabolism which increases the rate of blood flow, by maintaining hydration and preventing hemoconcentration, and by preventing the slowing down of blood through vasospasm in the collaterals, thrombosis may frequently be avoided. I do not believe that we have reached a point where every patient with peripheral arteriosclerosis should be given anticoagulants prophylactically. The dangers of hemorrhage are ever present and particularly with the possibility of cerebral vascular accidents in the hypertensive group, there is no indication for their routine use. However, in the acute arterial occlusion, immediate anticoagulant therapy with the rapid-acting Heparin is indicated and perhaps later the use of the slower-acting drugs to prevent the complication of an ascending thrombosis or an associated venous thrombosis. This is particularly true if the patient has to be put to bed at rest. These anticoagulants should be continued until vasospasm can be overcome and until the patient again becomes ambulatory or amputation has taken place.

Remove Local Areas of Obstruction.—I shall leave it to others on this program to discuss the surgical management of occlusive arterial disease of an arteriosclerotic nature, but we are all familiar with the excellent results obtained by segmental grafting or the use of by-pass shunts, or even in some cases of endarterectomy. Many patients have been given a marked return of function of the lower extremities and relief of rest pain by such surgical procedures. However, since this disease is a progressive one, unfortunately the use of arterial grafts is probably only a temporary measure. In some patients with long periods of remission, their value may be great. In others, secondary thrombosis shortly after the surgery or involvement of other main arterial trunks may make this procedure of little value. It will be several years before we shall be able to arrive at any correct evaluation of this therapy.

Treatment of Associated Disease.—As in all fields of medicine, the most satisfactory results are obtained when all factors in a given case are managed to the best of the physician's ability. Certainly diabetes in a patient with arteriosclerosis needs careful management to avoid some of the serious complications of this disease when it is out of control. Although many authors have stated that the incidence of gangrene is no greater in the diabetic than in the non-diabetic, it is my personal opinion from clinical observation that it is far greater in the diabetic. Even in those diabetics where control seems to be satisfactory for a number of years, the underlying arteriosclerosis seems to be a more major pathology than it is in the non-diabetic.

The treatment of any associated cardiac pathology is of utmost importance because satisfactory blood supply may not be obtained if cardiac function is not normal. This is especially true if there has been an acute cardiac episode complicating the management of peripheral problems or in those individuals where increasing exercise is wise in the management of the peripheral circulation. Many of our patients are handicapped because of the fact that coronary insufficiency limits the individual with intermittent claudication who should increase his walking but who has to diminish his walking because of the heart vessel involvement. Weight reduction is most important as the individual who is obese tends to have a more rapidly progressing arteriosclerosis. He also is greatly handicapped because of reduced activity since his legs with impaired blood supply will not carry excessive weight. Finally, these patients must be told to live within their capacities, and a careful consultation with them from this standpoint is important. If they understand their disease, its variability, and the need for relaxation, much will be gained in the management of the entire problem.

Summary

There have been many angles that I have not had sufficient time to discuss. The use of various mechanical apparatus to increase circulation has not been covered. These machines have found a diminishing use in the management of this disease. The entire question of posture to blood flow in the extremities so carefully investigated by Wilkins has not been reviewed. It should be emphasized again that each individual case should be carefully studied and that combined approaches to therapy should be used to obtain the maximal blood flow. Careful co-operation between the medical man and the surgeon with the patient treated as a complete individual, and not the extremity alone, will produce a most satisfactory outcome. Surgery will play a more important part in the management of peripheral arteriosclerosis than it has in the past. Drugs will play a lesser role. Nature in many instances will repair damage by the development of collateral circulation despite the aid of the doctor. Too often he takes or is given credit when the natural reparative processes of the body are responsible.

Discussion

Davitt A. Felder, St. Paul, Minnesota: In the drama of the remarkable and rapid advances being made in the treatment of arteriosclerotic problems today, one may easily lose sight of certain essential points charactertistic of this disease. Because of the sometime brilliant results of his accomplishments the surgeon may more often lose sight of the over-all picture of the patient than others. But we all might do well to consider the statement of the vascular surgeon, Haimovici, who said, "Critical evaluation of any type of treatment must obviously be made against the background of the natural course of the disease."*

It seems illogical to subject a patient to the surgical hazard and discomfort incident to the removal of an asymptomatic aortic aneurysm only to have him die then or soon afterwards of a coronary or cerebral ictus.

There are apparently some less obvious possible pitfalls than this in the treatment of arteriosclerosis that present themselves. I am sure that most of us in this audience have pondered the question of the effect of general vasodilatation on an ischemic arteriosclerotic extremity. Yet we use vasodilating agents daily. It is likely that the drop in blood pressure induced by these drugs plus the reduction in the amount of blood in a sclerotic segment of the periphery might be responsible for thromboses in these areas.

We have shown consistently in our laboratory** in a controlled environment that the extremity with sclerotic

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*Haimovici, H.: Peripheral arterial embolism. Angiology, 1:20-45, 1950.

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Clinical Aspects of Arteriosclerotic Aneurysm and Arteriosclerotic Occlusion of the Abdominal Aorta

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Aneurysm of the Abdominal Aorta

SINCE aneurysms of the abdominal aorta are not rare, physicians in this country may expect to encounter them even more often in medical practice in the future inasmuch as the average age of our population is increasing steadily.

The cause is almost always arteriosclerosis. Mills and Horton[1] found that 86 per cent of their series of patients with aneurysm of the abdominal aorta were more than fifty years of age and that twice as many men were affected as women.

The prognosis is poor for patients who do not have treatment. Patients who have been treated surgically have not been followed long enough so that the effect of such treatment on the long-term prognosis is known, but it is known that surgical treatment may be a lifesaving measure for some patients and that many patients have been relieved of symptoms and have lived for many months longer than they could have lived without such treatment.

In Estes'[2] series of 102 patients, of whom ninety-seven had aneurysms attributed to arteriosclerosis, a third of ninety-one traced patients died within a year following diagnosis, half of sixty-three traced patients died within three years and about three-fourths of forty-six traced patients died within five years. Of the patients who died, 63 per cent died from rupture of the aneurysm and 37 per cent from other causes. The severity of symptoms apparently had little relationship to the eventual prognosis as those who had no symptoms at the time the diagnosis was made lived no longer than those who had symptoms due to the aneurysm. This may not be a significant observation, however, as a larger relative number of patients with symptoms would consult a physician, and, therefore, would have been included in Estes'[2] study than those who had no symptoms.

Estes[2] compared the survival of his group with the life expectancy of the "normal" population of the same average age. He found that whereas 35 per cent of a "normal" population which is sixty-five years of age will die within eight years, 90 per cent of his group would die within this same period. The important question is how many patients would have died from complications of the aneurysm that might have been prevented or have been delayed by methods of treatment available today. From a survey of Estes' findings it appears that almost two thirds of the patients known to be dead died from complications of the aneurysm, usually a rupture, within the eight years after diagnosis. This complication presumably might have been avoided by surgical treatment if it had been available. This does not mean, however, that the life expectancy of these patients may not have been shortened anyway by other complications of their disseminated atherosclerotic vascular disease.

Abdominal pain and the presence of an abdominal mass noted by the patient are the two symptoms of aneurysm of the abdominal aorta most frequently encountered. The pain may be persistent or intermittent. Severe, persistent pain may be present even when there has been no rupture and is due probably to a periaortic inflammatory type of reaction sometimes found in patients with abdominal aortic aneurysms. The pain of abdominal aortic aneurysm commonly is a diffuse type of distress in the middle and lower portion of the abdomen or in the lower part of the back. There is a tendency for the pain to be more prominent on the left and to extend down the left side of the abdomen. Sometimes the pain is equally distributed on both sides of

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the abdomen and back with extension into both groins and anterior regions of the thighs.

The one common physical sign is an expansile, pulsatile mass in the abdomen. The bulk of the mass is located usually in the left side of the abdomen. The diagnosis of an aneurysm should not be made solely on the basis of this physical finding unless the mass can be grasped by deep pressure with both hands and localized bilateral, expanding pulsation can be felt. Nervous patients frequently complain of abdominal throbbing and pulsation and in such patients a prominent pulsation may be felt in an aorta of normal size. Patients who have lordotic spinal columns or elderly persons with sclerotic, tortuous aortas may have palpable abdominal pulsations which simulate the pulsations in an aneurysm. A thrill or bruit is present over an aneurysm in only about half of the patients and its presence or absence does not help much in the diagnosis.

It is significant that a third of the patients in Estes'[2] group had no symptoms referable to the aneurysm at the time of examination. The aneurysms of these patients were discovered either on routine physical examination or incidentally by x-ray examination or surgical exploration carried out because of other complaints presented by the patient. This emphasizes the value of careful palpation of the abdominal aorta in the course of the physical examination of all older patients.

Often the aneurysmal sac is partially or almost completely filled with old and new blood clots, and these may affect the degree of pulsation which can be felt as well as the finding on roentgenographic examination. Occasionally the renal artery may be included in the aneurysmal sac, and the blood flow through the renal artery may be impeded by the blood clot in the aneurysm or by the atheromatous plaques. I have observed two patients in whom this type of involvement of the renal artery apparently caused moderately severe hypertension. The most common serious complication is leaking or rupture of the aneurysm. Erosion of the vertebra and pressure on neighboring nerve trunks or organs are rare occurrences.

The diagnosis is not difficult and usually can be made on the basis of the findings on physical examination. A plain roentgenogram of the abdomen will usually show evidence of a soft tissue mass, calcified plaques or in some cases a curvilinear area of calcification which is characteristic of an aneurysm of the abdominal aorta. Aortography is not essential for diagnosis although it is helpful in the evaluation of treatment. The aortogram will give information of importance concerning the relationship of arterial trunks (particularly the renal and mesenteric arteries) to the aneurysm and the patency of these arteries. The presence and size of an intraluminal blood clot is usually well demonstrated in the aortogram. These blood clots may give a misleading picture in that they may almost entirely fill even a very large aneurysm and may cause the lumen to appear normal or nearly normal in size in the aortogram. In such a situation the location of the walls of the aneurysm may be apparent from the curvilinear area of calcification in the wall previously mentioned as being shown in the roentgenogram. This area may be several inches away from the lumen as demonstrated in the aortogram. Occasionally when physical examination and roentgenographic findings are inconclusive, abdominal exploration may be necessary to confirm or disprove the diagnosis.

Occlusion of the Abdominal Aorta

Although the symptom of high intermittent claudication with distress in the lower part of the back, hips and thighs on walking has been recognized for many years, only in recent years has it been realized that this syndrome was often due to a segmental occlusion of the abdominal aorta. Occlusion of the abdominal aorta is almost always a form of arteriosclerosis obliterans and except for its location in the abdominal aorta is essentially the same process that occurs in the arteriosclerotic disease affecting the arteries of the lower extremities, designated for many years as "arteriosclerosis obliterans." Rarely, simple thrombosis alone may cause occlusion of the abdominal aorta but almost always when thrombosis is present, the occlusion is due to a thrombus superimposed on one or more atheromatous plaques. Occasionally the abdominal aorta may be partially or functionally completely occluded by atheromata alone. The occlusion may involve the renal and mesenteric arteries and in some instances of this type complications such as hypertension or infarction of the bowel may result and eventually may cause the death of the patient.

Frequently periaortitis is present and causes adhesions to the vena cava and other structures in the region.

The disease is usually progressive over a period of five to ten years. Although men are affected more commonly than women, occlusive atherosclerosis in this location is the only form of arteriosclerosis that I know of that not infrequently causes symptoms in their early forties in women who do not have diabetes. Arteriosclerosis obliterans which involves the lower extremities almost never occurs in women, even in those with diabetes, before the age of fifty years or in women without diabetes before the age of sixty years. There is something unique and intriguing, especially as to the cause of arteriosclerosis, about this small group of young women without diabetes who have segmental atheromatous occlusion of the abdominal aorta.

Four points should be kept in mind in the clinical evaluation of a patient with occlusion of the abdominal aorta, especially when treatment is being considered: (1) Most of these patients have disseminated atherosclerosis, and complications from coronary or cerebral arterial occlusion are not unusual. (2) Segmental occlusion of the abdominal aorta alone does not often result in severe ischemia in the feet and legs, and ischemic ulcer or gangrene are infrequent complications, at least during the early years of the disease. (3) In addition to the occlusion in the aorta, segments of the arteries distal to the aorta such as the femoral or popliteal arteries may be occluded also by atheromata and thrombi. (4) Enough knowledge of the natural history of this condition has not yet been acquired so that any satisfactory conclusion can be reached as to the eventual incidence of loss of life or of limb from complications of the disease or of the life expectancy of patients who have occlusion of the abdominal aorta.

Chronic occlusion of the abdominal aorta is characterized symptomatically by the gradual development of fatigue and pain in the back, hips and thighs which at first occurs only on prolonged exercise and is relieved by standing. Occasionally in proved occlusion of the lower abdominal aorta or iliac arteries the distress of intermittent claudication may be confined to the calves of the legs. Later in the course of the disease the distance that can be walked without distress is lessened and in many instances may be reduced to less than one block. Some patients may complain of a vague distress and fatigue low in the back and in the hips on prolonged standing or even at rest. Usually this type of static distress is not considered to be due to arterial insufficiency but two patients have been largely relieved of this type of distress following successful removal of the occluded portion of the aorta and re-establishment of blood flow. I have seen two women who complained of distress low in the back, and in the hips and thighs while making beds. This was largely relieved by surgical treatment of an occluded abdominal aorta. Recently also I have had a man, a widower, who had to make up his own bed; he, too, had noted this type of distress, at least to a sufficient degree that he felt justified in getting someone else to make his bed. Dr. Ellis has recently operated on the abdominal aorta of this patient successfully but the patient has not as yet had an opportunity to try out his bed-making ability. Coldness and pallor of the lower extremities are unusual complaints. Inability to have and to maintain an erection of the penis may occur; this apparently is due to an insufficient supply of blood to that organ.

Careful palpation will disclose that pulsations are diminished or absent in the lower part of the abdominal aorta and below the bifurcation of the aorta. It is well to bear in mind that as in coarctation of the aorta occasionally pulses in the femoral arteries may be surprisingly good, although not normal, even when there is complete or almost complete segmental occlusion of the abdominal aorta. Examination may reveal varying degrees of coldness of the feet and legs and some pallor of the feet on elevation of the lower extremities. These changes are usually not marked unless the abdominal aorta is occluded extensively or unless the arteries are occluded below the bifurcation of the aorta. A systolic murmur may be heard over the lower part of the abdomen and in some cases in the back over the lumbar region.

Laboratory findings and plain roentgenograms are of little aid in the diagnosis of occlusion of the abdominal aorta although a roentgenogram of the region of the abdominal aorta may reveal varying degrees of calcification of the aorta. An aortogram usually will reveal the site and degree of the obstruction of the aorta and may reveal additional areas of obstruction in the iliac and upper portions of the femoral arteries if they

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Clinical Picture of Cerebral Arteriosclerosis

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WHEN the term arteriosclerosis is used in its broadest sense to include all of the abnormal processes which involve arteries, a rather large number of pathological conditions affecting the arteries of the brain can be classified under the regional caption, cerebral arteriosclerosis (Table I). Of course, all of these are by no means equally represented in the field of clinical medicine, and indeed the great majority of human illnesses which prove to be due to diseases of the cerebral arteries are the result of but one of the conditions listed, namely, atherosclerosis. The following remarks will, therefore, pertain for the most part to cerebral atherosclerosis, but before proceeding to the main topic, brief reference will be made to some of the other pathological processes included in the above list. While a few of these are more or less specific for brain vessels (ferruginization, capillary sclerosis, saccular aneurysmal dilatation, et cetera), many also involve arteries elsewhere in the body (hypertensive hypertrophy, medial hyalinization, arterial necrosis, Mönckeberg's medial sclerosis, Erdheim's cystic medionecrosis, the arteritides, "Buerger's disease," et cetera).

TABLE I. DISEASES OF THE CEREBRAL ARTERIES

1. Atherosclerosis.
2. Changes associated with arterial hypertension.
    (a) Medial hypertrophy.
    (b) Hyalinization of media in hypertension of long duration.
    (c) Arterial necrosis and fibrin impregnation of severe hypertension.
    (d) Vascular alteration underlying primary intracerebral hemorrhage.
3. Aging or "wear and tear."
4. "Thromboangiitis obliterans."
5. The arteritides—Polyarteritis nodosa.
    —Temporal "giant cell" arteritis.
    —Carotid "giant-cell" arteritis.
    —Non-infectious granulomatous arteritis.
    —Arteritis associated with meningitis —syphilitic, tuberculous, influenzal, et cetera.
    —Rheumatc arteritis.
    —Lupus erythematosis.
    —Septic embolism.
    —Unclassified arteritis.
6. Abnormalities underlying and accompanying the formation of saccular, fusiform, globular, and diffuse aneurysms.
7. Ferruginization of cerebral arteries.
8. Calcific deposition in internal carotid arteries within the cavernous sinus.
9. Thrombocytic acroangiothrombosis.
10. Mönckeberg's medial arteriosclerosis.
11. Cystic medionecrosis of Erdheim.
12. Capillary sclerosis.
13. Radiation arteriopathy.
14. Post-migrainous edema.

Hypertensive medial hypertrophy, judging from routine clinico-pathologic studies, does not compromise cerebral blood flow, the narrowing of the arterial lumen apparently being offset by the increase in the head of arterial pressure. Hyalinization of the media likewise does not influence blood flow. Arterial necrosis and fibrin impregnation are seen only in the most aggravated stage of severe advancing hypertension, and are but part of the pathological picture underlying what the clinician refers to as acute hypertensive encephalopathy. The vascular abnormality which results in primary intracerebral hemorrhage is no clearer to us now than it was 100 years ago. Using postmortem neoprene injections into the middle cerebral artery in whose territory a hemorrhage has arisen, it has been possible in several cases to find in the lenticulo-striate group of vessels a single ruptured artery which probably represented the bleeding point. However, the underlying vascular disease has not been studied as yet. The arterial changes which occur in normotensives with increasing age, including redundancy, enlargement, slight thickening, and increased opacity of the wall, splitting and fraying of the elastica, et cetera, have not been shown to lead to any recognizable symptomatology or to any pathological alterations in cerebral tissues. Mönckeberg's medial sclerosis is not seen in the cerebral vessels distal to the circle of Willis, but has been reported in the carotid artery within the

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cavernous sinus, where it probably never causes any circulatory disturbance. Erdheim's cystic medionecrosis at times involves the common carotid artery, and occasionally an aneurysmal dissection starting in the aorta extends upwards as far as the carotid sinus, resulting in carotid occlusion. The cerebral arteritides unfortunately must be completely omitted from this discussion because of the restrictions of time. Ferruginization of the capillaries and small intracerebral arteries is usually an incidental finding and is encountered in approximately 60 per cent of the brains removed at routine autopsy. Even though the vessels of the basal ganglia, at times become severely involved, extrapyramidal signs and symptoms seem never to arise.

Capillary sclerosis, in which a fine network of reticulin can be demonstrated around the smaller cerebral vessels by the use of silver stains, has in the past been held responsible for senile mental deterioration, the contention being that the connective tissue proliferation hampers a free exchange of nutriments between the vessels and the surrounding parenchymal elements. Gellerstedt, however, found no relationship between aging, mental change, and capillary sclerosis. Cerebral thromboangiitis obliterans, or the alteration of brain tissues classed under that term, is an important subject and will be briefly discussed later.

Turning now to the main theme, atherosclerosis, I think it can be agreed that for the most part cerebral atherosclerosis produces symptoms and signs by only one mechanism, namely, cerebral ischemia due to narrowing or occlusion of one or more cerebral arteries. The atherosclerotic plaque itself is probably asymptomatic, although hemorrhages into a plaque could conceivably lead to headache of a vascular type. Headache is a frequent accompaniment of cerebral thrombosis. Atherosclerosis is commonly held to be the cause of some of the fusiform and diffuse aneurysms of the basilar and carotid arteries. These aneurysms may give rise to symptoms via pressure on neighboring structures, and in this respect the clinical picture might be considered a special manifestation of atherosclerosis. However, there should be much skepticism concerning the rôle of atherosclerosis in these aneurysms for the same dilatations are seen in the absence or near absence of atherosclerosis. It is not improbable that the physical factors which influence the formation of atherosclerosis often lead to its early appearance in the walls of these aneurysms, and thus the deposition of atherosclerosis could be regarded as secondary, not primary.

Atherosclerosis has often been reported to be either a direct cause or at least a contributory cause of hypertensive intracerebral hemorrhage. There is, as yet, no substantial evidence that it plays any significant part. Indeed it is a fact that the actual site of hypertensive bleeding has never been identified microscopically, and therefore it must follow that it would be impossible with any certainty to link intracerebral hemorrhage to the presence of atherosclerosis. At times hemorrhage takes place when visible atherosclerosis is entirely lacking in the cerebral arteries, and on the other hand cerebral atherosclerosis may be most advanced and hemorrhage fail to occur. This negative evidence is of course only indirect and further study of the problem is required. Occasionally, acute subarachnoid hemorrhage is attributed to "rupture of a plaque of atherosclerosis" on one of the major cerebral arteries. This event has never been encountered personally in the neuropathological examination of many cases of vascular disease, and it must occur only rarely, if at all.

Atherosclerosis has also been reported to play a part both in the formation of saccular aneurysms and in their rupture, but this is quite unlikely since in many cases there is no sign whatsoever of atherosclerotic deposition. Not infrequently a plaque of atherosclerosis is found in the cap of a saccular aneurysm when the nearby cerebral arteries show none at all and this is suggestive evidence that special local factors probably physical lead secondarily to deposits of atheroma in the wall of the aneurysm rather than that atherosclerosis primarily causes the formation of an aneurysm. It is of interest that the occurrence of a plaque within a saccular aneurysm provides an opportunity to study an atheroma as it evolves in the subintima of an artery uninfluenced by the elastic lamina and the muscular media, since both of these are absent from the aneurysmal wall.

The few conditions just mentioned in which atherosclerosis is suspected of being a factor but has not yet been proved to play a causative rôle comprise the chief exceptions to the statement made earlier, namely, that cerebral atherosclerosis in most instances produces neurological illness only in so far as it creates cerebral ischemia.

In order to better understand the clinical consequences of cerebral atherosclerosis it is desirable first to examine some of the characteristics of atherosclerosis as they are portrayed at pathological examination. A brief review of its incidence, distribution, severity, secondary changes, et cetera, although knowledge of these is still imperfect, will provide the groundwork for a more thorough appreciation of the clinical picture.

Qualitatively atherosclerosis of the cerebral arteries is approximately the same as the process elsewhere in the body and differs only in minor respects, for example, atheromatous ulceration is infrequent. Its severity in cerebral vessels roughly parallels that of other organs of the body, aorta, heart, and limbs, but in most instances is somewhat less advanced, occasionally more so. Therefore, in the majority of cases in which atherosclerosis has produced cerebral disease, there will be evidence of atherosclerosis elsewhere, for example, angina pectoris, myocardial infarction, electrocardiographic abnormalities, absent pulsation in one or more vessels of the lower limbs, crural intermittent claudication and occasionally subclavian obstruction. It is wrong to emphasize that there are marked differences in the severity of atherosclerosis in cerebral vessels and vessels in other parts.

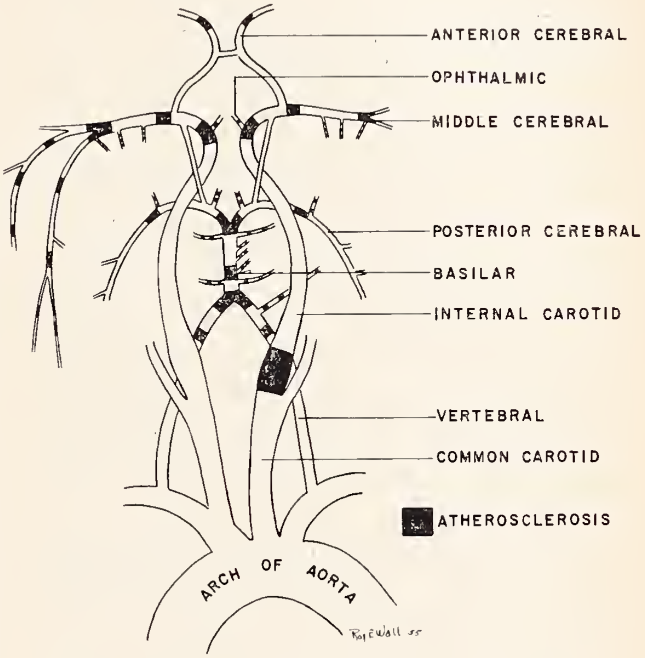

Fig. 1. Diagram of the chief sites of atherosclerotic deposition in the cerebral arteries. The tendency to localize at bifurcations, branchings, and curves is striking.

There are sites of predilection for the deposition of atherosclerosis within the cerebral arterial tree. In keeping with the influence of physical factors in the formation of atherosclerosis, the regions of predilection are for the most part at bifurcations, branchings, and curves. Figure 1 illustrates diagramatically this pattern which must be considered as only a general guide and not a hard and fast formula. The carotid and basilar systems are approximately equally affected although cases will be encountered in which one shows much more atherosclerosis than the other. In general, the large arteries at the base of the brain are the first and most severely involved, the smaller surface vessels often being spared, especially when the blood pressure is normal. The special sites of atherosclerotic deposition are as follows: Within the carotid sinus in the neck, in the carotid siphon extending from the origin of the ophthalmic artery distally to the region of the origin of the anterior cerebral artery, at the first major bifurcation of the middle cerebral artery, in the anterior cerebral artery as it curves posteriorly over the genu of the corpus callosum; in the vertebral artery as it penetrates the dura, in the vertebrals where they join to form the basilar artery, in the basilar artery 5 to 10 mm. above its origin, and in the region of its upper bifurcation often involving the proximal portion of the posterior cerebral arteries, and in the posterior cerebral arteries as they curve posteriorly round the cerebral peduncles. The intervening regions of these arteries also will often show deposits of atheroslcerosis, but the above sites usually are earlier and more severely involved and are therefore the places at which thrombosis will most likely occur. These regions of prediliction exist in the presence of both normal and high blood pressure, but hypertension in addition clearly influences the atherosclerotic process in at least three ways: (1) in general, the process is aggravated, appearing at an earlier age and being more severe; (2) the deposition is more uniform along the vessel either producing a tree bark effect or a dense yellow coating; (3) of greatest significance, however, is the effect of hypertension in leading to the deposition of atherosclerosis in the smaller arteries on the surface of the cerebral hemispheres and over the cerebellum, as well as in the small penetrating arteries which arise from the internal carotid,

TABLE II. FACTORS WHICH DETERMINE THE OCCURRENCE AND SEVERITY OF CEREBRAL ISCHEMIA IN CEREBRAL ATHEROSCLEROSIS

1. *The degree of narrowing of the arterial lumen*—the size of the plaque, the occurrence of superimposed thrombosis, the speed of thrombosis, and its extension; hemorrhage into the plaque, edema of the plaque—cerebral embolism.
2. *The availability of collateral blood flow*—the circle of Willis, the meningeal anastomatic channels, the external carotid artery.
3. *The maintenance of the intracranial arterial pressure and the intracranial blood flow*—the great importance of the systemic blood pressure—the influence of postural changes, sleep, oversedation, and hypotensive drugs. Carotid sinus sensitivity.
4. *Functional alterations in the caliber of the arteries supplying the brain*—vasoconstrictor and vasodilator nervous impulses. Chemical agents including O_2, CO_2, nicotinic acid, alcohol, amyl nitrite, papaverine, ergot and its derivatives, histamine, and adrenalin. Hyperventilation produces vasoconstriction. Effect of tobacco. Influence of warm baths. Physical factors such as missile wounds of neck, carotid puncture, et cetera, can lead to spasm of the carotid vessels in the neck.
5. *The composition of the blood*—anoxic anoxia, anemia, polycythemia, hypoglycemia, dehydration after diuretics, abnormal coagulability of the blood, the effect of anticoagulants on blood flow in small vessels.
6. *Embolism from the site of atherothrombosis.*
7. *The oxygen requirements of cerebral tissues*—cerebral senescence.
8. *Other mechanisms of undetermined nature*—the cause of transient ischemic attacks, stagnant thrombosis, the influence of the emotions (migraine), coughing, direct cerebral carotid sinus reflex, et cetera.

middle cerebral, posterior cerebral, and basilar arteries. These latter vessels supply the deeper cerebral structures including the basal ganglia, internal capsule, thalamus, and brain stem. The small intracerebral branches of the superficial cerebral arteries are also involved. All of these smaller arteries are for the most part spared when the blood pressure is normal, the only exceptions consisting of occasional cases in which the patient is of very advanced age or has diabetes mellitus. Therefore, involvement of these arteries and their territories need be considered in clinico-pathologic deductions only in the presence of high blood pressure.

It is customarily accepted that cerebral thrombosis and arterial occlusion usually occur at the site of the most advanced atherosclerosis. Routine neuropathological examination in our laboratory has shown that this probably holds true for the cerebral vasculature since the above sites of predilection for atherosclerotic deposition proved to be the same regions at which cerebral thrombosis was most prone to occur. Therefore from a practical point of view it is useful to keep the above scheme in mind when visualizing at the bedside the most likely localization of the vascular disease responsible for the patient's clinical picture.

Having roughly plotted the principle distribution of atherosclerosis along the cerebral arteries, we shall now consider in some detail the manner in which the atherosclerotic plaque can lead to cerebral ischemia, and a resultant disturbance of nervous function. As the plaque progressively encroaches upon the arterial lumen, and the flow of blood to the corresponding territory gradually becomes compromised, the occurrence and severity of local ichemia will be determined in the final analysis, by the interplay of many factors. These can be grouped conveniently under some eight headings as shown in Table II. The flow of liquid in any system is a function of the head of pressure which drives the fluid and the resistance offered by the channels through which the liquid flows; the many factors outlined below are for the most part only an expansion of this simple principle; unfortunately there is not space to consider each in detail.

Factors Which Determine the Occurrence and Severity of Cerebral Ischemia in Cerebral Atherosclerosis

Degree of Narrowing of the Arterial Lumen.—This will be determined or influenced in several ways. First and most obvious is the size of the plaque in comparison with the size of the lumen. Secondly, the effect of the plaque will be greatly increased should thrombosis be superimposed. It has been our experience that uncomplicated atherosclerosis, even of great severity, scarcely ever creates a neurological disturbance if the systemic blood pressure is maintained and the other factors to be discussed below remain constant. Under these circumstances the signs and symptoms of ischemia are produced only if thrombosis be added. The details of the thrombotic process which occurs in association with cerebral atherosclerosis are still incompletely understood. We do not know the circumstances under which thrombosis is initiated nor do we have any accurate idea of the range of speeds with which the thrombus builds up. In some cases it seems that total occlusion can supervene in a few hours, whereas at other times thrombosis appears to be a long-drawn-out process. It is perhaps impossible to obtain accurate data on this question from clinico-pathologic studies, since clinical events are not necessarily closely tied to pathological events in time and also because of the difficulty in assessing the exact age of thrombus material from microscopic examination. However, it is probably correct to say that the effects of a combined atherosclerotic thrombotic obstruction will be influenced by the speed of thrombosis, the extent of its spread along the vessel, and the occurrence of additional clotting of stagnant blood proximally and distally. Finally, it must be remembered that an embolus arising in the heart or elsewhere can become arrested at the point where an atherosclerotic plaque narrows the vessel lumen.

Availability of Collateral Blood Flow.—There are several sets of channels through which collateral circulation can become established: (1) the anterior communicating artery from one carotid system to the other or the posterior communicating artery joining the carotid and the basilar system. The pattern of the circle of Willis which is most variable can have far reaching effects on the result of vascular occlusion; (2) the subarachnoid meningeal anastomoses which connect the terminal branches of each major cerebral artery with those of its neighbors, abundant connections existing between the middle cerebral artery on one hand and the posterior cerebral and anterior cerebral arteries on the other (Fig. 2). In addition the larger cerebellar arteries have connections with neighboring vessels of the same and opposite hemisphere; (3) the external carotid artery and ophthalmic artery carry collateral flow in occlusion of the cervical portion of the internal carotid artery; (4) a small amount of blood may enter the basilar system along the cervical branches of the vertebral arteries or via the capillary channels of the labyrinth which is supplied in part from the carotid artery.

The degree to which these channels can enlarge or adapt over a period of time, while an adequate collateral circulation is becoming established, is not known, but in several cases in which they must have been functioning for months or years we did not find gross dilatation on pathological examination. It must be remembered that the development of a collateral flow can be precluded or impaired due to a previous atherothrombosis within the potential anastomatic system, for example, in the anterior or posterior communicating arteries or in one vertebral artery.

It is important to realize also that although there are excellent anastomoses joining the terminal arteries of the major superficial cerebral arteries, there is little or no anastomosis between penetrating and superficial vessels, between neighbouring superficial branches of the same artery, and between neighbouring penetrating branches, (the anterior choroidal artery is an exception). As a result when penetrating vessels become occluded, collateral flow other than that which comes via capillaries is not available.

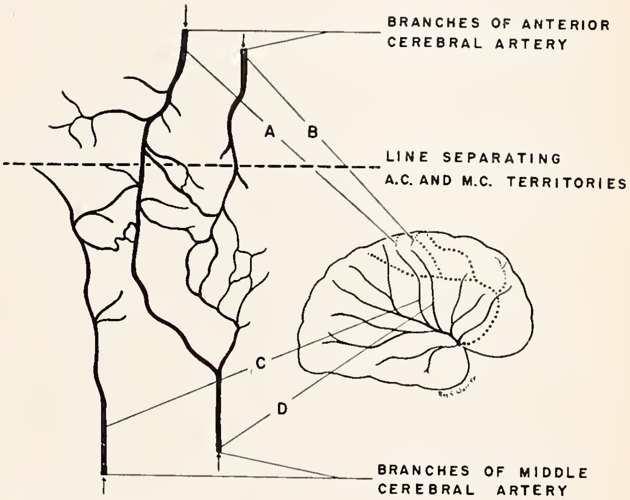
BRANCHES OF ANTERIOR CEREBRAL ARTERY
A
B
LINE SEPARATING A.C. AND M.C. TERRITORIES
C
D
BRANCHES OF MIDDLE CEREBRAL ARTERY

Fig. 2. Drawing of the network of anastomotic vessels connecting the anterior and middle cerebral arteries in the region of the central sulcus. Similar anastomoses are to be found along the remainder of the watershed zone between the major cerebral arteries.

Maintenance of the Intracranial Arterial Pressure and the Intracranial Blood Flow.—Of critical importance in this regard is the systemic blood pressure, and not infrequently well-localized neurological signs first develop when myocardial infarction, a massive intestinal hemorrhage, vascular collapse during surgery, or syncope leads to a precipitous drop in blood pressure. This may be due to the presence of a large otherwise silent plaque of atherosclerosis or to the failure of a previously adequate collateral blood flow. Less dramatic variations in cerebral blood flow can result while changing from the recumbent to the upright position, especially immediately after awaking from sleep. In most cases of cerebral thrombosis, symptoms develop during sleep or shortly after arising. The mechanism responsible for this is not clear, although it is well appreciated that the systemic blood pressure falls during sleep. The cerebral blood flow during natural sleep has not been adequately studied. Oversedation occasionally appears to have been a factor in the onset of a stroke. Hypotensive drugs have been responsible for many cerebral vascular accidents. Some patients with severe atherosclerosis of the carotid sinus develop sinus hypersensitivity and failure of the general circulation can at times be traced to an overactive sinus reflex. Cerebral intermittent claudication in which physical exercise precipitates attacks of cerebral ischemia has been reported in bilateral carotid artery occlusion. The mechanism, no doubt, is a fall in cerebral blood flow due to the upright position and a shift of blood to the active muscle tissue rather than being due to an increase in cerebral oxygen requirement as the term cerebral claudication might imply.

Functional Alterations in the Caliber of the Arteries Supplying the Brain.—These may involve the major vessel whose lumen is being occluded and/or the channels carrying the collateral circulation. In animals, stimulation of the sympathetics produces vasoconstriction of the cerebral arteries; stimulation of the vagus, vasodilatation. However, in humans, nervous impulses are commonly regarded as having insignificant influence on the caliber of the cerebral arteries, under both normal and pathological circumstances. There is not time at present to enter into a full discussion of the normal and abnormal vasomotor action of cerebral vessels, a problem which can be epitomized in the popular question, "Does cerebral vasospasm really exist"? Direct observations of human cerebral vessels have been made on only a few occasions and under circumstances which preclude any final judgment as to the existence or not of significant vasomotor changes in cerebral vessels under more or less physiological conditions. To be sure, those who would deny significant "functional" changes in the cerebral vasculature will immediately be faced with the difficulty of explaining the aura of migraine. Many neurosurgeons have encountered cases in which novocain block of the cervical sympathetic nerves has promptly relieved signs of ischemia which had developed in the territory of the carotid artery either as the result of carotid ligation or carotid angiography. Similarly, there have been several reports in which stellate ganglion block performed soon after the onset of a stroke has produced a dramatic, although often temporary, improvement in the neurological deficit. In some cases, in which worsening occurred as the first block wore off, repetition of the block has again had a beneficial effect.

Changes in the tone of the cerebral vessels are, of course, also produced by various chemicals including oxygen, carbon dioxide, nicotinic acid, aminophylline, alcohol, amyl nitrite, papaverine, ergot derivatives, histamine, adrenalin, and noradrenalin. The most powerful of the vasodilators is carbon dioxide, inhalation of a 5 per cent carbon dioxide mixture increasing the cerebral blood flow by some 50 per cent. In comparison the other vasodilators (nicotinic acid, alcohol, histamine, et cetera, have only a very weak effect.

Hyperventilation which produces cerebral vasoconstriction has apparently never been used in an attempt to elicit the fleeting prodromal symptoms which often herald the onset of a stroke. The influence, if any, of tobacco smoking on cerebral vessels is not known. Physical trauma such as near-miss bullet wounds can produce extreme vasoconstriction of the extracranial carotid arteries. Likewise, needling the carotid artery or stimulating it electrically also can occasion local spasm of that vessel. Hot baths are said to aggravate focal symptoms and signs in multiple sclerosis presumably by altering the vasculature in some way, but this effect has not been noted in cases of atherothrombosis.

Composition of the Blood.—When the circulating blood is faultily oxygenated, threatening ischemia may be aggravated sufficiently for symptoms to appear. Hypoglycemia can lead to local cerebral symptomatology and this may be determined by a preceding local vascular insufficiency. Polycythemia by virtue of increased viscosity of the circulating blood, produces slowing of the capillary circulation, but whether this is of clinical significance is not clear from the available data. Strokes have been reported following the excessive use of diuretic agents presumably due to the production of hemoconcentration. This interpretation, however, needs further confirmation, since the stroke could well be a coincidence. Increased coagulability of the blood, a condition concerning which there is much speculation, might be responsible for thrombosis at the stage of rather mild atherosclerosis. Conversely, it is almost certain that anticoagulants, both heparin and dicumarol, can in many cases prevent the transient ischemic attacks so commonly seen preceding thrombotic strokes. The mechanism of their action in these cases is not clear as yet, but it has been suggested that in addition to their anticoagulant effect, blood flow through small vessels is enhanced.

Embolism from the Site of Atherothrombosis.—A plaque of atherosclerosis within the carotid artery may rupture and the atheromatous material be widely dispersed distally. A thrombus as it builds up may break away and produce an embolic infarct. Embolic fragments may also be detached from mural thrombus formed upon atherosclerotic plaques within the carotid arteries.

Oxygen Requirement of Cerebral Tissues.—This is largely a theoretical consideration and it has not been conclusively demonstrated that normal "use" of the various cortical areas subserving vision, hearing, et cetera, increases the metabolism of these regions significantly above the "resting" levels. In regard to the effect of thinking, Benedict said, "The cloistered scholar at his books may be surprised to learn that the extra calories needed for one hour of intense mental effort would be completely met by the eating of an oyster-cracker or one-half of a salted peanut." This relatively constant oxygen utilization by the brain is in marked contrast to the conditions pertaining in the myocardium where wide fluctuations in the oxygen requirement, due to increased work, may determine the development of a relative ischemia, the coronary blood flow remaining relatively constant.

Under this heading might also be mentioned the influence of preexisting cerebral disease on the effect of ischemia. Elderly persons whose cerebral tissues have been touched by Alzheimer's disease or some other as yet unclassified or unknown "degenerative" process appear to suffer greater damage to brain and mind than younger patients with vascular insults of the same severity. However, this does not mean that the tissues of the elderly are especially susceptible to anoxia, but rather that cerebral functions are already impaired in a way which possibly escapes casual clinical observation with the result that the effect of the added ischemic insult becomes magnified.

Other Mechanisms of Undetermined Nature.—(1) The mechanism of the transient ischemic attacks which occur in association with cerebral thrombosis have so far not been satisfactorily explained, and although they probably arise in one or more of the ways mentioned above, some as yet unknown mechanism may be responsible. (2) The changing patterns of blood flow in a complicated network of channels, such as the cerebral arterial tree, can only be surmised at the present time. Some of our observations point to the possibility of local stagnation and temporary cessation of blood flow, particularly in regions of the brain which lie in the most peripheral territories of major cerebral arteries which have become occluded. Here the pressure in the normal artery of supply and in the collateral channels may be approximately equal with the result that blood flow within the intervening territory might become slowed or even cease. An arrangement of this kind would explain some of the unusual pathological findings in so-called cerebral Buerger's disease. (3) The influence of the emotions can also be placed in this group. Not infrequently, the patient will attribute his stroke to an emotional outburst, but the mechanism by which emotion elicits this effect is not understood. Migraine with and without aura provides a good example of the way in which emotional or personality factors can lead to "functional" vascular change. (4) Occasional cases are encountered in which coughing precipitates a fleeting attack of paralysis or numbness and in which the Valsalva effect seems not to be the mechanism involved. (5) The possibility of a direct cerebral carotid sinus reflex must be kept in mind although such cases are rare. The author has not seen an example of this syndrome.

It might seem that the foregoing discussion of the factors affecting the result of atherosclerosis upon cerebral circulation and nervous function is not highly pertinent to the subject of this paper. Certainly, the subject might well have been approached in other ways, for example, by attempting to outline all of the neurological signs and symptoms produced by occlusion of each of the many cerebral arteries, large and small. An undertaking of that nature would have provided an excellent opportunity for a short refresher course on cerebral physiology and cerebral localization; indeed I would have welcomed an occasion to lay before you the fascinations of neurology as a strokologist. However, no two stroke cases are the same clinically or pathologically. The clinical diagnosis of the nature and location of vascular lesions is not a simple rule-of-thumb matter, and it is a very humbling experience to have one's diagnoses carefully checked at the autopsy table. Well-recognized clinical syndromes associated with the occlusion of various arteries are found outlined in textbooks which nature for the most part seems not to have read, for in actual practice it is much more common to encounter only fragments of a syndrome and those often in such an admixture that identification is most difficult. Rather than attempting an artery by artery account of the clinical picture associated with cerebral thrombosis, I have instead made it my aim to outline some of the general principles which in various combinations are operative in all cases and knowledge of which will help the physician to interpret the interesting series of clinical events encountered in cerebral thrombosis, beginning with the earliest fleeting warning, progressing through the tragic period of the full blown stroke and on to demise or some degree of recovery. The great difference in the clinical course from patient to patient becomes meaningful in some measure only when the principles discussed in the previous section are applied. Furthermore, the use of rational therapeutic methods can for the most part be determined only by a knowledge of the underlying patho-physiological events. An *understanding* of the clinical picture is required for a correct interpretation of our therapeutic successes and failures, and a sound appraisal of the value, if any, of the various kinds of treatment which may be suggested. In the following paragraphs, the practical significance of the above general principles will be further emphasized.

Additional Observations Concerning Atherothrombosis

The final part of this paper will be devoted to a number of brief statements concerning some of the more important aspects of thrombotic disease. No doubt at least a few of the conclusions drawn are premature and will require modification in the future, but in general the author has found them useful practical guides at the bedside.

Atherosclerosis Without Thrombosis.—In my experience uncomplicated atherosclerosis, even though severe, does not seem to cause cerebral disease. The plaque is usually placed asymmetrically and a small lumen capable of conducting some blood remains open at one side unless thrombosis takes place. However, should a precipitous fall of the systemic blood pressure occur, ischemia, with or without thrombosis, may follow. The fall in blood pressure can be due to myocardial infarction, hemorrhage, syncope, traumatic shock, surgery and anesthesia, heart block, hypotensive drugs, anaphylaxis, cardiac arrest, carotid sinus sensitivity, et cetera. An occasional exception to the rule concerning the eccentricity of the plaque may be found in the vessels over the cerebral hemispheres in severe hypertension. Then, atherosclerosis may be deposited uniformly around the circumference of the lumen, finally leaving only a threadlike central channel which sometimes extends for several centimeters along the vessel.

Asymptomatic Cerebral Thrombosis.—Complete atherothrombotic occlusion is found on pathological examination in cases in which no cerebral lesion can be demonstrated and in which no clinical signs or symptoms had existed. This has been encountered in the common carotid, internal carotid, middle cerebral, anterior cerebral, anterior communicating, posterior communicating, posterior cerebral and vertebral arteries, and on two occasions the basilar artery. On the basis of these findings alone, one must be especially hesitant in attaching significance to plaques which only partially obstruct the lumen. When the middle, anterior, or posterior cerebral arteries become occluded asymptomatically, it must mean that the meningeal anastomoses are bringing in an adequate supply of blood from a neighboring artery. At the other end of the pathological spectrum thrombosis in some cases leads to softening and destruction of virtually the entire territory of a vessel. Between these two extremes the infarction resulting from occlusion will vary in site and size according to the interplay of all the factors heretofore described. One can expect, therefore, that the clinical picture and course of recovery will be highly variable.

Sites of Predilection for Thrombosis.—Atherothrombosis spares almost no cerebral vessel but corresponding to the special localization of atherosclerosis which was described above occlusion is found in certain regions more commonly than in others. Both the carotid and basilar systems are frequently affected. In view of the tendency prior to the last few years to airily assign virtually every stroke to the middle cerebral artery, it should be emphasized that the incidence of involvement of the basilar system does not lag far behind that of the carotid. Indeed most stroke cases particularly early in their course should be scrutinized carefully to see if they can be fitted into the picture of vertebral-basilar disease. The vertebral artery may prove to be one of the most frequent of occluded vessels. In order of frequency of thrombotic occlusion the large arteries at the base of the brain might be arranged as follows: internal carotid, middle cerebral, vertebral, basilar, posterior cerebral, and anterior cerebral. When hypertension and atherosclerosis co-exist, the small penetrating arteries will be affected in addition to the large basal arteries. Occlusion of a penetrating vessel results in a small infarct several mm. in diameter in the deeper part of the cerebrum or brain stem and the cystic cavity which remains behind in the healed stage constitutes the so-called lacune. When several of these are present, the term état lacunaire is used. These lesions which have not been shown to be clearly due to thrombosis, although that is their most likely cause, are probably the commonest of all thrombotic strokes, and not infrequently at autopsy several such lesions are found in one brain. Lacunes are often the substrate for the brief or mild strokes to which hypertensives are prone and when multiple lesions occur pseudobulbar palsy, dementia, marche à petits pas, et cetera, result. The presence of hypertension in the clinical picture indicates, therefore, that the vascular occlusion may possibly be in the smaller vessels, whereas in normotensives this possibility is largely excluded.

Transient Ischemic Attacks.—Transient ischemic attacks in which fleeting symptoms come and go are very common in association with atherothrombotic occlusion. Occurring for a variable period before the onset of a stroke, the neurological details of the attack are an accurate forerunner of the form the stroke will finally take and presumably arise as the result of a temporary ischemia which later will no longer reverse itself. There has been much discussion as to the exact mechanism of these episodes, vasospasm, reflex physiologic vasoconstriction of the affected vessel, a fall in systemic blood pressure, undetectable changes in the pattern of cerebral blood flow, repeated embolization, fluctuation in the size of the thrombus, et cetera, but so far no adequate explanation has been provided. Primary intracerebral hemorrhage practically never gives rise to such episodes and cerebral embolism only very rarely. Therefore, a stroke in which a history of prior prodromal transient attacks can be obtained is immediately stamped as thrombotic in type. Eliciting a careful detailed history of the development of a stroke is one of the most important steps in reaching a conclusion as to the nature of the stroke. Hemorrhage most often comes on gradually over a period of several minutes to an hour or more, while the patient is awake and is not preceded by any type of warning. Hypertension is nearly always present. Cerebral embolism comes on abruptly in most cases and evidence of a source in heart (myocardial infarction, auricular fibrillation, auricular fluttter, mitral stenosis, endocarditis, et cetera) and the occurrence of embolism elsewhere in the body substantiate the diagnosis. In saccular aneurysms there is often a stuttering onset associated with headache, loss of consciousness, seizures, and focal neurological signs, but the severity of the headache and the presence of blood in the cerebrospinal fluid will help to establish the correct diagnosis.

The neurological features of the transient ischemic attacks which are encountered in association with thrombosis are not only of several different types but form combinations of the most varied nature. Their character will, of course be determined by the site of the occlusion and also by the many factors which influence the development of ischemia in general. In the middle cerebral artery the attack may be characterized by headache, numbness, paralysis, or paresis of the contralateral side, dysphasia, scintillating scotomata, hemianopia, confusion, and apraxia. Numbness and weakness of the opposite leg, and to a lesser extent the opposite arm, incontinence, apraxia, and confusion may occur with involvement of the anterior cerebral artery. The picture in occlusion of the posterior cerebral artery will be comprised of one or more of the following: blindness, dark vision, scotomata, hemianopia, numbness, tingling, weakness, and confusion. Involvement of the vertebral-basilar system will occasion many different combinations of the following: dizziness, light-headedness, staggering, veering, diplopia, dark vision, blurred vision, blindness, tubular vision, faintness, unconsciousness, confusion, dysphagia, dysphonia, collapse, weakness of arms and legs, respiratory difficulty, numbness of the extremities of one or both sides, headache, head noises, tinnitus, deafness, twitching, et cetera.

Attacks may last a few seconds up to an hour or more. Repeated attacks usually mimic each other rather exactly and in the intervals between attacks, symptoms and signs usually clear up. Although these transient episodes indicate that a stroke is to be expected, it may be long delayed and indeed sometimes does not eventuate. There may be only one episode before the onset of the stroke, or hundreds or even more than a thousand may occur without a stroke supervening. Warnings may occur for only one day or persist for months and years. The attacks often come on without any recognizable precipitating factors, but not infrequently they will be related to change of posture, exercise, waking up, emotion, standing still, coughing, et cetera. It is our experience that if transient symptoms are found to be present on awakening, the development of a stroke in the very near future is to be feared. In some cases it will be difficult to distinguish transient ischemic attacks from cerebral seizures, Ménière's disease, and migraine, as well as from other types of vascular disease. A proper appraisal of these episodes is of great practical importance since the use of anticoagulants at the stage of fleeting reversible symptoms may allow prevention of a stroke. The time for therapeutic measures is prior to the development of the full stroke, and later no treatment seems to be of avail.

Development of the Stroke.—(Only thrombosis is being discussed and not embolism or hemorrhage.) The final development of the full-blown stroke is a most unpredictable matter. Undoubtedly, in some cases, symptoms accrue suddenly and without any recognizable prodrome, the differentiation from cerebral embolism then being most difficult. A common type is that referred to above in which one or more fleeting warnings indicate that disaster threatens. When the final stage is reached the entire neurological deficit may develop abruptly, often during sleep or shortly after arising, or on the other hand, paralysis may come on in stuttering fashion over a period of several days or even a month, additional fragments of worsening being added from time to time.

Thus, depending on the stage of the illness at which the patient is examined, the physician may ask several questions: (1) Are the fleeting symptoms ischemic attacks or not? (2) Are the transient episodes a harbinger of worse things to come? (3) Is an increasing neurological deficit persisting between the attacks? (4) Is the neurological deficit which the patient now shows all that is to be expected in the near future? This last question is often most difficult to answer and not infrequently a patient admitted to hospital with only weakness of one side or paralysis of one limb or with aphasia, while under observation, is found one morning to have a most severe hemiplegia. When the basilar system is affected, a patient complaining of mild dizziness or dysphagia one evening may be found the next morning in deep coma. There is no satisfactory terminology to indicate this evolutionary aspect of strokes, and at present we use the terms *complete* and *incomplete* stroke to distinguish those in which we think a stable circulatory state has been achieved from those in which a further worsening can be expected.

If anticoagulants given at the stage of the incomplete or partial stroke should prove capable of altering the course of events, it would become a most important matter to be able to recognize the stage at which any stroke stands when first seen clinically. At the present time, we are inclined to feel that when the carotid system is involved, any deficit less than a severe hemiplegia involving face, arm and leg means that further ischemic infarction can be expected in the future, depending on all the factors which determine the degree of ischemia and which were outlined in the previous section. In involvement of the vertebral-basilar system, if the clinical picture indicates that the thrombosis is limited to the vertebral artery, serious progression is not to be expected immediately, although when the other vertebral artery is vestigial or previously occluded, obstruction of the remaining vertebral artery will have the same significance as basilar artery occlusion. When hypertension is a factor a brief, mild stroke may indicate a lacunar lesion rather than a partial stroke, in which case no progression would be anticipated. It should be emphasized that it is impossible in many, indeed in most instances to predict with any confidence the future courses of strokes-in-evolution.

Use of Anticoagulants in Atherothrombosis.—It is becoming clear that anticoagulant therapy alters the natural course of events in many cases of cerebral thrombosis and postpones indefinitely the arrival of a threatening stroke. However, it is not possible at present to estimate its real value since many strokes which were apparently prevented by giving anticoagulants at the stage of warnings might not have occurred had nothing been done. In some cases transient ischemic attacks have recurred for two or three years and then subsided without resulting in a stroke. There is need of a careful study which will compare the progress of a group of cases receiving anticoagulants with a similar control group in which anticoagulants are not used Many questions are awaiting an answer: Should all patients exhibiting transient ischemic attacks receive anticoagulant therapy? If not, when does sufficient indication exist? For how long should such therapy be continued? Is excessive hypertension a contraindication to therapy?

From personal experience with a small number of cases, it appears to be quite certain that anticoagulants will in many cases abolish the fleeting attacks which so often herald a stroke. Furthermore, no patient started on anticoagulant therapy at the stage of transient ischemic attacks has gone on to develop a stroke. It is a large undertaking to prescribe anticoagulants in every case in which transient ischemic attacks are present, but at present we think it is justified to consider each patient who has had more than one transient cerebral ischemic attack a candidate for anticoagulant therapy for at least a period of two months. A trial period without therapy is then begun and the patient's future course will determine whether anticoagulant therapy must be restarted. Start and stop therapy is also an excellent method of testing the efficacy of anticoagulants in general. We have had several cases in which symptoms recurred when anticoagulant therapy was stopped, and which cleared when therapy was once more instituted.

Anticoagulants are also indicated when the actual stroke begins to evolve, hoping that the thrombus will diminish in size or that progression can be arrested or delayed while collateral flow is becoming established. Another important group is that in which a partial stroke or incomplete stroke is present and the prospect of a further bout of worsening is great. These patients should also receive anticoagulants. When speed is essential, heparin and dicumarol are used, otherwise dicumarol alone is prescribed. Patients first seen after the development of a full blown stroke are not given anticoagulants, although it has not been proved that anticoagulants are without benefit even in this type of case. It must be emphasized that we cannot expect all patients to be benefited by anticoagulant therapy since old thrombus will not dissolve and in some cases collateral circulation will never prove capable of compensating for the reduction in flow which resulted from thrombosis. Also it is to be remembered that even though anticoagulant therapy is beneficial at one stage, with the passage of time and the gradual worsening of the atheroslerotic deposition in arteries, a point will be reached at which anticoagulant therapy can no longer rescue the patient from his fate.

Regarding the History.—After a stroke has occurred, there may be difficulty in obtaining the details of the development of the illness. When the dominant hemisphere is affected, dysphasia which may be present will often make it impossible to take a history from the patient himself. In non-dominant lesions the patient who is relatively alert may be able to speak clearly and give a satisfactory history. However, in cases with lesions of this type anosognosia is frequently present particularly at the onset of the illness, the patient therefore actually failing to appreciate his illness with the result that the historical details, which were so plausibly presented, prove to be quite false. Therefore, to the obvious difficulty of obtaining a history from a dysphasic patient with a dominant lesion must be added the much less obvious hazard of being misled in cases with non-dominant lesions and anosognosia. The memory function of the elderly is often inconspicuously impaired, making it advisable to have their story corroborated, by younger persons, if possible. Everyone is well aware of the general need for clarity and proper interpretation of the terms used by the patient to describe his illness, but in the case of transient ischemic attacks this need should be especially emphasized. It requires great patience on the part of the clinician if he is to comprehend the exact meaning of the words so often used by these patients, for example, dizziness, light-headedness, blurred vision, numbness, heaviness, tiredness, headache, noise in the head, et cetera.

Development of Coma in Strokes.—The cause of the onset of coma in cerebral hemorrhage is usually quite obvious, namely, a greatly raised intracranial pressure, distortion of brain stem structures, secondary pontine hemorrhages, and often rupture of the hemorrhage into the ventricular system. In cases of brain softening and infarction, the development of coma is not always so easily explained, although extensive involvement of the tegmentum of the brain stem in the basilar territory characteristically leads to deep coma. When either cerebral hemisphere is affected, profound unresponsiveness developing over a few days is not due to "extension of the clot," an explanation so frequently offered, but usually is the result of brain swelling and herniation of brain tissue through the tentorial opening, under the falx, or into the foramen magnum. Unsuspected extensive involvement of both hemispheres is another possibility. Uncomplicated softening in the middle cerebral territory of one hemisphere will in most cases not produce deep coma.

Headache.—It is not widely appreciated that headache or head pain frequently accompanies cerebral thrombosis. Although this type of headache has not been studied in detail a few observations may be recorded. Certainly it does not accompanying all thrombotic strokes or even the majority. Usually the headache is mild but definite, and only rarely is it a prominent complaint. In involvement of the internal carotid artery the pain is most often above the corresponding eye but may extend posteriorly over the side of the head. Thrombosis of the posterior cerebral artery is referred forwards to the anterior and lateral frontal regions. In disease of the basilar artery, headache is usually mid-line in the occipital region. Vertebral artery pain is referred to the area behind the mastoid process. Headache not infrequently occurs with transient ischemic attacks. The headache most often is deep and steady, but occasionally it is throbbing in character.

Dementia.—The mental deterioration of senescence both in its mild and more aggravated forms is often attributed to cerebral arteriosclerosis or "hardening of the arteries" without any clear idea of the underlying pathological picture or the pathophysiological implications of such a statement. Mental decline in which the clinical picture is characterized for the most part by impairment of memory is usually a reflection of Alzheimer's disease or some other "degenerative" process of undetermined nature, conditions in which impaired cerebral blood flow and cerebral infarcts play no part as far as is known. On the contrary, when cerebral atherosclerosis produces dementia it usually does so by causing ischemic infarcts which are so located as to damage those parts of the brain especially subserving mental function. In most instances, therefore, it will be possible to discover in addition to mental deterioration, focal neurological signs which will suggest the presence of vascular disease. Dyphasia, apraxia, hemianopia, hemiparesis, altered tendon reflexes, or abnormal plantar responses, may be found. Furthermore, the onset is liable to be abrupt when infarction is responsible, whereas in the degenerative processes mental recession is usually quite gradual. The concept that undiscovered small strokes in "silent" areas are responsible for mental decline is an attractive one, but further clinico-pathologic studies are awaited before it can be accepted. In our experience, mental deterioration clearly attributable to cerebral atherosclerosis usually is accompanied by tell-tale neurological abnormalities.

Cerebral Thromboangiitis Obliterans.—The pathological condition which goes by this name is characterized by sunken yellowish brown areas of old infarction scattered continuously or discontinuously over the cerebral hemisphere, especially in the watershed region separating the middle cerebral territory from that of the anterior and posterior cerebral arteries. A unique feature, however, pertains to the arteries within the infarct which are reduced to white threads. The lumen of these vessels is filled with a fine connective tissue meshwork. Although there is no arteritis, the name thromboangiitis obliterans was applied because of the resemblance of the vessels to those described by von Winiwarter in the arteries of an amputated foot. In several cases described in the literature, a large artery, internal carotid, middle cerebral or anterior cerebral, was also occluded, but this was felt to be co-incidental. In our material, similar occlusions of large vessels (internal carotid, middle cerebral) have been found in most instances and it is suggested that the characteristic cerebral lesion is not related in any way to thromboangiitis obliterans (itself an ill-defined entity). On the basis of the location of the infarct far peripherally in the middle cerebral territory and the intervention of rather healthy patent artery between the white occluded portion and the occlusion more proximally in the larger parent vessel, it is suggested that intravascular clotting occurs due to the local stagnation of blood when the pressure in the main artery of supply balances the pressure in the collateral channels. The fine connective tissue which fills the lumen of the white worm-like arteries is the result of the organization of blood clot. There has not been any evidence of arteritis in our cases and the designation of the lesion as an angiitis is quite unwarranted.

Summary

1. A résumé of cerebral arterial disease has been presented.
2. Some of the pathological aspects of cerebral atherosclerosis have been described.
3. Some of the factors which influence the local cerebral blood flow distal to an atherosclerotic plaque have been briefly outlined.
4. Several aspects of cerebral atherothrombosis have been dealt with, particularly the more important clinical features.

Discussion

A. B. Baker, University of Minnesota: Dr. Fisher has presented a very excellent review of the subject of cerebral arteriosclerosis. One must certainly agree with most of his opinions and observations. In his discussion, Dr. Fisher has emphasized primarily the involvement of the larger basilar vessels since these structures are most commonly implicated in those patients who are considered to be suffering from the striking syndromes of cerebral thrombosis. Generally the smaller intracerebral vessels, those under 100 microns, are not given too much consideration, particularly in individuals who are normotensive. We have been much more impressed by the changes in these vessels, particularly in individuals who do not show evidence of hypertension.

These smaller intracerebral vessels always show a constant structural alteration with age. As early as the third and usually during the fourth decade of life, these vessels from 20 to 150 microns in diameter begin to show changes in their structure. The elastica interna becomes reduplicated, fragmented, and often loses its normal tinctorial properties. The media undergoes fibrosis with hyalinization and occasionally calcification of some of its elements. Often by the fifth and sixth decades, these vessels show indistinct staining of all the medial elements and a definite narrowing of the vessel lumen. We have examined a large number of brains of normotensive individuals in which this narrowing has resulted in tiny areas of perivascular tissue change. These changes within the smaller intracerebral vessels are variable in degree but definitely increase with age. Certainly within the older age groups namely the fifth and sixth decades, the intensity of alteration and the degree of vascular narrowing is quite variable.

In view of the fact that these changes within the smaller intracerebral vessels are always present and progressive, one wonders whether they may not be playing some role in the final clinical picture seen in the thrombosis of the larger basilar vessels. May not the final clinical picture in the obvious cerebral thrombosis, and perhaps some of the variability in the clinical symptoms as well as the ultimate prognosis, be modified or influenced by the changes that normally occur within the smaller intracerebral arteries?

"As doctors and clinicians, our problem is to balance beneficial effects of ovary removal against the loss to the patient of orderly hormone interchange, plus the loss of protective action of ovarian secretion. From the standpoint of the heart specialist, the harmful effects on the circulatory system outweigh the possible beneficial effects, except in the exceptional instance," Dr. George C. Griffith told some 1,200 doctors attending the fourth annual clinical meeting of the American Academy of Obstetrics and Gynecology, in Chicago.

Dr. Griffith said the practice of removing ovaries, usually in the course of other pelvic surgery, in women who have passed the child-bearing age is based on the old and erroneous belief that the glands are useless by that time and that their removal prevents possible development of ovarian cancer, which strikes down one woman in each 100 women past the fortieth year.

There is a growing realization that the ovaries are not concerned only with the sexual and reproductive life of the individual, but, continued Dr. Griffith, among the other important functions of the ovary is the regulation of metabolism. Furthermore, in cases of impaired pancreatic activity, ovarian hormones protect against diabetes.

The Internist and Some Tools

Arteriosclerosis in African Populations

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MY PARTICULAR task is to review the available evidence on the incidence of arteriosclerosis in different populations in the African Continent with particular reference to whether differences in incidence can be related to genetic or environmental differences between populations.

The term arteriosclerosis is used in a very broad general sense to cover all forms of degenerative sclerosis of the arterial tree. The term atheroma is used to cover the well known degenerative lesion of the intimal and immediate sub-intimal tissues. Atherosclerosis is used to cover a related condition in which the process involves the media as well as the intima. It is typically seen in the advancing years of life (decrescent arteriosclerosis) and may or may not be associated with hypertension, renal disease or diabetes. The term Mönckeberg's arteriosclerosis is used to cover a group of cases in which calcification is widespread and radiologically visible in the large and medium sized arteries.

These four disorders are not clearly demarcated and merge into each other. They may all be idiopathic and normotensive, but may all be associated with (1) idiopathic hypertension (2) renal hypertension (3) diabetes.

All forms of arteriosclerosis may vary in the extent to which they affect respectively the aorta, the coronary arteries, the renal arteries, the arteries of the limbs, the cerebral arteries, and the pulmonary artery and its branches. There is also a variation in the respective incidence on large, medium and small arteries.

All the factors discussed above must be taken into consideration in attempting to compare the incidence of arteriosclerosis in different populations.

This communication will be confined to Africa south of the Sahara (a WHO region). The two main population groups in this region are Negro and Bantu. Hailey[1] (1938) describes the peoples of Africa as deriving from three principal stocks, Bushmen, Negro and Hamite. The Hamites are represented mostly north of the Sahara but the so-called Eastern Hamites probably invaded Africa in successive waves from Southern Arabia. The Bantu group arose from modification of a predominantly Negro race by the Hamitic influence (see map). They spread westward and southward but mainly south of the Bantu line which is never very far from the equator. The term Bantu is mainly linguistic in its significance but is associated with certain cultural traits of Hamitic origin. These people had been migrating southwards from the equator and had largely displaced the original Bushmen-Hottentot mixture of southern Africa before they met the European invading the continent from the south. Hamitic influence is absent or slight in the Negro migrations north of the equator which peopled West Africa and from which presumably the Negro people of the United States derived.

There remain small pockets of distinct and probably more primitive peoples in the Congo basin (Pygmies) and in the Union of South Africa (Hottentots and Bushmen). Europeans born and reared in the African continent are almost confined to the so-called White Highlands of Kenya and Southern Rhodesia and to the Union of South Africa. In the Union of South Africa there are approximately a million Cape Coloured people who are more or less culturally and racially homogenous on a European rather than African pattern (Brock[2] 1949).

Their derivation is mainly from the original

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Hottentot inhabitants with considerable admixture of slaves brought from Indonesia and Malaya and also with promiscuous whites. From our point of view it is important that they are genetically distinct from the Bantu.

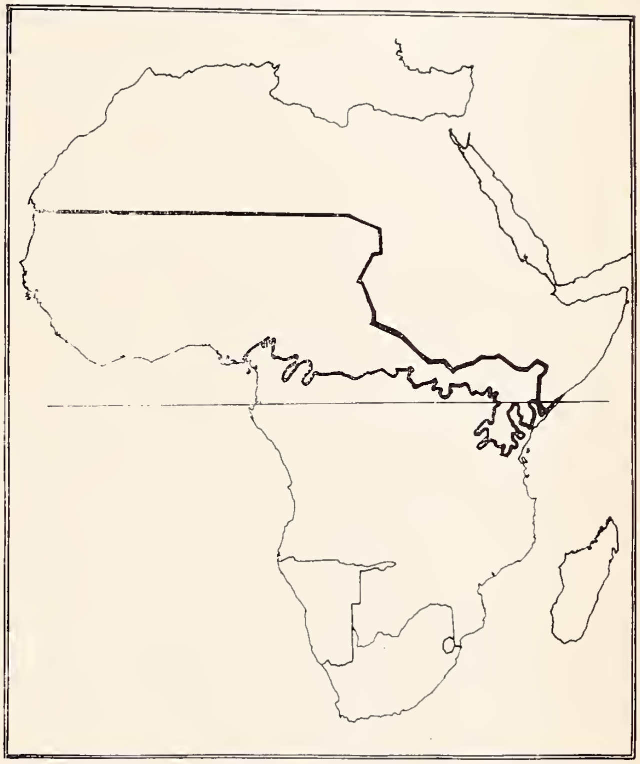
Study of the sickling phenomenon, the ABO and other blood groups, and the different hemoglobins is beginning to throw interesting light on racial origins in the African continent, but so far none of these new data have been correlated with the problem of arteriosclerosis.

Among the background publications on interracial variations two books should be specially mentioned, namely, "The Biology of the Negro" by J. H. Lewis[3] (1952) and "Civilization and Disease" by C. P. Donnison[4] (1937). The first is a general study of the appearances of disease in the American Negro. The second is a general study of world trends in disease from the particular point of view of one who had spent considerable time in the practice of medicine in Central Africa.

Published information on the subject under review from Africa is fragmentary and scattered. It has been suppemented by correspondence with internists and pathologists in certain centers whose opinions are acknowledged as personal communications. In the last year or two, under the influence of increasing interest in the relationship between diet and atherosclerosis and the blood lipoproteins, reports have been published by Walker, Higginson et al[5,6] from Johannesburg (1954) and an intensive study of three population groups in Cape Town has been carried out by a team directed by the authors. The methods used have been correlated with those used by Ancel Keys who with Mrs. Margaret Keys spent a month in Cape Town in the survey. Results of this survey will be referred to as the Cape Town survey. Acknowledgment is made to those who participated.*

It is our intention to take as a starting point the two papers published from Johannesburg in 1954 and to proceed both forward and backward from this starting point. The forward progression will confirm and expand the Johannesburg work from the experience of the Cape Town survey and will show that, using identical techniques in a single climatic and geographical environment, markedly different experiences of coronary thrombosis are found and show linear correlation with differences in serum cholesterol and lipoproteins and with dietary fat intake.

The historical review will attempt to show that coronary heart disease, at least among primitive peoples, does not necessarily show close correlation with other forms of arteriosclerosis, and



The African continent showing the WHO African Region (south of the Lahara) the Bantu line (wavy line cutting across the equator) and the borders of the Union of South Africa.

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*The Cape Town Coronary Heart Disease Survey: This survey was carried out within the municipal boundaries of the City of Cape Town from March to May 1955. It was directed by John F. Brock, professor of medicine in the University of Cape Town, and organized by B. Bronte Stewart, assistant physician at the Groote Schuur Hospital on the Joint Medical Staff with the University of Cape Town. Other medical contributors include B. Kaplan (medicine) and R. Singer (anatomy). Miss A. Moodie, research social worker in the Department of Medicine, played a very active part in the organization and conduct of the survey, and A. Antonis, Ph.D., organized the laboratory work. Acknowledgment is made to the technicians and secretaries whose enthusiasm and hard work made the survey possible and to the representatives of many employing bodies who assisted in the provision of human volunteers. Acknowledgment is made finally to Ancel Keys and Mrs. Margaret Keys for their assistance in the organization of methods and standards and for their very hard work during their stay in Cape Town. The survey was financed in part by the S. A. Council for Scientific and Industrial Research through the budget of its Research Unit in Clinical Nutrition directed by Professor Brock; in part also by research funds provided through Ancel Keys.

that the problem of coronary heart disease needs to be considered separately from the problem of arteriosclerosis in general.

In the historical review it will be necessary to refer to articles in journals that are not well known to American readers and some of which may be difficult of access. It may be an important part of our contribution to list some of these titles so that they can be consulted by those who wish to get at the original facts,

To summarize first the 1954 contribution from Johannesburg, there are companion papers by Walker and Arviddson[5] and by Higginson and Pepler.[6] The first paper shows that the blood cholesterol level of the Johannesburg Bantu is low compared with European standards and that the fat intake in the diet is low. The second paper deals with atherosclerosis and coronary heart disease in the same group of people. It should be emphasized that the authors confine themselves to a study of the coronary arteries and the aorta. They analyzed 523 unselected necropsies performed under the supervision of one pathologist at Baragwanath Non-European Hospital. Atherosclerosis was classified according to the grading of Wanscher, Clemmesen and Nielsen[7] (1951). In this series there were 150 deaths from cardiovascular disease (including tuberculous pericarditis): eighty-four men and sixty-six women. Of these cardiac deaths only eight (four men and four women) were attributed to coronary disease.

They compare the incidence of the severer grades of atherosclerosis (grades 3 and 4) in their Bantu series with the findings by the same grading in the Danish series of Wanscher, Clemmesen and Nielsen and the difference is most striking. For example, among men in the age range 40-49 the incidence of grade 3 atherosclerosis was 3 per cent in the Bantu series and 32 per cent in the Danish series, and of grade 4 1 per cent in the Bantu series and 8 per cent in the Danish series. They tried to use the same grading as the Danish workers but comment that "it is probable that several cases graded as 3 or 4 in this would have been graded less by the Danish workers." The Danish authors for example described the coronary arteries in grade 4 as being "transformed to stiff and narrow chalky pipes without elasticity" but they feel that not a single case graded as 4 by them could be justifiably so described.

In the same paper they refer to a larger series of 1,328 consecutive necropsies carried out by various members of the staff at the same Hospital over a period of five and one-half years and which includes the smaller series of 523. In this larger series there were seven cases of coronary thrombosis or myocardial infarction and one case in which the diagnosis of ischemic heart disease was considered tenable.

A most important publication on arterioclerosis in the Bantu and Coloured races of South Africa is a series of articles by Becker[8] (1946). This is a most exhaustive study by a pathologist in Johannesburg of "cardiovascular disease in the Bantu and Coloured races of South Africa." The distribution of the races is given in the first paper. It covers the various Bantu tribes of Southern Africa but includes 17 per cent of Coloured people, referred to by Becker as Eurafricans. This latter group is very similar to the group described in the Cape Town survey as Cape Coulored people, but probably has more Bantu blood.

Writing on atheromatosis, Becker states:

> "The incidence of atheromatosis in the ascending period of life in Bantu and Coloured subjects is not far removed from that in other races; the incidence of the senescent type may be slightly less. Pathological effects of atheromatosis are limited to the senescent type. The lesions tend to occur particularly in those with hypertensive heart disease."

When discussing heart disease, he says:

> "The general incidence of hypertensive arteriosclerosis is judged to be rather less than that which is found in the American Negro and rather more than in the white races of the United States. There is a steadily rising incidence with advancing age reaching a maximum in the sixth decade of 15 per cent. It is the commoneset cardiovascular disease among the Bantu and Coloured people, being responsible for one third of all cases of congestive failure and for nearly 6 per cent of all deaths. In spite of the fact that the coronary vessels are frequently diseased in the condition, and the myocardium is frequently gravely damaged, coronary thrombosis is an uncommon lesion. Similar conclusions have been arrived at in regard to the Coloured races in America."

An excellent recent general review which will not easily be available to American readers is that by Elliot[9] (1953). This study is entitled "Coronary Arteries and Their Diseases in the Bantu." The author discusses the rarity of coronary heart disease in Bantu from three angles, namely, experience of myocardial infarction, of cardiac aneurysms and of sudden deaths.

Myocardial Infarction.—Elliot had no proved cases admitted over a two-year period to 150 medical beds for non-European patients in Johannesburg. (One suspected case was found at autopsy to have syphilitic coronary ostial stenosis; one was discharged and not seen again).

Grek analyzed 6,000 Bantu patients passing through sixty-five medical beds in a period of six years. Of twenty-two cases initially diagnosed as myocardial infarction fifteen were later reasonably attributed to syphilitic coronary ostial stenosis. Of the remaining seven only three were followed to autopsy, and of these three only two were reasonably attributed to coronary thrombosis. At the same time and place in 8,000 admissions to the European Medical Wards of the Johannesburg General Hospital 1950-1951 there were 235 cases of coronary thrombosis with myocardial infarction.

Cardiac Aneurysms.—Over a twelve-year period at the Johannesburg General Hospital, the adjoining Non-European Hospital and at the Johannesburg Medico-Legal Laboratories there were fifty-seven European and fifteen Bantu cases of cardiac aneurysm. Among the fifty-seven European cases severe grades of coronary atherosclerosis were noted in every case, whereas in the fifteen Bantu cases no notable coronary atherosclerosis was found. The peak incidence for the fifty-seven Europeans was 60-69 years, whereas among the fifteen Bantu it was 30-39 years. In ten of the fifteen Bantu cases, other causes were found for the aneurysm. These were syphilis of the cardio-vascular system, six; tuberculosis of the heart, one; mycotic aneurysm, one; congenital myocardial defect, one, and Loeffler's parietal endocarditis, one. In the remaining five cases the cause of the aneurysm was not definitely established. The slight atheromatous changes could not reasonably be regarded as causative.

Sudden Deaths.—Elliot quotes figures for a six-month period at the Johannesburg Medico-Legal Laboratories. Forty-two European deaths were all due to myocardial infarction resulting from coronary thrombosis. Of forty sudden deaths in Bantu none were due to coronary thrombosis or myocardial infarction. The age range for the Bantu sudden deaths was of course much lower than that of the European sudden deaths.

Arteriosclerosis in General.—In marked contrast to the apparent rarity of coronary heart disease and of severe coronary atheroma in the Bantu is the comparative frequency of hypertension and of arteriosclerosis of other forms. Elliot's analysis can be summarized as follows:

Hypertension.—Ordman[10] (1948) examined the blood pressure of 1,522 Bantu subjects in urban and rural areas scattered over South Africa and found hypertension in about the same frequency as in the European.

Grusin and Gillanders examined 1,000 urban Bantu subjects in Johannesburg and concluded that hypertension in the Bantu is about as common as in the North American Negro. Ordman[10] and Becker[8] (1946) review the subject of hypertension in the non-European races of the world.

Arteriosclerosis, All Forms.—Elliot quotes J. C. Gilroy, V. H. Wilson, A. B. Gillanders and I. J. Grek, all of whom are experienced in Bantu medicine in Johannesburg, as agreeing that all forms of arteriosclerosis including atherosclerosis and Mönckeberg's sclerosis are common in the Bantu. Cerebral thrombosis and hemorrhage based upon hypertensive atherosclerosis are not uncommon but are perhaps a little less common than in the European. Hypertensive retinal changes of all degrees are equally common in both races. Hypertensive nephrosclerosis is probably more common in the Bantu and more severe in its effects. Peripheral vascular disease is not uncommon.

On the subject of Mönckeberg's sclerosis Elliot is quoted above as saying on the authority of several experienced people in Johannesburg that all forms of arteriosclerosis including atherosclerosis and Mönckeberg's sclerosis are common in the Bantu. Evidence has been sought from radiologists as to the incidence of recognizable calcification in the aorta and arteries of the lower legs in Bantu subjects. The impression is that it is uncommon but there is not enough evidence on which to base a firm opinion.

In our recent survey of approximately 600 adult males under age sixty in Cape Town, we found that the proportion exhibiting blood pressures over 145/90 was not significantly different (15.6 per cent European, 21.9 per cent Coloured, 20.4 per cent Bantu) in our three racial groups which were similar with regard to mean age and sample size.

From Salisbury in Southern Rhodesia, Gelfand[11] (1952) reports that in 1,500 consecutive admissions to his Bantu Hospital Wards in the last eighteen months, seventy (4.67 per cent) had systolic pressures above 150 and 101 (6.73 per cent) had diastolic pressures above 90. Unfortunately, the ages of his patients are not recorded and it must be remembered that the average age in one of his wards would be very much lower than in the average European Hospital Ward. In the same paper he quotes his own experience of the variety of heart disease in his Bantu medical wards. Among 189 cases of heart disease of all types (114 male and seventy-five female) hypertensive cardiac disease was found in twenty-eight (14.81 per cent). This can be compared with the incidence of other types of heart disease in this series; rheumatic heart disease, fifty-one (26.98 per cent), nutritional heart disease, twenty-nine (15.34 per cent), essential hypertension, twenty-eight (14.81 per cent), syphilitic heart disease, twenty (10.53 per cent), congenital heart disease, seventeen (8.94 per cent), anaemic heart disease, eleven (5.82 per cent), tuberculous pericarditis, nine (4.76 per cent), cor pulmonale, seven (3.7 per cent), arrhythmias, seven (3.7 per cent). In this series coronary heart disease, sub-thyroid heart disease and beri-beri were not encountered. He comments, "It may be safely stated that coronary heart disease (coronary thrombosis and angina pectoris) is extremely rare in the African."

An important series of contributions to knowledge of arteriosclerosis in the African comes from East Africa and particularly from Kampala, Uganda. Williams[12] (1944) in a series of papers laid the foundations while Davies[13] (1948) gathered up the clinical experience of Williams and his own experience as a pathologist in Uganda. Davies says that his experience in Uganda is similar to that published by Vint[14] (1937) who found that "minor degrees of atheroma of the aorta were general in Africans in Nairobi after the age of eighteen years, but in only 8.9 per cent of cases was the lesion well marked." At Mulago Hospital in Kampala, Uganda, Davies found "minor degrees of atheroma of the aorta were common, while in thirty-eight out of 2,994 autopsies an advanced degree of atheroma was present." Particulars for age are incomplete but of the thirty-eight cases twenty-four had an average age of 46.6 years. Davies says that "apart from their infrequency, the arteriosclerotic lesions were exactly similar to those found in Europeans." He comments also that "one of the most puzzling features of atheroma in Negroes is the rarity of coronary thrombosis." In the late 1930's various workers in East Africa had commented (Donnison, C. P.,[4] 1937; Vint, F. W.,[14] 1937; and Hennesey, R. S. F.,[15] 1938) on the comparative rarity of hypertensive heart disease and cardiovascular hypertrophy. Comparing the later work of Williams in Uganda and of Becker in Johannesburg Davies concludes that "the careful work of Williams and Becker suggests that the earlier views were ill-founded and that hypertensive disease is nearly as important to the African in Africa as it is to the African in America."

Williams[16] (1955) writing recently from Uganda says, "With regard to coronary infarction and angina of effort, my experience is entirely negative. I have never yet made a clinical diagnosis of coronary infarction in an East African which has been corroborated post-mortem. As anyone who knows Mulago Hospital will believe this is not for lack of autopsies. The occasions on which I have entertained a clinical diagnosis of coronary disease in a local African have been exceedingly few; not only is the negative autopsy experience a deterrent, but I have so rarely met with anything resembling cardiac pain in African patients here. I shall not be a bit surprised if this situation should alter in the next few decades. It is altering so radically already in some connections, e.g., peptic ulcer. I no longer regard African medicine as static."

Davies[18] (1955) has recently confirmed the above views in the following words: "While mild degrees of atheromatosis are not uncommon, a severe degree of atheroma contributing to heart failure is uncommon in East African natives although more common in the South African Bantu." He adds to this, "I have never seen a case of coronary thrombosis in an African here, and know of only one in our records since 1931. Atheroma of the cerebral vessels is very rare. Mild aortic atheroma is seen, but very rarely is there severe atheroma unless syphilis is present."

From West Africa Edington[19] (1954) confirms the low incidence of myocardial infarction in the West African Negro. In an analysis of 3,645 autopsies performed at Accra, Gold Coast Colony, during the years 1921 to 1953 he found a low incidence of cardiovascular disease. This he thought could be accounted for in part by the smaller number of autopsies in the older age groups but expressed the opinion that it is due also to the low incidence of coronary disease. Disease of the coronary arteries was considered to be the cause of death in only thirty cases (6.4 per cent of cardiovascular conditions).

The foregoing evidence shows a striking immunity of the Bantu and Negro peoples of Africa to the effects of coronary occlusion. Although their life expectation is considerably less than that of the European, the immunity is evident in the decade thirty to forty years and is too great to be attributed to lesser life expectation. This opinion is confirmed by exact figures quoted later for comparison of three racial groups in Cape Town. There is evidence, particularly in the work of Higginson and Pepler[6] (1954), that this immunity may be due to a considerably less severe degree of coronary atheroma in these African people than in Europeans. In view, however, of the fact that Africans are by no means free of some of the other manifestations of arteriosclerosis and hypertension it would be unwise to ignore other factors that may account for their immunity to the effects of coronary heart disease.

The factors which need to be considered seriously are:

1. The possibility that the African is genetically endowed with a coronary artery system which allows a freer anastomosis between the different branches, and therefore a lesser tendency to ischemia even in the presence of severe narrowing of a coronary artery.

2. That the African possesses an inherent or genetically determined peculiarity in cholesterol metabolism that protects him from coronary atherosclerosis.

3. That either for genetic or for environmental reasons the African has a lesser tendency to intravascular clotting in the coronary arterial system even in the presence of coronary atherosclerosis.

Anatomical Differences in Coronary Arterial Tree.—Brink[20] (1949) studied the possibility of a different coronary artery pattern in the Bantu. He claimed that in the Bantu there is an especially developed third primary division of the left coronary artery. This subject was fully discussed by Elliot[9] (1953) who concluded that "the finding of a difference in the Bantu and European coronary artery pattern is certainly of social and anthropological interest. The differences that have so far been demonstrated appear to have little if any bearing on the incidence of coronary artery disease and disease syndromes, the peculiarities of which in the South African Bantu remain an unsolved problem." This opinion is supported by the preliminary finding of Singer[21] (1955) who has found the same coronary branch anomalies in European hearts. A genetic difference in coronary vascular supply cannot at present be finally excluded as the cause of the African's immunity to coronary heart disease but it would be unwise to give this possibility serious consideration until the effects of environmental factors have been excluded.

Inter-racial Differences in Incidence of Thrombo-Embolic Phenomena.—An uncompleted inter-racial study at present in progress in Cape Town has concerned itself with blood clotting mechanisms.[47] This study was initiated in order to determine whether the Bantu possessed differences in clotting time to account for the widely held view that thrombo-embolic phenomena are rare in this ethnic group. In view of the findings of Strom and Jensen[22] (1951) on this aspect during the war years in Norway it seems highly probable that environmental and not genetic factors are at play. The decline in death rate in Norway during the war years occurred rather too suddenly to be accounted for by resolution of atheroma if such is possible. Furthermore the views of Morris[23] (1951) on the unchanged severity of coronary atheroma over the last four decades again emphasize that more attention should be paid to intravascular clotting in determining the effects of coronary heart disease.

Inter-racial Differences in the Incidence of Coronary Heart Disease and the Mean Levels of Serum Cholesterol in Cape Town.—We cannot subscribe to the view that the geographic or climatic environment in any way accounts for the

TABLE I. MEANS AND STANDARD DEVIATIONS FOR CLINICALLY HEALTHY MEN AGED 40 TO 58 YEARS IN THE CAPE PENINSULA

	Bantu	Coloured	European
No. of Subjects	132	118	114
Mean Age	45.8±5.5	46.5±5.0	47.0±5.2
Mean Total Serum Cholesterol mg per 100 ml	166.3±47.2	204.1±54.8	234.0±52.9
B Lipo-Protein Fraction	122.3±47.9	160.5±54.4	194.5±52.8

All of the differences shown (except mean age) are statistically highly significant.

TABLE II. STANDARDISED MORTALITY RATIOS OF MEN AGED 20-64 (1950) GREAT BRITAIN

	Social Class				
	I	II	III	IV	V
Coronary heart disease	150	110	104	79	89

Standard mortality ratio may be defined as the number of deaths occurring among men aged 20-64 in a given occupation expressed as a percentage of the number of deaths that might have been expected to occur if the given occupation had experienced within each age group the same death rate as that of a standard population consisting either of all males or of all occupied and retired males only. Constitution of the social classes is I—professional etc., II—intermediate, III—skilled, IV—partly skilled, V—unskilled occupations.[28]

differing incidence of coronary heart disease and the different levels of serum cholesterol that have been reported from studies on different populations. Europeans in South Africa whose mode of life is not unlike that of individuals of Western European descent residing in Western Europe and Northern America possess a mean serum cholesterol and death rate from coronary heart disease not unlike that of the Northern American. In Cape Town the death rate ascribed to coronary heart disease in the individual of pure European descent is more than twice that of the Cape Coloured in the age group thirty-five years and over (M.O.H. Annual Report[24] 1946). In the Bantu townships of this city, the registration of a death as being due to coronary heart disease is an exceedingly rare occurrence. Possibly one to three are registered per year (M.O.H. Annual Report[25] 1925) in an estimated population of over 100,000 Bantu. The comparative rarity of the disease is reflected in our hospital statistics, clinical observations and post-mortem studies. Vogelpoel and Schrire[26] (1955) recently have analyzed over 5,000 electrocardiograms taken on adults at the Cardiac Clinic at Groote Schuur Hospital. This hospital caters for the poorer section of the community of Cape Town and the hospital attendance figures shows a bias in favour of the non-European. Despite this, of the 550 classical myocardial infarction electrocardiogram patterns, 448 were in Europeans, 100 in Cape Coloured and only two in Bantu: The numbers of electrocardiographs requested by physicians for the different racial groups are very different indicating a greater expectation by physicians of abnormal patterns in the European than in the non-white groups. When the figures are expressed as myocardial infarct patterns per 100 electrocardiographs taken on each racial group the figures are European 13.54, Cape Coloured 6.80, Bantu 0.9. When they are analyzed per decade of age from 30 to 59 years the distribution is not significantly different for white and combined non-white people. These figures should dispel any lingering inclination to doubt the remarkable immunity of the Bantu people to myocardial infarction. The geographic or climatic environment cannot explain these wide differences between the European and non-European populations in Cape Town. Other environmental factors must be at play.

Cape Town is favourably situated for an interracial study as each race in the main, still adheres to its traditional customs. Consequently a large number of environmental factors become available for study. During March to May, 1955, we sampled approximately 200 working adult males under the age of sixty years, from each of the three main racial groups, to study some of these environmental factors. The study involved clinical, electrocardiographic, anthropometric, socio-economic, hematological and bio-chemical examinations. A summarized version of the results in the middle aged group now follows.

Of the 383 men aged between 40 and 58 years there were only nineteen exclusions due to probable ill health. One European had xanthoma tuberosun, there was one diabetic in each racial group and the remaining fifteen were excluded on clinical or electrocardiographic evidence of probable present or past experience of coronary heart disease. Commensurate with our clinical and hospital experience the proportion of these fifteen exclusions in Bantu, Cape Coloured and European was respectively 1 to 5 to 9. Furthermore the one Bantu had doubtful electrocardiographic changes only.

The mean serum total and β lipoprotein cholesterol levels for these three ethnic groups of like mean age show a pattern with the Bantu at one extreme and the European at the other. The Cape Coloured lie in-between (Table I). The differences are highly significant ($P<0.001$). The problem immediately arises whether these cholesterol differences are due to genetic or to environmental differences between the three racial groups

Over the last two decades evidence has accumulated that wide differences in susceptibility and whether the differences in coronary heart disease have the same cause. to coronary heart disease and different levels of serum cholesterol have occurred not only between ethnic groups but also within a given ethnic group. The last world war contributed greatly through the changing mortality experience (Strom and Jensen[22] 1951) and changing serum cholesterol levels (Schettler[27] 1950) in Europe. This is also illustrated by the decennial occupational mortality statistics of England and Wales (Registrar General[28] 1954). This publication shows the susceptibility of the professional and executive classes in particular (Table II). The fact that a similar trend exists in wives over sixty-five years of age and in retired men suggests that socio-economic factors are of greater importance than the nature of the occupation. Of the many social factors, economic privilege becomes the most outstanding.

It was of interest to us to study whether economic privilege affected the serum cholesterol pattern within each racial group. Subdivision into what could be considered as high, middle and low income levels for the particular ethnic group led to a very wide inter-racial economic range with low income Bantu at one extreme earning less than eight dollars a week to high income. Europeans earning 100 to 200 dollars a week. Within each ethnic group the more privileged economically had higher mean serum total and especially β lipoprotein cholesterol levels (Fig. 1). Genetic racial influences now appear less important when one notes that individuals on the same economic plane, irrespective of their racial origin exhibit similar cholesterol levels.

Apart from income, many other environmental factors, some related to the socio-economic status, distinguish our three ethnic groups. Many of these have at some time or another been advanced as factors in the pathogenesis of coronary heart disease. Amongst these, exposure to infection, the degree of physical and mental activity, the consumption of alcohol and tobacco, obesity and the diet have all received consideration in this survey. The detailed results have been published elsewhere (Bronte-Stewart, Keys and Brock[29] 1955) and Bronte-Stewart, Moodie and Brock[30] 1955). In brief, trends that are systematized, i.e., with Bantu at one extreme, European at the other and Cape Coloured in-between, do exist for some of these factors. Some correlate positively with the rise in serum cholesterol and the increase in susceptibility to coronary heart disease from Bantu through Cape Coloured to European. Others show a negative correlation in that there is a systematic decrease in magnitude. A few factors show no systematic trend.

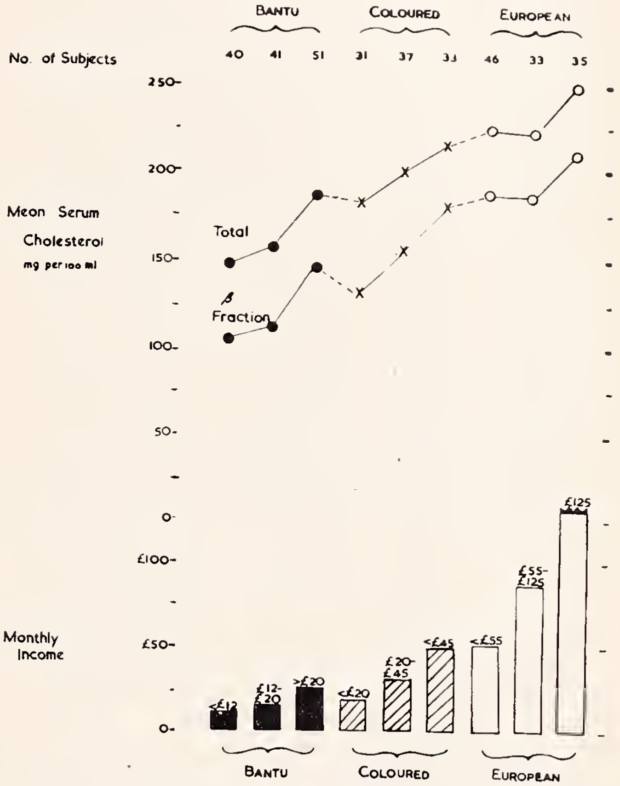

Fig. 1. Mean values for serum total cholesterol and cholesterol content of the beta lipoprotein fraction for the three racial groups each classed according to monthly money income into three sub-groups as follows: *Bantu* less than 35 dollars, 35 to 55 dollars, over 55 dollars; *Coloured* less than 55 dollars, 55 to 125 dollars, over 125 dollars; *European* less than 155 dollars, 155 to 350 dollars, over 350 dollars.

The negative trends are the proportion with elevated erythrocyte sedimentation rates, the proportion classified as heavy manual labourers, the proportion of heavy pipe tobacco smokers and the proportion of non-cigarette smokers. The positive trends are the proportion of sedentary workers, the proportion in employment involving supervisory responsibility, the proportion who are heavy cigarette smokers and the proportion of calories derived from fat. The factors studied that show no systematic trend are alcohol consumption, non-smoking and the degree of obesity as measured by the arm skinfold.

When each ethnic group was subdivided on the basis of income, only two trends remained systematized from low to high income and from Bantu through Cape Coloured to European. These were the degree of supervisory responsibility in the work and the consumption of fat containing foods.

Provided due allowance was made for race and income level there were no significant differences between the mean serum cholesterol levels of individuals with and without elevated erythrocyte sedimentation rates, and of individuals in heavy or light physical labour. The close association between the increase in heavy cigarette smoking and the rising incidence of coronary heart disease (Cuyler Hammond and Horn[31] 1954) was not borne out in our series with respect to the serum cholesterol levels. Nearly twice as many medium income Europeans are heavy smokers as compared with low income Europeans, yet their mean serum cholesterol levels are almost identical. Obesity was as common in the high income Bantu as in the European and in the latter the proportion who were obese was identical in each income group. If these factors do operate in the pathogenesis of coronary heart disease, they must do so through mechanisms other than the serum cholesterol.

Mental stress has often been advanced as a factor in the rising incidence of coronary heart disease though no convincing evidence of this is at hand (Arnott[32] 1954). The greater susceptibility to coronary heart disease of the London bus driver as opposed to the conductor (Morris et al[33] 1953) could as easily be attributed to the greater degree of mental stress as to the lack of physical exertion in the driver. It could explain the susceptibility of the professional and executive occupational classes to coronary heart disease. There is evidence too, that acute emotional stress may shorten the clotting time (Garb[34] 1955). The drop in the incidence of coronary heart disease during the war years in the occupied countries (Strom and Jensen[22] 1951) is often quoted as evidence that prolonged mental strain plays no dominant part. The considerable number of physiological reflexes that come into play in reaction to acute mental stress, are not necessarily exhibited in a reaction to prolonged stress. One of the reactions to acute stress is a rise in blood pressure and another is a rise in serum cholesterol (Kuhl et al[35] 1955). Experimentally it has been shown that only a slight rise in blood pressure or a very labile blood pressure leads to a greater degree of atheroma in rabbits (Bronte-Stewart and Heptinstall[36] 1954) and the hypertension need not be long sustained to produce this effect (Wilens[37] 1943). Furthermore the hypertensive rabbits had slightly higher mean serum cholesterol levels throughout (Heptinstall and Bronte-Stewart[38] 1954). Objective analysis of mental stress as applied in our three racial groups ranging from poverty to wealth is an extremely difficult task. If the lack of any supervisory responsibility at work be taken as a measure, then good correlation emerges with the serum cholesterol levels and our experience of the inter-racial incidence of coronary heart disease.

There remained the diet. The customary Bantu diet, inadequate in certain amino-acids and vitamins carries with it the risk of chronic protein malnutrition with its attendant liver fibrosis, gynecomastia and testicular atrophy. With hormonal theories prominent in coronary heart disease clinical signs of endocrine deficiency were diligently searched for but never found. If birth rate is an index of androgenic activity in the male, perhaps a dubious concept, the Bantu and Cape Coloured are certainly not defective. The customary Bantu diet has yet another link with current problems in the pathogenesis of coronary heart disease. Being deficient in the sulfur-containing amino acids in particular, it provides the Bantu with a dietary protein background not unlike that on which Mann and his co-workers[39] (1953) produced atherosclerosis with cholesterol feeding in the cebus monkey.

Our test for liver disease (Mallen et al[40] 1950) was universally negative, and Walker and Arvidsson[5] (1954) could find no differences in the mean serum cholesterol levels in the presence or absence of liver disease. Calorie excess with its expression in obesity shows no systematized trend.

Batson[41] (1953) had shown that the Europeans consume twice the amount of fat as measured in percentage of total calories derived from fat (35 per cent) as the Bantu (17 per cent). The Cape Coloured lie in-between (25 per cent) (Fig. 2).

Our simultaneous dietary survey, semi-quantitative in that it was assumed that each individual consumed an "average" helping, showed a similar ratio of fat consumption. These marked differences in fat consumption according to our survey were due primarily to an increased consumption of fat-containing foods of animal origin while the differences between the mean serum total cholesterol levels were due to differences in the cholesterol content of the beta lipoprotein fraction (Fig. 1). When these ethnic groups were subdivided on the basis of income, it became apparent that the more economically privileged consumed greater amounts of animal fat and in many instances this appeared to result from the substitution of animal fat for vegetable fat in cooking methods. Subdivision on the basis of income revealed a striking parallelism between the mean serum cholesterol levels and the animal fat intake.[29] Subgroups on the same economic plane, but of different racial origin, possessing similar mean cholesterol levels, consume similar quantities of animal fat. Low and middle income Europeans, the former residing in sub-economic housing estates and the latter attempting to ape their higher income counterparts with regard to the standard of living, consume similar quantities of animal but not vegetable fats. Their mean serum cholesterol levels are almost identical.

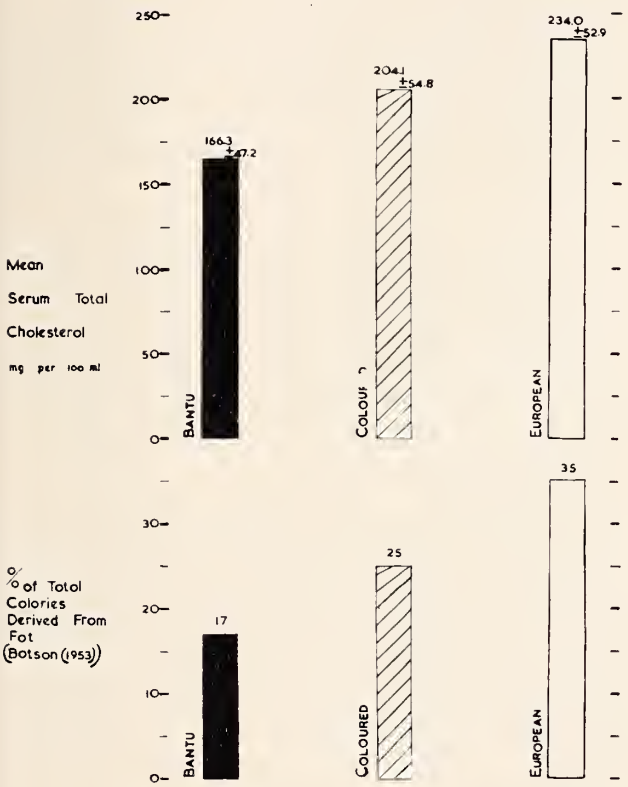

Fig. 2. The relationship between the mean serum total cholesterol of each ethnic group and the percentage of total calories derived from fat.

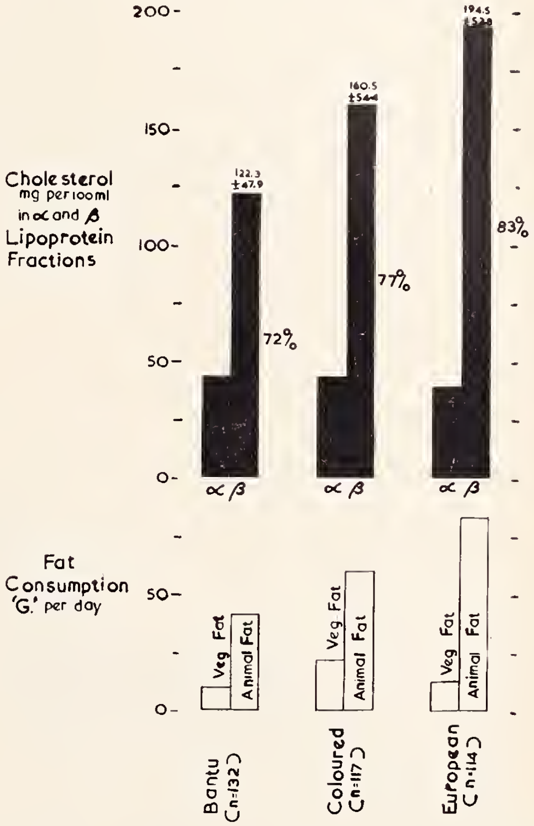

Fig. 3. A rise in the animal fat consumption parallels the rise in the cholesterol concentration of the beta lipoprotein fraction and the mean percentage of the total serum cholesterol located in the beta lipoprotein fraction.

In no instance did the mean serum total or beta lipoprotein cholesterol levels correlate well with the vegetable fat intake in this survey. This adds weight to the studies on vegetable fat feeding by Groen et al[42] (1952) and Kinsell[43] (1954), and the studies on true vegetarians by Donath et al[44] (1953) and Hardinge and Stare[45] (1954). It is possible that the conflicting views on this matter (Mayer et al[46] 1954) may have arisen from insufficient attention being paid to the nature of vegetable and fat products and the general pattern of the diet.

The results of our Cape Town survey add weight to the theory that the dietary fat intake influences the level of the serum cholesterol, particularly that in the beta lipoprotein fraction and in turn may be one of the major factors influencing the pathogenesis of coronary heart disease. This close parallelism offers the greatest hope that this disease may eventually be controlled and prevented. Certainly there are few problems that merit more attention at the present time.

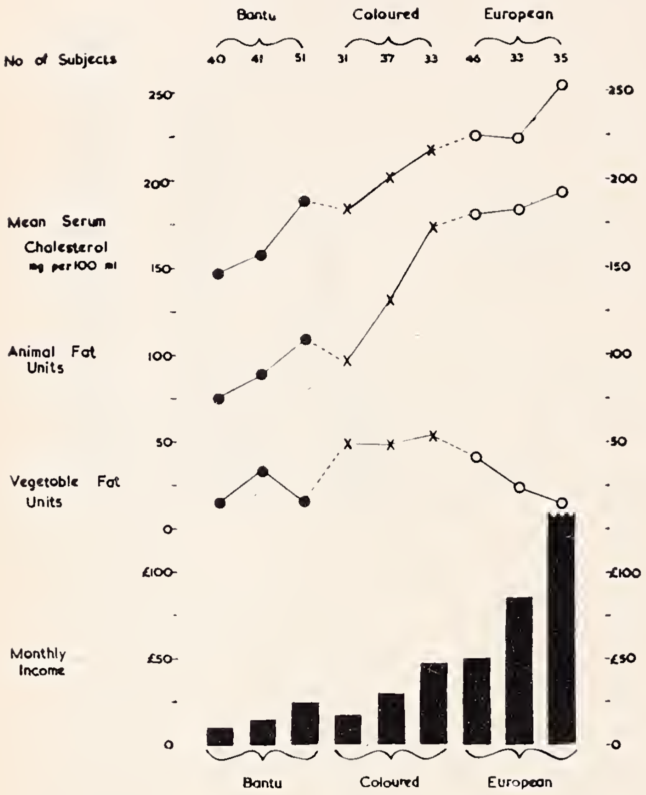

Fig. 4. Mean values for serum total cholesterol and weekly dietary fat intakes (1 fat unit=3.15G (average value) for the three racial groups, each classed according to monthly money income into three sub-groups as follows: *Bantu* less than 35 dollars, 35 to 55 dollars, over 55 dollars; *Cape Coloured* less than 55 dollars, 55 to 125 dollars, over 125 dollars; *European* less than 155 dollars, 155 to 350 dollars, over 350 dollars.

Conclusions

1. The results of the Cape Town survey[29] reinforce the conclusions from previous surveys in Africa that the Bantu have a remarkable freedom from coronary atherosclerosis and effects of coronary occlusion.

2. The analyses of electrocardiograms (Vogelpoel and Schrire) shows this freedom to be real for the Bantu in Cape Town. It shows also that the Cape Coloured people are intermediate between the European and the Bantu.

3. The comparative experience of coronary heart disease between these three racial groups is paralleled by (a) mean serum total and β lipoprotein cholesterol (b) total and animal, but not vegetable fat intake in the diet.

4. Comparison of low, medium and high monthly money income subgroups within each racial group shows similar parallelism and suggests that the parallelism between coronary heart disease and serum cholesterol fractions is dependent upon socioeconomic rather than upon genetic factors.

5. There are many possible explanations for the parallelism of trends, which do not necessarily indicate causative associations.

6. The environmental factors related to economic status which justify further examination are (a) stress and strain which is difficult to measure (b) exercise and lack of exercise (c) smoking and (d) diet.

7. In the Cape Town survey the diets which, through economic privilege, are associated with rising levels of blood cholesterol are characterized by rising fat/calorie ratios, due to rising quantities of animal, but not of vegetable, fat. They are characterized also, however, by rising quantities of animal protein foodstuffs with attendant mineral and vitamin patterns and by decreasing quantities of starch and cellulose.

8. The dietary and other environmental factors which are associated with rising mean levels of blood cholesterol and its fractions might determine a rising incidence of coronary heart disease both through the promotion of coronary atherosclerosis and through the promotion of more ready coagulation of blood within the coronary arteries.

Summary

1. Survey of populations in Africa shows a remarkable freedom of Bantu and Negro from coronary heart disease (myocardial infarction) and from coronary atherosclerosis.

2. These races are by no means free from idiopathic and symptomatic hypertension with its attendant arteriosclerosis (cerebral and renal).

3. The freedom from myocardial infarction is far greater than can be attributed to lower average age and life expectation.

4. It is due in part at least to milder degrees of atherosclerosis of the aorta and coronary arteries.

5. There is no satisfactory evidence that it can

be attributed to genetic differences in coronary vascular pattern and potential collateral channels.

6. There is some unproven evidence of lesser tendency to intravascular clotting.

7. A controlled interracial study is reported from Cape Town on three racial groups, and tentative conclusions are drawn about the causes of inter- and intra-racial parallelisms between myocardial infarction, serum cholesterol fractions, dietary fat intake and socioeconomic status.

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Little Pamela Schmidt of Minneapolis was born with a gaping hole in the wall separating the pumping chambers of her heart. Apparently normal at birth, Pamela was five months old before doctors discovered her condition—a condition for which there was no known treatment at that time (1950).

For four years, Pamela's mother hoped and prayed for discovery of surgery that would enable her child to live. During the same span, a young team of University research scientists worked hard on a new method of direct-vision surgery within the heart.

Their answer came dramatically on April 23, 1954. The answer was a donor—a donor whose heart would pump the blood supply for two human beings while the original patient's "dry" heart could be mended. Four-and-one-half hours with her father as the donor, and Pamela was on the road to normal life. Today, she lives the happy life of any other little girl of six.

Six years of heart research produced the successful operation which healed Pamela's heart. Today, a human donor is no longer needed. Further research has produced an oxygenating machine which does the donor's job artificially.

Treatment of Hypercholesteremia

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GERHARD WIGAND
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THE frequency with which severe hypercholesteremia is associated with atherosclerosis in relatively young ages definitely argues for a causal relationship. It is therefore but natural that many attempts have been made to control hypercholesteremia. Many ways have been tried and several therapeutic methods based on more or less scientific grounds have been described. Unfortunately, most of these methods have failed to produce anything like reliable results. This is perhaps not so surprising if it be borne in mind that effective treatment of a disease or symptom requires above all clear idea of the cause, and, as far as hypercholesteremia is concerned, our knowledge is still scanty. One thing, however, seems to be certain, and that is that more than one factor is capable of causing hypercholesteremia; therefore we can hardly expect to find a universal therapy, i.e., treatment that will give relief in all cases.

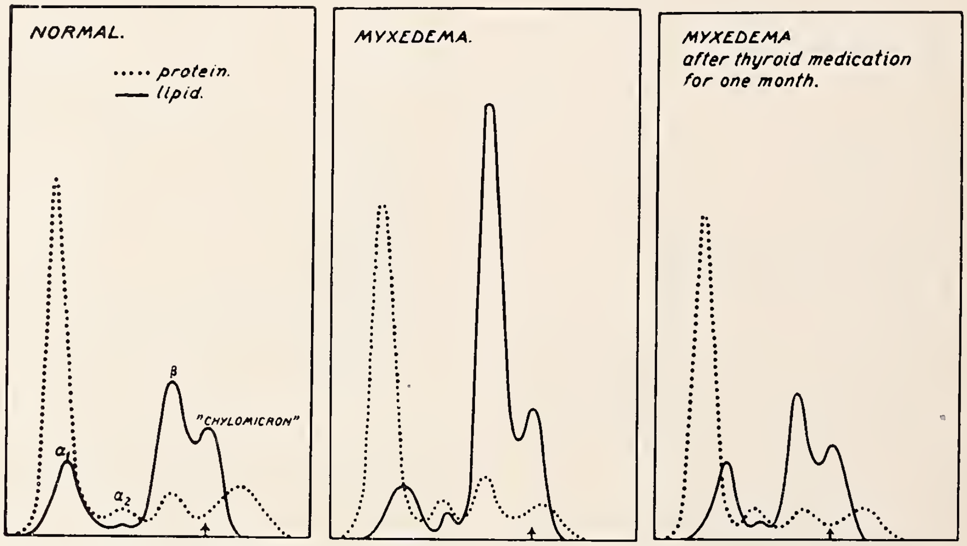

Fig. 1. *(left)* Electrophoretic pattern of normal serum. The lipids are stained with Sudan black according to the method of Swahn.

Fig. 2. *(center)* The lipid spectrum in a case of myxedema shows a very high β peak. Total cholesterol 769 mg./100 ml.

Fig. 3. *(right)* Same case as in Figure 2 after treatment with desiccated thyroid for one month. Total cholesterol: 210 mg./100 ml.

The type of hypercholesteremia regularly seen in untreated myxedema is most likely due to deficiency of the thyroid hormone. It is true that it has not yet been definitely cleared up how hypercholesteremia develops in these cases, but the administration of desiccated thyroid in sufficient doses will bring the serum cholesterol down to normal level within a few weeks. At the same time the blood lipid spectrum will be normalized, a change readily demonstrable by electrophoresis in a filter paper medium by the method of Swahn[1-6] (Figs. 1, 2 and 3). There is a danger in suddenly increasing the metabolism of myxedematous patients who have had angina pectoris or other cardiac symptoms. Several cases of myocardial infarction are on record in which a large initial dose of thyroid preparation had a fatal effect. Therefore, the experienced clinician proceeds carefully and starts the treatment with a very small dose. These fatal cases also provide a

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From the Department of Medicine, University of Lund.

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warning against a less critical use of desiccated thyroid in the management of hypercholesteremia in association with other diseases in which the basal metabolism is not decreased, such as in essential familial hypercholesteremia, especially if the patient has had myocardial infarction or angina pectoris. Moreover, in these cases thyroid compounds do not produce a very impressive effect on the hypercholesteremia.

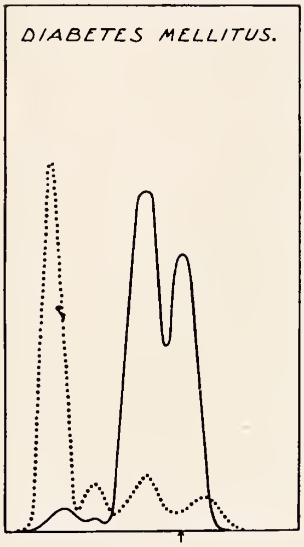

Fig. 4. The lipid spectrum in a case of diabetes mellitus shows a marked increase in both the β-fraction and the "chylomicron"-fraction.

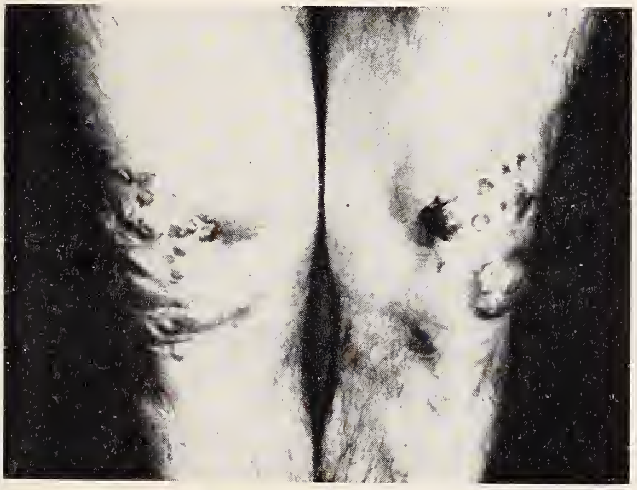

Fig. 5. Xanthoma tuberosum in a case of essential hyperlipemia.

As known, hypercholesteremia is fairly common in diabetes, especially in uncontrolled cases with severe ketosis. The serum is then often creamy, which shows that the neutral fat is also markedly increased. The lipid curve shows a high β peak as well as a high peak for the "chylomicrons" (Fig. 4). The adequate treatment of these cases is to control the disorder of the carbohydrate and fat metabolism by the administration of insulin in sufficient doses. On such treatment the hyperlipemia and the hypercholesteremia usually disappear within a short time. In some cases of diabetes, however, hypercholesteremia persists despite an apparently satisfactory dose of insulin. In these cases the serum is as a rule not creamy. In such patients the hypercholesteremia might have nothing to do with the diabetes but possibly be a manifestation of essential, familiar hypercholesteremia.

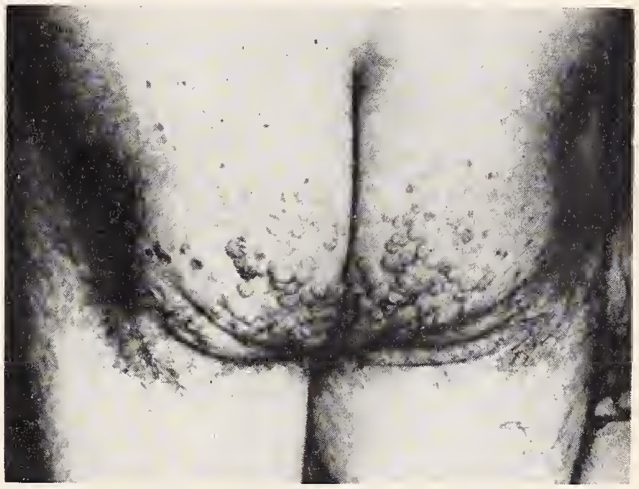

Fig. 6. Eruptive xanthomata over the buttocks in the same case.

Creamy serum is also sometimes seen in essential hyperlipemia. Then the neutral fat is markedly increased, an increase accompanied by a pronounced hypercholesteremia. Cutaneous changes are often seen in the form of xanthoma tuberosum—yellow, pea- to nut-sized nodules, most commonly located on the extensor aspects of the elbows and knees, as well as numerous eruptive xanthomata over the buttocks (Figs. 5 and 6). Tendon xanthomata may sometimes occur, though less frequently. Xanthelasma on the eyelids is also rare. These cases of essential hyperlipemia thus differ distinctly from those of essential familial hypercholesteremia in which the cholesterol deposits are located mainly in the tendons and on the eyelids. Essential hyperlipemia was formerly regarded as a fairly rare disease. Until last year there were only thirty cases on record in the literature. But in a fairly short time we have been able to collect as many as ten cases.[6] Last year Lever and co-workers[7] reported seven cases. Gofman and associates,[8] likewise in 1954, published liproprotein analyses of nine cases as well as of twenty-three cases with xanthoma tuberosum, in which ultracentrifugal analysis showed essentially the same lipoprotein spectrum. Gofman expressed the opinion that patients with xanthoma tuberosum should be assigned to a special group and that only those patients with creamy serum but without cutaneous changes should be classed under the heading of essential hyperlipemia. Personally, we think that this classification is artificial since we have found that of one and the same family, some members had xanthoma tuberosum, while others had a milky serum but not skin lesions. Thus here is an analogy to familiar hypercholesteremia, in which some members of a family have tendon xanthomata, while others have only hypercholesteremia.

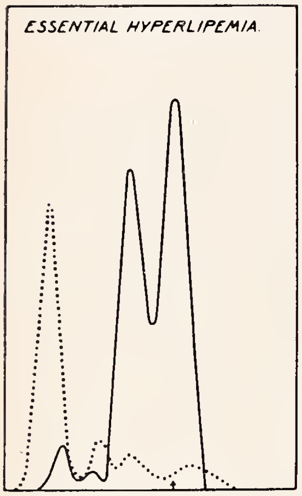

Fig. 7. *(left)* The lipid spectrum in essential hyperlipemia shows a very high "chylomicron" peak. The β-fraction is also increased.

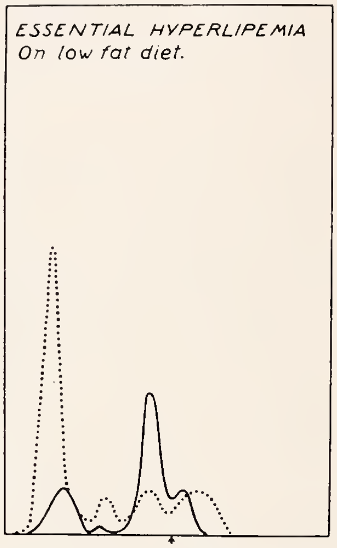

Fig. 8. *(right)* Same case as in Figure 7 after treatment with low fat diet.

Essential hyperlipemia has received increased attention since it has been shown that, contrary to the view of Thannhauser,[9] patients with this condition are by no means spared from atheromatous changes of the arteries.[6] Of our patients six had angina pectoris and three of them had also had myocardial infarction. In such cases a correct diagnosis is of extreme importance, because very effective treatment is available for essential hyperlipemia. This treatment consists simply in the restriction of fat intake to a reasonable minimum. Already within a few weeks of low fat diet the serum becomes clear. The serum cholesterol also drops to normal level, and if the dietary restriction is strictly observed by the patient for a long time, the cholesterol values and the lipid spectrum will become normal (Figs. 7 and 8). The skin changes can also disappear, and what is particularly encouraging is that the patients can obtain complete relief from their angina pectoris. The management of these cases is thus very rewarding.

Essential familial hypercholesteremia is a common disease, much more common than widely supposed. Systematic examination of the serum cholesterol level in all patients seen because of cardiac infarction or who have angina pectoris will often show increased value. If, in all those cases with a cholesterol value of more than 300, we examine all the brothers and sisters and other relatives for serum cholesterol, we often find distinct hypercholesteremia in one family member after the other. In many of them, the condition is symptomless for a long time. Many, however, have tendon xanthomata, xanthelasma and arcus corneae. Despite the high cholesterol level, the serum is clear and contains a relatively small amount of neutral fat. The most important thing is, of course, that the prognosis of familial hypercholesteremia is very serious because of the regular occurrence of atherosclerosis of the coronary arteries.

The treatment of these cases has been discouraging. A vast number of remedies have been suggested. We have tried choline, inositol and methionine without any definite effect. Heparin and estrogens do not appear to control the serum cholesterol level effectively. On the other hand, Barr[10,11] and others have shown that the administration of estrogens can modify the abnormal lipid spectrum (Figs. 9 and 10). However, when administered in large doses to men, the female sex hormone produces such troublesome side effects that the method can hardly be used routinely.

Sitosterol and other plant sterols will prevent the absorption of dietary cholesterol in chickens and rabbits. We have tried a phytosterol compound (6 gm. per day) in some cases of essential hypercholesteremia, but have in most cases only seen a transient effect on the serum cholesterol.

In 1954, Cottet, Mathivat and Redel[12] described a new compound, sodium-phenyl-ethyl-acetate, which was said to be able to inhibit the synthesis of cholesterol and thereby to suppress the blood cholesterol level. We have had the opportunity of trying this compound on some of our patients with hypercholesteremia, but we have not noticed any definite effect (Fig. 11).

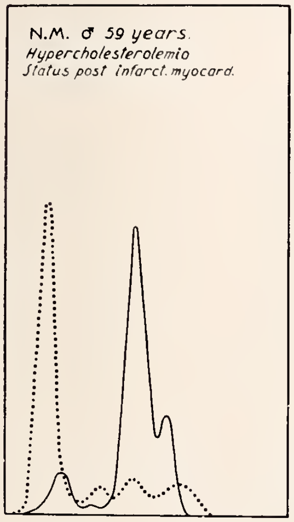

Fig. 9. *(left)* The lipid spectrum in essential familial hypercholesteremia shows a high β-peak.

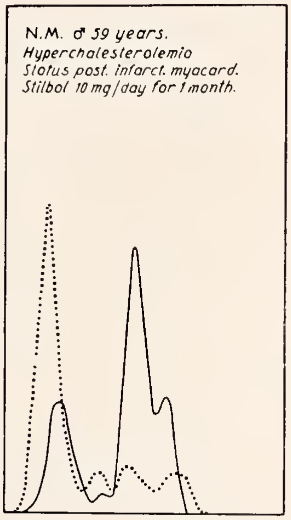

Fig. 10. *(right)* Same case as in Figure 9 after treatment with oestrogen for one month. The α-fraction has increased. There is some decrease in the β-fraction.

Dietary measures have long been tried in the management of familial hypercholesteremia. Moderate restriction of the fat intake will not produce any impressive effect, but if the patient can live on a practically fat-free diet, such as Kempner's rice-fruit-diet, the blood cholesterol can be reduced considerably. In practice, however, it is difficult to live on such a diet for any length of time. Neither is it sure whether strict observation of such a dietary regime might not with time involve certain risks.

A question that has recently been the subject of much discussion is whether vegetable fat has the same effect on the serum cholesterol level as animal fat. Groen and associates[13] found an exclusively vegetable diet to reduce the blood cholesterol even if the fat content of the diet is fairly high. Kinsell[14-18] has also found that vegetable fat does not increase the cholesterol level but even decreases it. Keys and associates[19-22] and Hildreth[23,24] as well as others, however, are of the opinion that both vegetable fat and animal fat enhance the blood cholesterol. Ahrens,[25] on the other hand, recently came to the same conclusion as Kinsell that vegetable fat decreases the blood cholesterol.

It is difficult to draw any conclusions from these reports about the possibility of treating familial hypercholesteremia with a diet containing only vegetable fat. Some of the patients in whom the use of vegetable fat was followed by an increase in the serum cholesterol had previously been on a low fat diet. Ahrens' series consisted of obese patients who did not have hypercholesteremia. Kinsell's series included one case of familial hypercholesteremia. In that patient the total cholesterol decreased from 420 mg. to 260 mg./100 ml. in the course of the experiment, which lasted thirty days.

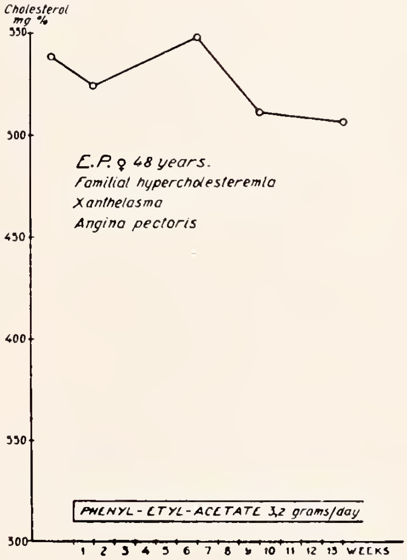

Fig. 11. A case of familial hypercholesteremia treated with phenyl-ethyl-acetate for three months without any obvious effect.

During the last few months we have had the opportunity of trying the value of a diet containing abundant vegetable fat in some cases of hypercholesteremia. The vegetable fat was given in the form of artificial milk consisting of corn oil and fat free milk powder, cheese prepared from corn oil, skim-milk and fat free milk powder and ice cream, likewise made of corn oil, milk proteins and sugar. The patients also received bread, cereals, fruits, vegetables, potatoes, rice, spaghetti and sugar ad libitum, although not more than was necessary to fulfill the caloric requirement. Adequate amounts of iron and vitamins were given. In the beginning of our experiments some of the patients also received margarine made from coconut oil and rape oil with no admixture of animal fat.

We tried this vegetable fat diet on different types of hypercholesteremia and also on some cases with normal serum cholesterol. The commencement of the vegetable fat die was regularly followed by a distinct fall in the serum cholesterol.

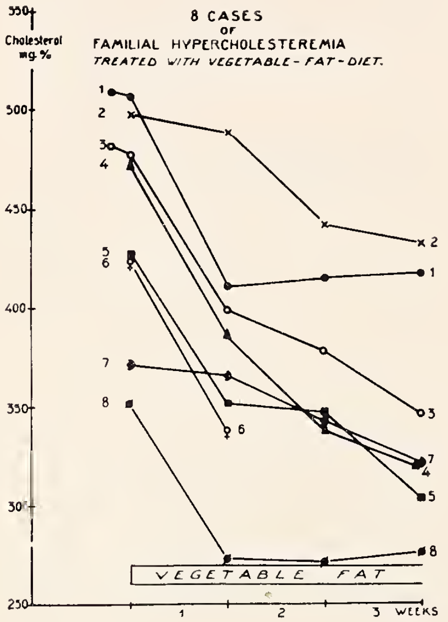

Fig. 12. Eight cases of essential familial hypercholesteremia treated with vegetable fat diet.

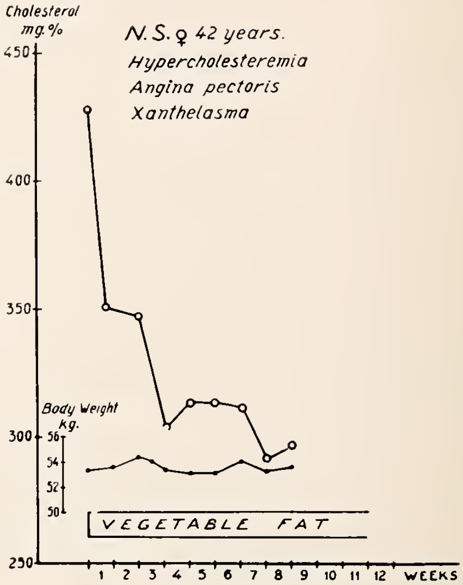

Fig. 13. A case of essential hypercholesteremia treated with vegetable fat diet for two months.

Of special interest are eight cases of hypercholesteremia of the essential, familial type, as such cases do not respond very well to other therapy. Four of them belonged to one and the same family. Two had tendon xanthomata, six had angina pectoris and of these, four had had myocardial infarction. As shown in Figures 12 and 13, the cholesterol fell abruptly, when animal fat was replaced by vegetable fat. Most of the patients have not yet been on the diet for more than a few weeks. It being known from similar experiments with low fat diet that the serum cholesterol often increases after a temporary decrease, the trial will be continued for a long time.

One of the patients had fairly severe gastric distress during the test period. The patient thought it might have something to do with the corn oil "milk," although the flavor of this preparation was not disagreeable. It turned out, however, that he had a peptic ulcer. Gratifying the desire of the patient we then changed the diet. He received a modified ulcer diet in which part of the corn oil "milk" was replaced by vegetable margarine. He still received no animal fat. On this regime the ulcer healed in four weeks.* When placed on the modified ulcer diet, the cholesterol, however, gradually rose again to the previous level. We tried to counteract this increase by administration of a phytosterol compound but it had only a transient effect on the serum cholesterol (Fig. 14).

It is, of course, not possible from this single experiment to say whether the rebound of the serum cholesterol to the previous level had anything to do with the change in the diet or whether it was spontaneous. However, in some other cases we also observed a similar effect, i.e., when vegetable margarine was added to the diet or when it was used instead of corn oil, the serum cholesterol increased. For that reason we have recently eliminated margarine from the diet and use nothing but corn oil.**

These observations suggest that not all kinds of vegetable fat produce the same effect on the serum cholesterol. This might also help to explain some of the apparently contradictory results obtained by various workers in this field.

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*Later on we tried a modified ulcer diet including abundant corn oil "milk" in other cases of peptic ulcer with satisfactory results. The ulcers have healed in three to four weeks.

**Some weeks ago, when Ahrens[26] visited Lund, he told us that investigations at the Rockefeller Institute in New York had shown that coconut fat does not produce the same decrease in serum cholesterol as corn oil.

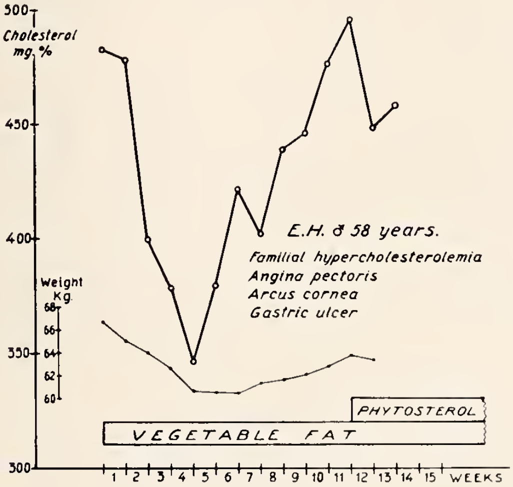

Fig. 14. A case of familial hypercholesteremia treated with vegetable fat for three months. When a gastric ulcer was discovered after one month the diet was changed to a modified ulcer diet in which part of of the corn oil "milk" was replaced by vegetable margarine. He still received no animal fat. The serum cholesterol curve rose again in the next two months to the previous level.

Many different factors may influence the cholesterol metabolism; it is therefore important that the experiments are kept as clear cut as possible in clinical investigations of this type. Wilkinson, Boyle, Jackson and Benjamin[27] have shown that a preceding period with low fat diet can influence the results of a later high vegetable fat diet. This is also evident from one of our cases of essential hyperlipemia (Fig. 15).

This patient had been on a low fat diet for years during which his earlier creamy serum had become clear and the cholesterol values normalized. But when we placed him on a high vegetable fat diet the serum again turned creamy and the cholesterol values increased. We stopped the trial and again placed him on a low fat diet. The patient was then sent home. However, some time later he got tired of the low fat diet and began to eat ordinary food including butter, eggs and other animal fat in fairly large amounts. Already within a few weeks the serum was creamy and the cholesterol values had risen to 640 mg./100 ml. The patient was again placed on a vegetable fat diet and this time the response was different: although the serum was still milky, the cholesterol decreased.

The way in which vegetable fat influences the cholesterol metabolism is not known. It is hardly the absence of cholesterol in the vegetable fat that is of decisive importance. The favorable results obtained with corn oil might possibly be due to the abundance of essential fatty acids in this kind of fat. As much as 57 per cent of corn oil consists of linoleic acid. According to Holman,[28] the essential fatty acids play an important rôle in the transport of cholesterol. The effect of various fatty acids on the level of the blood cholesterol and on the development of arteriosclerotic changes in the arteries constitutes a particularly important field for future work.

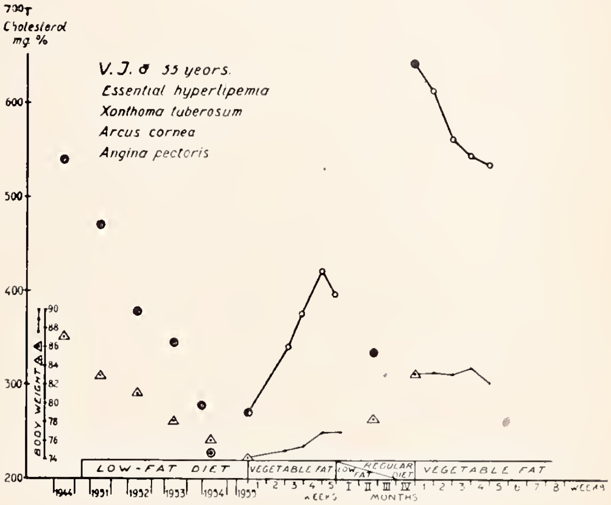

Fig. 15. A case of essential hyperlipemia successfully treated for several years with low fat diet. When the diet was changed to a high vegetable fat diet the cholesterol rose again. After a period of regular mixed food containing much animal fat a diet high in vegetable fat resulted in a fall of the serum cholesterol.

Acknowledgments

It is a pleasure to acknowledge the contribution of Dr. Bengt Swahn to these investigations. The series could not have been collected without the kind co-operation of Professor J. Waldenström, Malmö, and Dr. G. Myhrman, Örebro. We are very grateful to Docent Bengt Borgström for valuable suggestions and advice concerning the vegetable fat diet and inspiring discussions. Acknowledgment is due to Dr. G. Wode, Stockholm, for supplies of corn oil, and to Professor P. Swartling, Alnarp, and his staff for preparing the artificial milk and cheese. We are indebted to Miss K. Sonesson and Miss Hanna Nilsson for the preparation of the vegetable fat diet and to Miss I. Kockum for technical assistance.

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DISCUSSION—ARTERIOSCLEROSIS OF THE EXTREMITIES

(Continued from Page 835)

arterioles will show a decreased pulse volume on warming of the entire body to create vasodilatation and in a cold environment upon the use of vasodilating agents. Furthermore, some of these individuals who have had and are helped by sympathectomy in a cold environment experience this same pulse volume diminution on whole body vasodilatation by heat or priscoline.

It is reasonable to say that we should be discriminating in prescribing vasodilating drugs to arteriosclerotic individuals and in particular we should abandon this use in those patients who have undergone sympathectomy. For the same reasons we should probably abandon the use of an exceptionally warm environment for these same patients after a sudden occlusion.

Another interesting finding was made in experiments conducted on rabbits by my colleague, Dr. Thomas Murphy. Rabbits on an atherogenic diet were found to develop marked atherosclerosis of the entire aorta down to and including the renal arteries. A similar group of animals subjected to bilateral lumbar sympathectomy developed a more extensive atherosclerosis involving the distal iliac and femoral vessels. Little or no atherosclerosis was found in the group of animals on a normal diet with or without sympathectomy.

The examination of the long range effect of sympathectomy is obviously a difficult one to accomplish. It is remotely possible that sympathectomy may enhance atherosclerosis of the larger vessels in atherosclerotic patients as it was found to do in the rabbit while it opens the arterioles and other collaterals. This does not contraindicate sympathectomy in individuals where a vasomotor component is repeatedly depriving the extremity of its nourishment. But it does make one reconsider the advisability of sympathectomy in the extremity with a questionably detrimental vasomotor component.

Electrocardiogram in Coronary Heart Disease

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THERE is no type of electrocardiographic abnormality which has not been observed in coronary heart disease, and the accumulated literature is enormous. But it is not my present purpose to review the present state of the various aspects, problems, and approaches. I propose here to discuss the broad problem of the differentiation of normal from abnormal, a question which has received comparatively little attention in general and, in regard to electrocardiography, in particular.

TABLE I.

Coronary artery lumen (per cent of artery area) in 21 cardiovascular normal young (Y) and 20 older Minnesota (O) men, and selected ECG items in 47 young and 52 older men in Minnesota and in Italy. M = means; SD = standard deviations; .. = mean difference between age groups and their statistical significance = t.

	Coron.* Lumen %	Minnesota			Italy**		
		ST mm	Axis° QRS	Axis° T	ST mm	Axis° QRS	Axis° T
Y M	44.52	7.46	68.87	44.85	5.35	42.83	35.04
SD	7.88	2.01	17.44	19.92	2.70	30.76	20.86
O M	31.50	5.16	45.19	26.65	4.94	35.56	34.85
SD	8.10	1.67	29.97	23.66	2.15	26.10	21.77
..	—13.02	—2.29	—23.68	—18.19	—0.41	—7.27	—0.19
t	5.2**	6.2**	4.7**	4.2**	0.8	1.3	0.04
Per cent change	—29.3	—30.9	—	—	—7.6	—	—

*P Lober **A. Keys

A progressive development of coronary sclerosis is one of the most impressive normal biological age trends in man. Allowance for the effect of this on the electrocardiogram appears to be essential for the interpretation of electrocardiogram in coronary heart disease.

Statistically highly significant differences in most electrographic (ECG) items were found when a group of 157 normal young men was compared with a group of 233 middle-aged men in Minnesota.[1] Fortunately, one of the most detailed autopsy studies on arteriosclerotic changes in 536 hearts was made, independently, on members of the Minnesota population (Lober),[2 a,b] so that at least in geographical respect the groups are comparable.

Table I shows, in column 1, the difference in relative coronary artery lumen (which is one of the best criteria of arteriosclerotic degeneration) between twenty-one younger and twenty older men who died from trauma or acute illness. This is compared with the age difference of a few selected ECG items between forty-seven young and fifty-two older men in Minnesota (columns 2-4) and in Italy* (columns 5-7). Both ECG groups were randomly selected but matched as to number, body weight distribution and mean age.

Table I shows a definite parallelism between relative coronary artery lumen and ECG changes in the Minnesota men. The Italians, however, do not show these ECG changes with age. This seems to be highly suggestive of a causal relationship because the incidence of advanced coronary sclerosis and of coronary heart disease is much lower in Italy. It seems to be a reasonable hypothesis, at least meriting more extensive study, that such ECG age trends as we see in Minnesota are, in fact expressions of progressive coronary atherosclerosis. This would mean, among

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 9, 1955.

*Recently collected by Dr. A. Keys.

other things, that the ECG might provide a useful estimate of the incidence of coronary sclerosis in the general population, but, of course, not necessarily for the individual.

TABLE II.

Means (M) and Standard Deviations (S. D.) of selected ECG items in 15 patients (P) with angina pectoris and normal resting ECG, compared to 233 normal older men (O)

Group		R_2 mm	T_1 mm	T_2 mm	ST mm	QRS axis°	T axis°	T-V_4 mm
P	M	7.05	1.71	1.46	3.93	11.00	16.67	3.61
	S.D.	3.79	0.87	0.77	1.35	29.86	33.13	2.24
O	M	7.75	2.01	2.26	5.20	37.77	32.87	7.08
	S.D.	3.16	0.89	0.95	1.74	34.67	23.96	2.49
	. .	—0.70	—0.30	—0.80	—1.33	—26.77	—16.20	—3.47
	t	0.82	1.27	3.19	2.77	2.92	2.54	1.71
	P	0.42	0.21	0.002	0.006	0.005	0.015	0.09

Many people with advanced coronary sclerosis do not develop coronary heart disease, but all who do so have extensive and advanced arteriosclerotic degeneration. If clinical coronary heart disease can be considered as the extreme result of a process related to age a gradual transition rather than a sharp separation between the so-called normal population and patients should be expected. This is actually the case. In a high percentage of patients with typical angina pectoris, the ECG is within the normal range limits as customarily defined.

Although the ECG of an individual patient with coronary heart disease may be "normal," significant group differences between such patients and the normal population do exist, as we have found. We compared selected items in the resting ECG of 233 older clinically healthy men and in fifteen angina pectoris patients whose electrocardiograms in rest where normal but became abnormal after exercise (Table II).

All changes in the group of patients are in the direction of average age trends we find in clinically healthy men in Minnesota and they are statistically highly significant in four of the seven items studied. In another item (T-V_4), the difference approaches statistical significance. The results show that the ECG is affected already in the initial phase of coronary disease though the changes are not sufficient to move the ECG of many individual patients clearly outside the wide normal range limits. The results correspond to the concept of a gradual transition from normal age trends to coronary heart disease.

We have approached this problem from still another angle, that is the variability of the ECG in repetitions. That the ECG is more variable in patients with coronary heart disease than in normal persons is common clinical experience, but no precise information was avilable.

The intraindividual standard deviation was determined from repetitions after six weeks with fifteen coronary patients whose electrocardiograms were normal in rest and with thirty coronary patients with abnormal electrocardiograms in rest; these represent earlier and a more advanced phase of the disease (Table III). Patients with acute episodes of severe coronary insufficiency of myocardial infarct were not included. For comparison, data on the annual repeat variability of 100 older men was available. The intervals between repetitions are not identical, but if anything, the longer interval of the normal group should be expected to result in larger variability. In all items except ΣT the repeat variability in the patients with abnormal resting ECGs is significantly higher than in the "healthy" men. The repeat variability of the T axis in these patients is enormous. Coronary heart disease, therefore, increases significantly the repeat variability as the ECG becomes abnormal. The repeat variability of the coronary patients with normal ECG is significantly increased only in the T-axis, which seems to precede the development of frank abnormalities in the electrocardiogram.

This finding may have potential diagnostic interest, and in any case it conforms to the concept of a gradual transition from normal age trends to the picture of frank coronary heart disease.

Wide overlapping of normal and abnormal distribution was recently demonstrated for Q_3 and twenty-two related ECG items even in such an advanced stage of coronary heart disease as posterior wall myocardial infarct.[3]

In this situation, various stress situations such as exercise, anoxia, meal intake, and drug effects have been used with advantage for detection of coronary heart disease. All of these tests produce an abnormal ECG response in an appreciable number of patients with angina pectoris whose electrocardiograms are normal in rest but there is wide disagreement as to the percentage of abnormal responses produced by the various stresses. This is due to the lack of independent objective criteria to define latent coronary disease, and because different electrocardiographic criteria have been used for differentiation between normal and abnormal responses.

TABLE III.

Six-week variability (S.D.) ECG items in patients with coronary heart disease and normal (Cor. N) and abnormal (Cor. A.) ECG, compared to annual variability in older normal men (N). Statistical significance shown by F test.

Group	No.	P-R int. 1/100 sec.	T_2 mm	Tv_4 mm	SQRS mm	ST mm	QRS Axis°	T-Axis°
N	100	11	0.54	1.66	2.47	1.12	8.44	10.29
Cor. N	15	13	0.38	2.13	2.38	0.92	10.94	26.29
Cor. A	30	26	0.73	2.69	4.99	1.03	13.13	63.98
$F\frac{\text{Cor. N}}{\text{N}}$	—	N.S	N.S	N.S	N.S	N.S	N.S	6.52**
$F\frac{\text{Cor. A}}{\text{N}}$	—	5.59**	1.82*	2.63**	4.07**	N.S	2.42**	38.62**
$F\frac{\text{Cor. A}}{\text{Cor. N}}$	—	4.00**	3.75**	1.60	4.41**	N.S	1.44	5.92**

A considerable overlap should be expected also in the distribution of "normal" and "abnormal" responses when clinically healthy subjects are compared with patients. Actually, this is precisely what is found in spite of highly optimistic claims for this or that test as a means of detecting coronary heart disease. Master[4] has claimed a 95 per cent diagnostic accuracy for his two-step exercise test but this is certainly misleading. Such a high percentage of "positives" among patients with coronary heart disease can only be achieved at the expense of including a very large number of false positives among the clinically healthy population.

In our own studies normal range limits for conventional and vectorial ECG items were recently obtained for two types of exercise (treadmill walking and step test) in our laboratory.[5]

All ECG stress tests have been developed primarily for detection of latent coronary disease, but "abnormal" responses are also obtained in other cardiac and non-cardiac diseases. However, no large groups of patients with diseases other than coronary heart disease have been studied, and therefore we cannot tell with certainty how much more specific an abnormal exercise test would be for coronary heart disease than for other types of pathology.

Further work in these directions, using the conventional as well as the newer vectocardiographic methods, appears to be promising.

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The Minnesota Heart Association raised a record $379,000 in the 1955 Heart Fund campaign. Hennepin County produced $161,063 of the total. Generosity and genuine concern of Minnesotans regarding the heart disease problem is reflected by their 1955 performance. Minnesota ranks eighteenth in population, yet only seven other states raised more than Minnesota in the Heart Fund drive in 1955. Goal in the state for the February, 1956, drive is $380,000. State chairman will be E. B. Eliason, Jr., of Minneapolis. Approximately 30,000 volunteers will undertake the drive in Minnesota come February 1. Despite past successes, Mr. Eliason says, MHA was able to supply only half the research funds requested by state projects in 1955.

Quantitative Electrocardiography

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RATHER than attempt a detailed discussion of Dr. Simonson's excellent work, I thought I would discuss how advances may be made in the quantitative interpretation of the electrocardiogram, using a few examples from the literature and some from our own recent attempts.

The process which is essential and final for the physicist, physiologist, or physician dealing with electrocardiographic wave forms, no matter how obtained or how recorded, is the interpretation of the record. The process of interpretation of electrocardiograms by the physician has for many years been essentially unchanged.

Interpretation of the electrocardiogram has consisted of making a few quantitative measurements such as the PR, QRS, and QT intervals, and then describing in qualitative or correlative language the size and shape of the deflections. Qualitative descriptive words such as "low," "high," "sagging," "peaked," and "depressed" are frequently used; or, correlative words such as "strain," "coronary type," and "digitalis type" are also frequently used. The mental process of the interpreter of the electrocardiogram is remarkable. It involves a visual estimate of the size and shape of the electrocardiographic waves in many leads, an estimate of the size, shape, and direction of the QRS relative to those of the T waves, and finally a comparative estimate with the limits of normal variations of these estimates and with the various patterns of abnormal variations. This is a fairly complicated process. Most of this interpretive process is not quantitative, nor is it expressed in values which can easily be compared, reproduced, or communicated to other interpreters.

Dr. Simonson has properly emphasized that one of the most difficult parts of the process of interpretation is to define the limits of normal variation and to separate clearly the normal from the abnormal. In addition, there is the problem of expressing degrees of abnormality. The final step is to correlate the changes in the electrocardiogram with other pieces of evidence obtained from the historical facts and physical examination, and from histological, pathological and physiological chemical observations. The interpretation of the electrocardiogram in the last step can only be an accurate predictive diagnosis if there is previous correlative experience of an accurate clinical, pathological, or physiological nature. In other words, for clinical purposes, the electrocardiogram is only as accurately diagnostic as the information with which it has been previously correlated.

Aside from research which correlates the electrocardiographic picture with disease, drugs, stress, or physiological findings, there are two other approaches to electrocardiographic research which have had some recent bearing on the process of electrocardiographic interpretation. I have divided electrocardiography into three categories of study, the first of which has been mentioned, namely, *correlative* or *diagnostic electrocardiography*. The second category might be termed *analytic electrocardiography*. In this category are the endeavors of research workers to map the electrical field in and on the surface of the body, and to study the factors influencing the conduction of the generator signals through the medium of the body.

The third type of research might be *synthetic electrocardiography*. In this category are the attempts to put into a more unified picture the pieces of the electrical forces of the heart obtained from isolated leads or reference points. Most of the attempts to do this have been based on the general assumption (or hope) that the heart acts essentially as a point source or generator in the center of an electrically homogeneous conducting medium, namely, the body. This is the basis for the use of the vector method for representing the electrical information from the

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 9, 1955.

heart. The vector theory is a unifying concept of the heart's electrical activity. All the known clinically useful information about the heart's electrical activity should be contained in the vector figure. It should be possible to find three bipolar reference leads representing all the electrical information in the three planes of the body. It should also be possible then to derive any local or special surface electrocardiographic view one desires from a primary electrocardiographic information obtained at a distance.

A good deal of experience with the technique based on the vector theory shows that practically all the electrocardiographic information which we obtain by applying leads at multiple points on the surface of the body, particularly on the chest, can be derived from recordings from distant reference axes. Between the observations of Milnor and ourselves, we have examined over a thousand subjects with all sorts of abnormalities. We have yet to find information which is of any use for clinical correlation or interpretation in the electrocardiograms obtained from multiple surface leads which cannot be derived from three simple bipolar electrocardiographic leads taken at a distance representing the electrical information in the three axes of the body.

The question arises: What does one consider clinically significant information? At the moment, we can only state that physicians who have had ten to fifteen years' experience in heart stations reading twenty to forty electricardiograms per day can find no more diagnostic information in the conventional twelve lead electrocardiogram than can be obtained by separate independent interpretation of the distant three vector reference lead electrocardiograms recorded simultaneously.

An example of this technique is shown in Figure 1. Above are the electrocardiograms as recorded by conventional techniques using the V leads across the chest. Below are the electrocardiograms which were derived or calculated by an electronic instrument, from the three electrocardiograms recorded from distant leads on the body. You can see that the information which is diagnostic, and which has been perhaps in the past thought to be rather special local or semi-direct information, is contained in distant leads which are nowhere near the usual locations of the chest leads. These electrocardiograms are obtained by a synthetic calculating process from three leads formed from a total of four electrodes placed on the back and near the right shoulder, with only one electrode on the front of the chest.

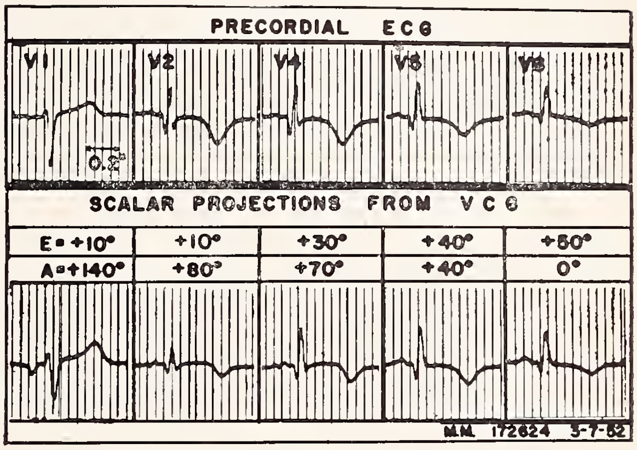


Fig. 1. Illustration from the work of Milnor et al. Above are the actual precordial electrocardiograms from a patient with an anterior myocardial infarction. Below are the electrocradiograms derived with a Schmitt instrument (variously called a "resolver," a "panoramic unit" or a "vector aspect changer"). The primary information from which the leads are derived consist of three bipolar leads placed according to Milnor's system. E is the elevation and A the azimuth in degrees of the reference line of the projections (see Figs. 2 and 3). This demonstrates the practical validity of the vector concept of electrocardiography, and encourages us to proceed upon the assumption that all the useful clinical information is contained in three bipolar leads properly placed at a distance from the heart. Conversely, it means that there is practically no special or "semi-direct" information in multiple leads on the chest anatomically closer to the heart.

The questions arise: What is the use of this different set of leads? What sort of advance is this? The value of this demonstration is that it allows us to think of the electrocardiographic picture from the total integrated viewpoint rather than from the viewpoint of each lead giving us some special isolated underlying information. It reduces the electrocardiogram from twelve leads to three. Secondly, this method may lead us to more quantitative expressions of interpretative correlative data. As an example of what is meant by this, let us consider the problem of the significance of the presence of a Q wave is standard Lead III of an electrocardiogram (Fig. 2). The vector force which creates a Q_3 points upward in the frontal plane away from the foot. However, when this initial "Q_3" vector is viewed from the side of the body rather than from the front of the body, we find that in the normal subject it points forward and upward. In patients who

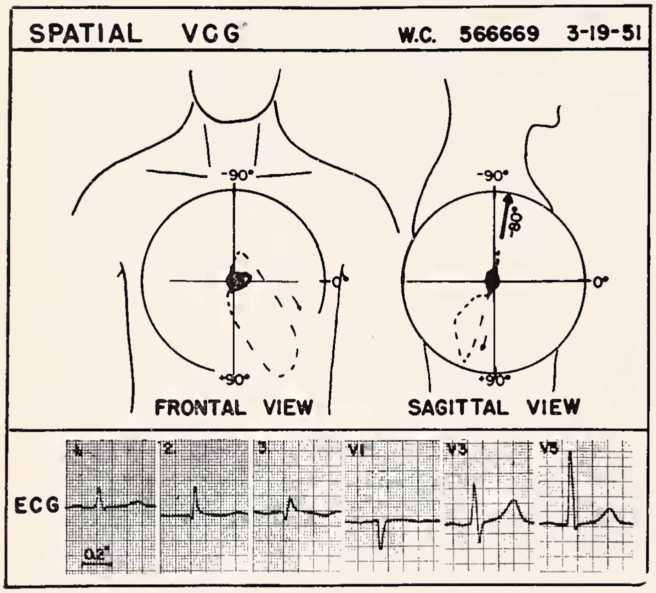


Fig. 2. This shows the vector cardiographic tracing in a patient with posterior myocardial infarction (from Milnor et al), and the scalar electrocardiographic leads. The initial 0.04 vector which results in a Q_3 deflections. points toward the head and to the left.

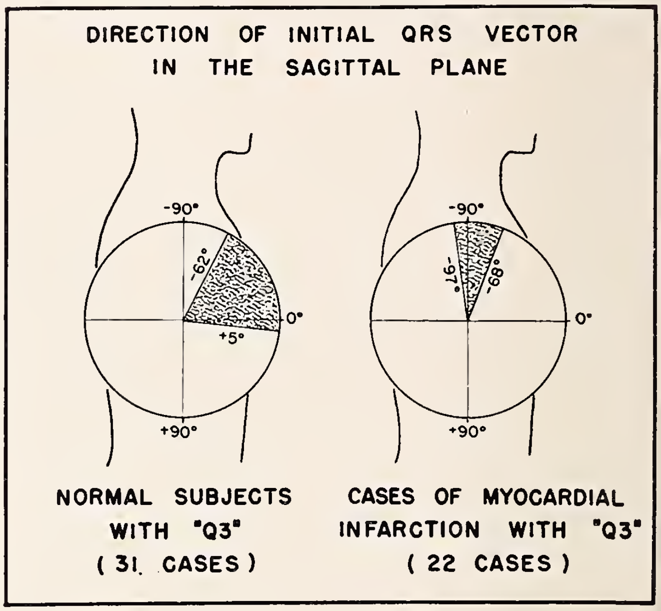
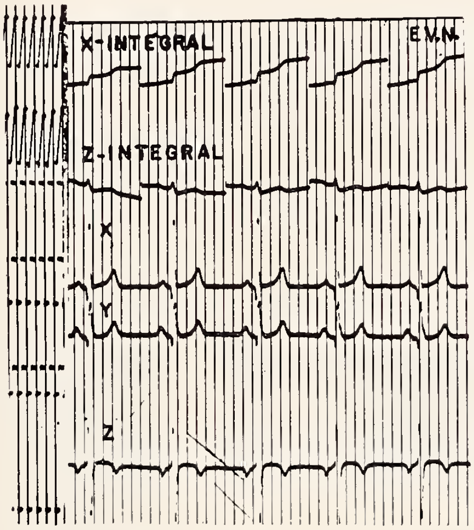


Fig. 3. An analysis of direction of the initial 0.04 vector in normal subjects and cases with posterior myocardial infarction with Q_3 deflections. The best quantitative differentiation of the normal from the abnormal is obtained by the vector addition of forces in all directions (from Milnor et al), obtained from a simple bipolar three lead system representing the electrical forces in the three planes of the body.

have been judged to have had a posterior myocardial infarction from other definite clinical criteria, we find that the "Q_3" vector points directly upward and frequently backward (Fig. 3). As more data is accumulated, some normal subjects and some patients with myocardial infarction may have initial 0.04 vectors in the saggital view which point in the same direction. This again leads to difficulty in interpretation. However, the method is helpful because the limits of normal can be quantitatively defined. One would like to have a completely accurate diagnostic tool, but I am afraid the instrument or the method of interpretation will not alone resolve all problems. All we can hope to do is sharpen the differences between the normal and abnormal. If we can express these things in quantitative terms, we have a chance for more accurate interpretation. The fact that the Q_3 wave or the initial 0.04 vector tends to point upward and backward in posterior myocardial infarction is another expression of the fact that in posterior myocardial infarctions the R waves of the initial 0.04 vector in the anterior chest leads; that is, V_1, V_2, and V_3 tend to be low or absent. The difficulty in interpreting the height of the R waves in V_1, V_2, and V_3, however, is one of quantitation. It is very difficult to visually and mentally estimate the height. However, if the information is correlated with the movement of the vector in another direction, more quantitative reproducible information can be obtained and we can assign not only a size but an accurate direction in space to the potential.

There is another concept of vector additions which may be useful. A long time ago, Wilson injected some quantitative ideas into the interpretation of the electrocardiogram when he introduced the concept of ventricular gradient. Ventricular gradient is an attempt at quantitative expression of the mental process mentioned previously, namely, the process of making an estimate of the size and direction of the QRS deflection relative to those of the T wave of each lead. Dr. Johnston was one of the first, in 1950, to report an attempt to facilitate the process of determining the areas under QRS and T waves by using electronic circuitry. Recently, in our laboratories, Thomas Arnold and Dr. James McGovern have made advances in instrumentation which will facilitate quantitative analysis of the areas of the QRS and T waves.

We have combined the unifying concept of the vector method with the idea that comparison of the areas of the QRS and T waves is fundamentally important in interpretation. If all the known clinically useful information about the heart's electrical activity is contained in three bipolar reference leads, it should be possible to obtain a three-dimensional unified concept of the relative areas. The hope is that this synthesis of information would dissolve the differences between normal people, and sharpen the differences between the abnormal patterns and the normal.



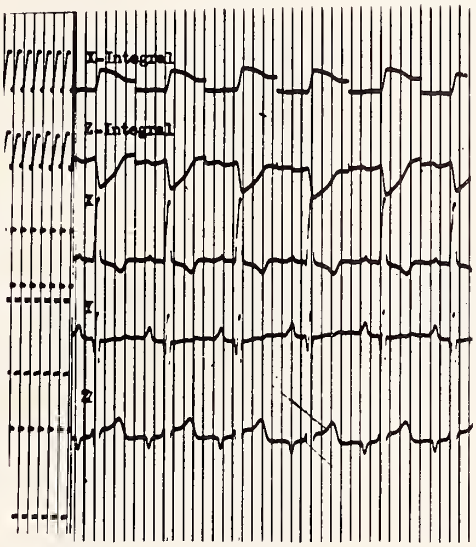


Fig. 4. *(left)* The three normal scalar electrocardiograms and two scalar simultaneous integrals. The X, Y, Z leads are bipolar. The X lead is formed by electrodes on the back in the mid-clavicular lines directly opposite the second anterior interspaces on both sides. The Y lead is between the right back electrode above mentioned and an electrode placed directly below at a distance equal to that between the two X lead electrodes. The Z lead is formed by placing an electrode on the right anterior chest directly opposite the right upper back electrode. The positive sense of both the electrocardiogram and integral deflections is toward the patient's left, front, and foot.

The calibration deflections represent 1.0 millivolt input for the electrocardiograms and 0.05 second millivolt input for the integrals. Each cycle is integrated separately beginning and ending the integration in the T-P interval. (The variation of the calibration signal the Z integral channel was due to temporary instability of the signal generator.) Time lines are 0.10 seconds.

Fig. 5. *(right)* The X Y Z scalar electrocardiograms and the X and Z simultaneous scalar integrals in a patient with left ventricular hypertrophy due to aortic stenosis. Technique and calibrations same as for Figure 4.

Many of the differences in electrocardiographic patterns are "positional," and at the present stage of our knowledge it would be difficult to say which electrical view of the heart is the "correct" one. The correct view from the practical clinical standpoint is the one which gives the most reliable differentiation of disease. Utilizing the vector concepts and area measurements, and encouraged by Schmitt on the validity of vector leads, we have proceeded to develop electronic methods for the vector addition of area measurements. This procedure might be called the three dimensional vector integral electrocardiogram. Integrating circuits were devised which would deliver a voltage proportional to the area accumulated under each QRS T complex. An example of this continuous process is shown in Figure 4 for a normal person. The scalar electrocardiograms are called X, Y, and Z representing respectively the electrical forces in the side-to-side, up-down, and front-back directions of the body. The continuous simultaneous integrals of each cycle are recorded for the X and Z co-ordinates. These two co-ordinates form the transverse plane. The same process is shown in Figure 5 for a patient with left ventricular hypertrophy. When the integration for each cardiac cycle is completed, the integration starts for the successive cycles at the new baseline.

The continuous accumulation of area in two directions can now be added vectorwise continuously, using an oscilloscope. Illustrations of the vector additions of the scalar electrocardiograms and the scalar integrals are presented for

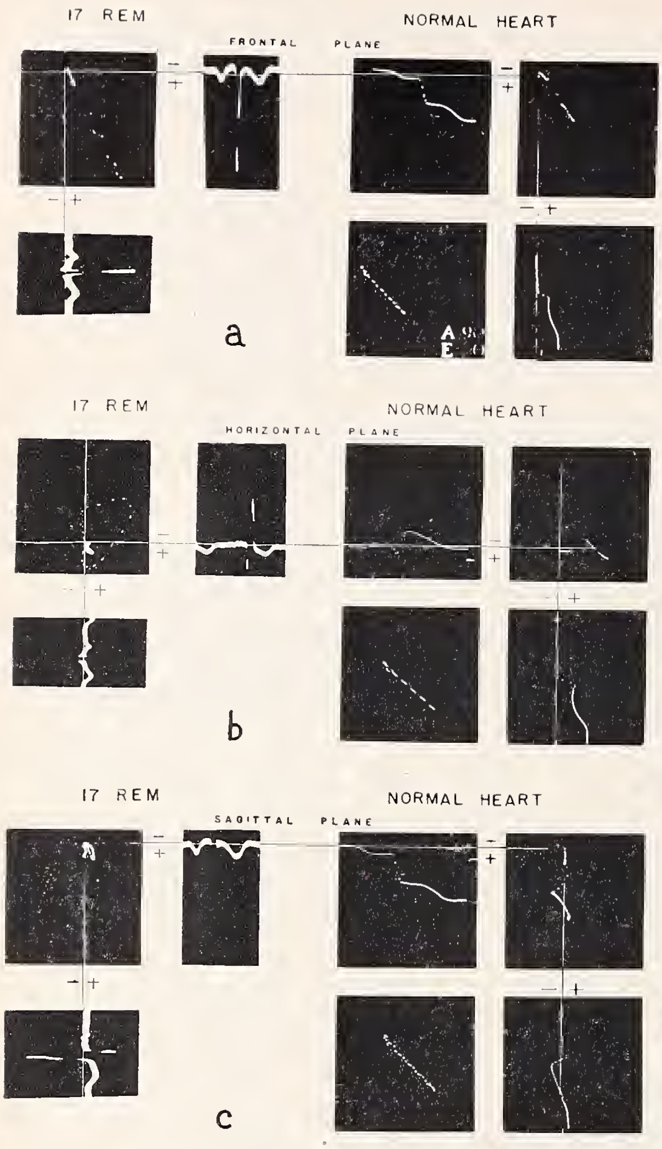

17 REM
NORMAL HEART
FRONTAL PLANE
a
17 REM
NORMAL HEART
HORIZONTAL PLANE
b
17 REM
NORMAL HEART
SAGITTAL PLANE
c

Fig. 6. (a) A composite chart showing, in a normal subject, the frontal plane vectorcardiogram (VCG) (top left) and the frontal plane vector integral cardiogram (VICG) (top right) with the scalar components of each arranged in their spatial relations. Calibration signal included which produces the line at 45° to both axes.

Time markings are made in the VCG and VICG, and on the scalar integrals by interrupting the beam. Thus, during the rapid accumulation of area during the QRS, the dots are farther apart, while during the slower T wave area accumulation, the dots tend to fuse.

(b) The normal horizontal plane vectorcardiogram and vector integral cardiogram with the scalar components of each.

(c) The normal sagittal plane vectorcardiogram and vector integral cardiogram with their scalar components.

a normal subject in Figures 6a, 6b, and 6c, and for a subject with left ventricular hypertrophy in Figures 7a, 7b, and 7c, in the three planes of the body.

Summary

I have limited my comments to possible ways to make advances in the process of quantitative electrocardiographic interpretation. The process of electrocardiographic interpretation has been too seldom a quantitative one. The progress in validation of the vector theory of the heart's electrical activity has helped somewhat to free us from isolated viewpoints of electrocardiographic leads. Some of the vector additions of electrical information may help in separating the normal from the abnormal, and describing the degrees of abnormality. The concept of ventricular gradient which is another type of vec-

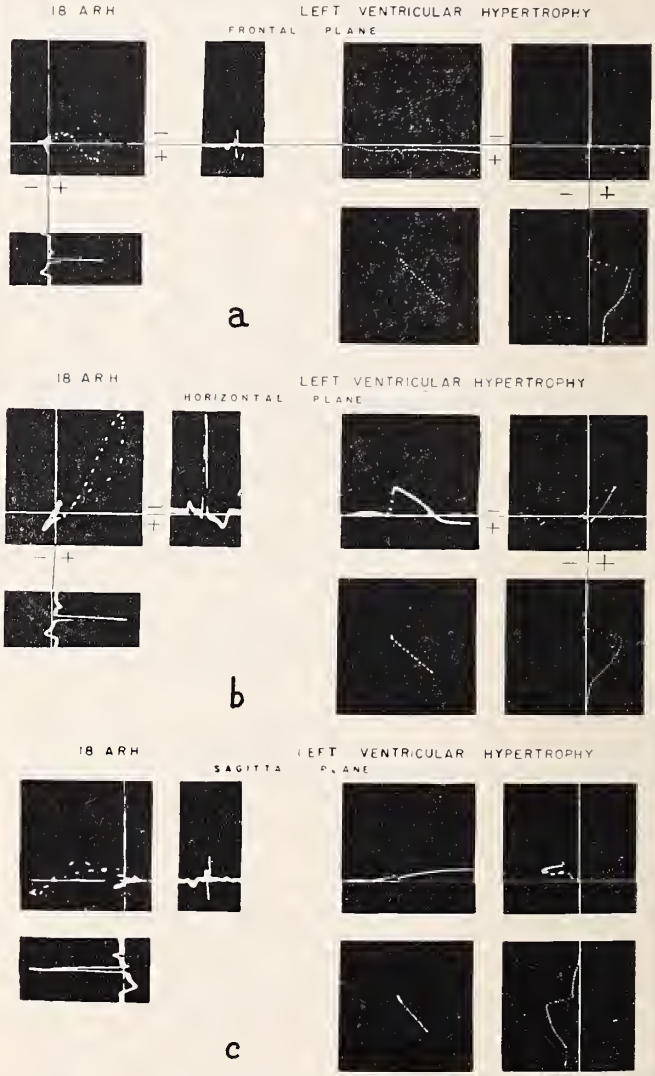

18 ARH
LEFT VENTRICULAR HYPERTROPHY
FRONTAL PLANE
a
18 ARH
LEFT VENTRICULAR HYPERTROPHY
HORIZONTAL PLANE
b
18 ARH
LEFT VENTRICULAR HYPERTROPHY
SAGITTAL PLANE
c

Fig. 7. (a) A composite chart showing, in a patient with left ventricular hypertrophy due to aortic stenosis, the frontal plane vectorcardiogram (VCG) and the frontal plane vector integral cardiogram (VICG) with their scalar components.

(b) An abnormal horizontal plane VCG and VICG with scalar components in a patient with aortic stenosis.

(c) The VCG and VICG with their scalar components in a patient with left ventricular hypertrophy.

tor addition has always been important in interpretation, and I believe new methods now make it possible to further test and extend the value of this concept of quantitation in the future. By utilizing a simple three lead system and continuous integration with vector addition, a three dimensional vector integral electrocardiogram (VICG) may be obtained. The value of this method of representation remains to be proved, but it is now possible to proceed with the facility and accuracy of automatic computation. Finally, even though we can quantitatively represent the electrocardiograms, none of the quantities will be useful unless they are correlated carefully with equally accurate quantitative information obtained from clinical facts, from pathological and histological examination, and from physiological and chemical observations.

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ANEURYSM OF THE ABDOMINAL AORTA

(Continued from Page 838)

are present. It also will show the relationship of the area of obstruction to the renal and mesenteric arteries and will give some information of value concerning collateral circulation. As is the case in aneurysm of the aorta the aortogram does not always reveal correctly all details of the true situation, and if from the appearance of the aortogram there is uncertainty as to operability of the lesion, an abdominal exploration may be justified.

The two points of the clinical evaluation of occlusion of the abdominal aorta which are of greatest importance when treatment is being considered are: first, to determine by a careful history how much the symptom of intermittent claudication is really impairing the usefulness and happiness of the patient, and secondly, to determine, if possible, by examination whether or not any of the large arteries distal to the bifurcation of the aorta are occluded.

As more experience is gained with this condition and knowledge concerning arteriosclerosis in general increases, we physicians will be better able to evaluate the degree of arterial vascular disease in the lower part of the body and particularly in the extremities of these patients, and to evaluate the probable future complications and the hazards to life and limb if the condition is untreated. Then we can apply this clinical knowledge with more certainty than is possible at present in advising our patients as to the best method of treatment.

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Ballistocardiogram in the Diagnosis of Coronary Atherosclerosis

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I THINK we will all agree that perhaps the greatest challenge to medical research now is that presented by arteriosclerosis and, in particular, its most lethal manifestation, coronary artery disease. And I think we may also agree that our methods for detecting coronary disease are woefully inadequate. Completely normal findings with our most modern diagnostic techniques on one day guarantee our patient no immunity whatsoever from an attack the following day. We are, indeed, virtually blind to the presence of the disease until it suddenly strikes. The detection of advancing coronary disease in asymptomatic individuals is difficult, to say the least, but the unfortunate truth is that our methods are so insensitive we can often provide no objective evidence of its presence in patients whose typical symptoms denote advanced disease. So, we clearly need different and better methods for detecting coronary atherosclerosis and following its course. The quest for the cause of this disease and for therapeutic methods aimed at arresting or preventing its progress goes on unremittingly. If and when that ultimate therapeutic goal is reached, its effective clinical application will depend, in no small measure, on the detection of coronary atherosclerosis at an early stage in its progress, when therapy is most likely to be effective. The challenge to improve our diagnostic methods has been made and it must be met.

During the last decade the ballistocardiograph has been applied with increasing frequency to the problem of coronary artery disease. This new technique is suffering the same growing pains experienced by electrocardiography and other new methods during the early phases of their application. There is always the tendency for preliminary results to produce divergent views and conflicting opinions and so becloud the real issues that it is difficult for anyone outside the field to form a definite opinion; and so it is with respect to the value of ballistocardiography in the coronary disease field. My purpose will be to examine the evidence and to try to provide you with data on which an opinion may be based.

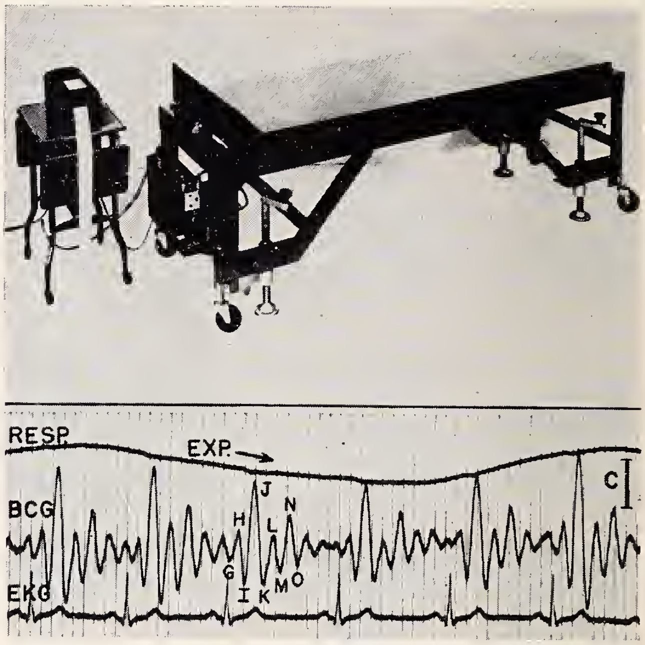

Fig. 1. (A) High-frequency ballistocardiograph, commercial model. This instrument has a natural frequency of 12 to 14 cycles per second when loaded with 150 pounds dead weight. The electronic transducer and amplifier are mounted on the foot end of the frame. (Courtesy Technitrol Engineering Co.). (B) Normal ballistocardiogram recorded with a high-frequency bed. The upper tracing (RESP) is the pneumogram (expiration downward), the middle one (BCG) is the ballistocardiogram, and the lower one is the electrocardiogram (Lead II). Heavy time lines are 0.1 second apart. The bracketed vertical line (C) in the upper right corner represents the calibration deflection (280 GM. = 1 cm.). Reproduced by permission of *American Heart Journal).*

For the benefit of those who may be unfamiliar with the method I would like to comment on it briefly.

The ballistocardiograph is an instrument designed to record the minute motions of the body

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caused by the rapid ejection of blood from the heart with each beat. The instrument shown in Figure 1 is a commercial version of the Starr high-frequency bed, which is but one of several general types in use. The ballistocardiogram is a record of the motion of the platform on which the subject reclines. The ballistocardiogram shown here is normal and is typical of those from all young healthy individuals. The pattern is consistent and repetitive. The size and form of the pattern is related to the force with which the heart ejects blood into the great vessels. However, in the heart weakened by disease this normally great propulsive force is altered and the resulting ballistocardiogram is strikingly different from the one shown here. The waves are generally smaller and the consistent pattern is usually lost.

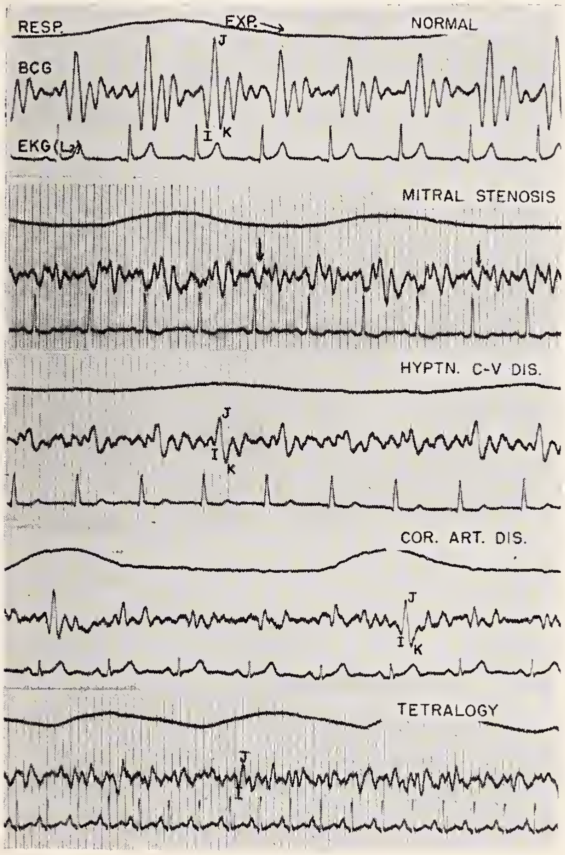

Fig. 2. The ballistocardiogram in various types of cardiovascular disease. The record at the top is a normal one for comparison.

Figure 2 shows a normal record at the top and four abnormal records from patients with various types of cardiovascular disease.

Early in his work Starr[1] found that many of his patients with coronary disease had abnormal ballistocardiograms; even more interesting was his observation that in some of them an abnormal ballistocardiogram provided the only objective evidence of heart disease. In the years since that time his observations have been repeatedly confirmed. Studies by different investigators using the Starr or the direct-body ballistocardiographic instruments on large groups of patients with either classical angina pectoris or a history of myocardial infarction have, in general, yielded similar results.[2,3,4,5] Since those obtained by our own group are fairly representative, we will examine them;[6] 328 patients were compared with 369 clinically normal subjects from twenty to eighty-four years.

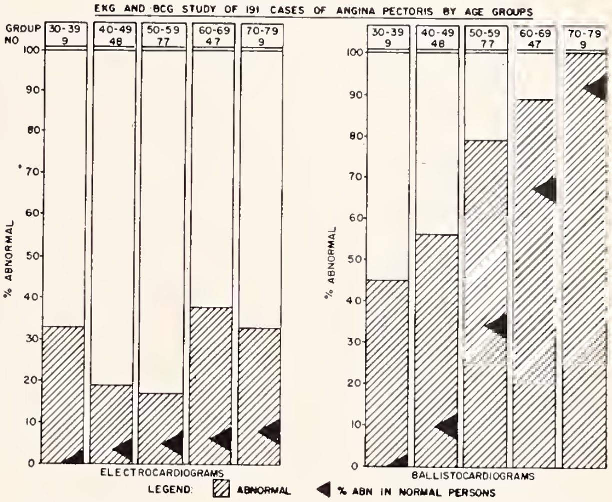

Fig. 3. Electrocardiographic and ballistocardiographic study of 191 cases of angina pectoris. There is a low incidence of electrocardiographic abnormality (24 per cent, for the whole group) and a high incidence of ballistocardiographic abnormality (75 per cent, for the whole group). The frequency of abnormal electrocardiograms and ballistocardiograms is significantly higher in the patients with angina pectoris than in the normal control subjects (arrowheads) in each decade except the eighth. There is an increasing frequency of abnormal ballistocardiograms in patients as well as in normal subjects. Only 45 per cent of the patients in the fourth decade had abnormal ballistocardiograms; this incidence rose to 100 per cent in the eighth decade. (Reproduced by permission of *American Heart Journal)*

On the right in Figure 3 are the results of the ballistocardiographic studies on 191 patients with angina pectoris. The height of the cross-hatched block represents the percentage of patients with abnormal records in a given decade. The ballistocardiogram was abnormal in 75 per cent of the whole group but there was a definite age trend from about 45 per cent for the thirty-year-olds to 100 per cent for the seventy-years-olds. The black arrows indicate the results from our control group of normal persons; there were no abnormal records below the age of forty, but above this there was a steadily rising incidence of abnormality reaching 92 per cent of normal subjects in their seventies. The electrocardiographic results are on the left; only 24 per cent of the patients with angina pectoris had abnormal electrocardiograms.

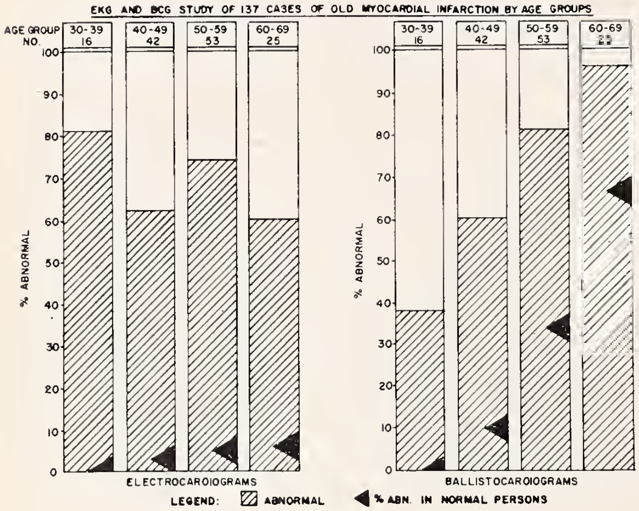

Fig. 4. Electrocardiographic and ballistocardiographic study of 137 cases of old myocardial infarction. Number of patients in each decade is indicated below the age group designation. Electrocardiograms abnormal in 69 per cent and ballistocardiograms abnormal in 72 per cent of whole group; both incidences are significantly higher than in clinically normal persons (arrowheads). There is an increasing frequency of ballistocardiographic form abnormality with age in patients with myocardial infarction as well as in normal subjects. Only 38 per cent of the patients below the age of forty had abnormal ballistocardiograms but the incidence increased to 96 per cent in the seventh decade. (Reproduced by permission of *American Heart Journal.)*

The results in 137 patients with previous myocardial infarction (Fig. 4) are similar to those from the patients with angina pectoris. Of those below the age of fifty only about one-half had abnormal ballistocardiograms. Abnormal electrocardiograms were more frequent in this group with healed infarcts, as one would expect.

I would like to set aside for a moment the question of the causes of the abnormal records in the clinically normal subjects and assume them to be, in fact, normal and free of heart disease. From the combined results of these and other studies we may conclude: (1) that about three-fourths of all patients with coronary artery disease have abnormal ballistocardiograms, (2) that above the age of fifty abnormal records are even more frequent in these patients, but they are also quite frequent in clinically normal persons, (3) that below the age of fifty the incidence of abnormal records is low in normals but it is also relatively low in patients with coronary artery disease. Are there any differences in pattern or waveform which would serve to distinguish between patients and normals? The answer is *no,* if we exclude the coronary artery disease patients who are in congestive failure. It would be well to point out here that there is no ballistic pattern which is typical, characteristic or pathognomonic of coronary disease. Nor do quantitative measurements of the records improve the separation of subjects with and without overt coronary disease.[6]

So we are forced to conclude that by itself, the resting ballistocardiogram fails to provide a satisfactory separation of the two groups of individuals. Many supplementary techniques, such as the exercise and anoxemia tests, have been used to improve the diagnostic value of both the ballistocardiogram and electrocardiogram but most of these have failed to add very much. In contrast to the results reported by Master,[7] our results with his "two-step" test have been discouraging.[8] The only "stress test" which, in our hands, has proved of definite value in separating subjects with and without coronary disease is the so-called "cigarette test." Henderson[9] first observed that the ballistocardiograms of patients with coronary disease frequently deteriorated after smoking a cigarette whereas this response was unusual in normal persons. Davis[8] of our group confirmed his results, as did Russek[10] more recently.

Figure 5 shows the effect of the "cigarette test" on three different subjects. In each case the record labeled A is the control, and B is the record taken immediately after smoking. The upper and middle pairs of records are from two patients with angina pectoris and both show the deterioration which marks a positive response. In the lower pair of records there is no change after smoking. This is a negative test on a normal physician.

Figure 6 shows the results of tests on the 200 subjects in Davis' original study. The cigarette test was considered positive if there were definite deterioration of the post-smoking ballistocardiogram. The height of the black bars indicates the percentage of patients with positive tests in each age group. The results from the normal subjects are on the left, those from the coronary disease patients are on the right. The over-all average of positive tests was 7 per cent in the normals and 59 per cent in the patients, a ratio of about 8:1.

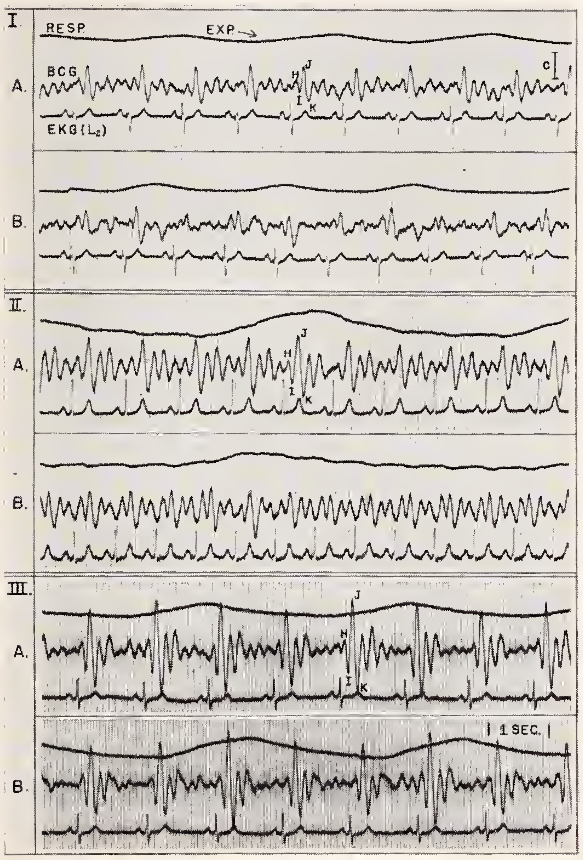

Fig. 5. The effects of cigarette smoking on the ballistocardiogram. I. A. Control record from a fifty-one-year-old man with angina pectoris; B. Ballistocardiogram immediately after smoking. Amplitude decreases, the H wave shows relative augmentation, while I and J are shortened and rounded. Blood pressure rose from 114/80 to 122/88 mm. Hg. This represents a positive test. II. A. Control ballistocardiogram from a fifty-year-old man with angina pectoris and hypertension. B. Ballistocardiogram immediately after smoking a cigarette. The G and H waves are considerably accentuated, J shortened, producing many "early-M-like" complexes. This is a positive cigarette test. Blood pressure rose from 176/106 to 204/120 mm. Hg. III. A. Control ballistocardiogram from a healthy fifty-five-year-old man, a physician. B. Record immediately after smoking. No significant change is apparent. This is a clearly negative cigarette test. (Reproduced by permission of *American Heart Journal.)*

Electrocardiographic changes after smoking were so rare that they deserve no mention. A recent summary[11] of our more extensive experience with the cigarette test in 450 subjects yielded results similar to these just mentioned. The use of filters or de-nicotinized cigarettes failed to protect the sensitive individuals.

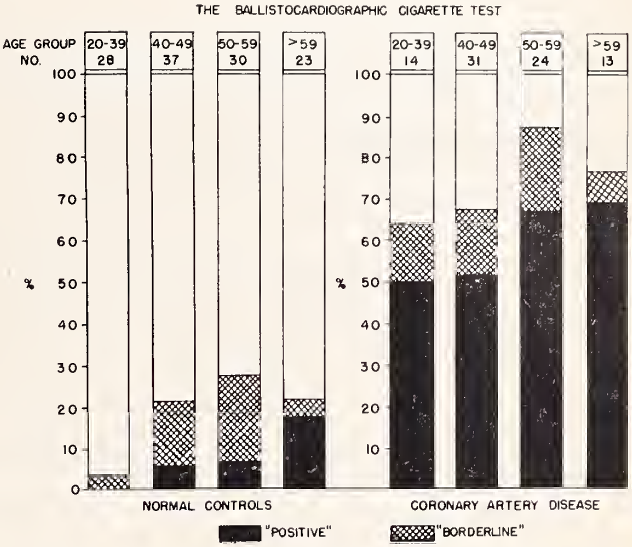

Fig. 6. The incidence of positive and borderline responses of the ballistocardiogram to smoking a cigarette in normal subjects and in patients with coronary artery disease. (The difference between normal and diseased groups is significant in each decade). (Reproduced by permission of *American Heart Journal.)*

The mechanism through which cigarette smoking operates to produce the ballistocardiographic changes described is not known. Nor is it clear why its selectivity is so high for patients with coronary artery disease. There is evidence that nicotine is the active agent but we have as yet no proof that nicotine evokes these changes by producing coronary vasoconstriction, although we cannot deny this as a possibility. While we are not sure that the ballistic deterioration seen after smoking is caused by myocardial ischemia, we are sure that acute ischemia is capable of causing deterioration of the record. Figure 7 illustrates my point. These records are from a sixty-two-year-old man with angina who obliged us by developing a typical attack while lying on the ballistocardiograph bed. Record A was taken before the attack and is an unusually good one considering his age and his disease. Record B, taken during the attack, shows gross deterioration; no normal complexes are present. In addition, the EKG shows rather marked S-T segment depression. In record C, taken after the attack was relieved by nitroglycerin, the ballistocardiogram again resembles the control record; the EKG changes have not completely regressed. So in this case, acute ischemia produced transient abnormalities in both mechanical and electrical function.

In trying to improve the diagnostic value of the ballistocardiograph in coronary artery disease many other leads have been followed but the results do not justify their mention here.

The one major obstacle to a complete and critical evaluation of the ballistocardiograph as a diagnostic tool in coronary disease lies in our uncertainty as to the cause or causes of abnormal records. It has not been established that an abnormal ballistocardiogram is caused *only* by cardiac disease; nor has it been possible to estimate the relative importance of extra-cardiac factors in the genesis of these records. For this reason we cannot be certain about the significance of the abnormal records observed so frequently in older clinically normal subjects. The obvious question, whether these individuals are really free of heart disease, is one which cannot be answered. It has been suggested[12] that most of these abnormal records are caused by sub-clinical coronary artery disease and this seems quite possible. There is, however, no direct or conclusive evidence to support this view; such evidence as does exist is indirect and circumstantial. It is certainly true that the rising incidence of ballistocardiographic abnormality with age resembles or parellels other curves relating clinical or anatomical coronary artery disease to age.[13] However, this does not constitute proof that there is a direct, casual relationship between coronary disease and ballistic abnormality; the only conclusion it does allow is that both variables are related to age and may or may not be related to each other.

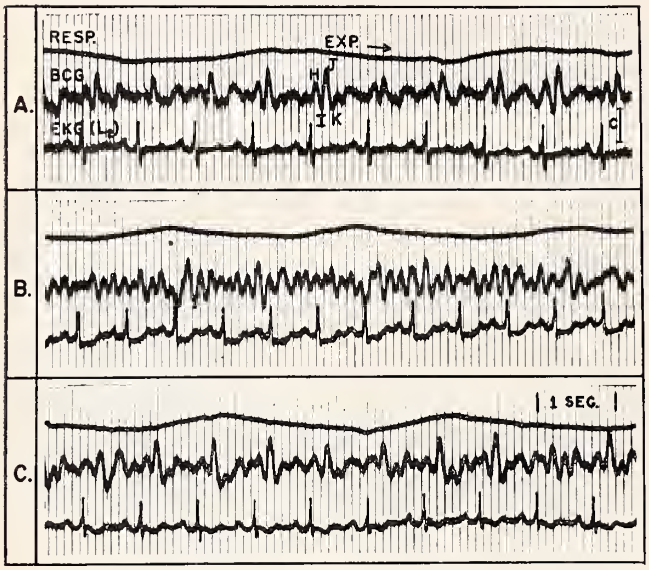

Fig. 7. The effects of myocardial ischemia on the ballistocardiogram. This series of tracings is shown because of the clear demonstration of the changes in ballistic form which occurred during a spontaneous attack of angina pectoris which developed while the ballistocardiogram was being obtained. The sequence of ballistic form changes observed in this subject is similar to that which occurs during a positive cigarette test. The subject was a sixty-two-year-old man with severe coronary insufficiency and angina pectoris. A. Control ballistocardiogram, showing some slurring of I and J, with rounding and shortening of K. B. Ballistocardiogram obtained while subject experienced pain. Gross deterioration is apparent, with short to absent I waves, distorted J and K, and relatively prominent disastolic waves. C. Ballistocardiogram nine minutes after relief of the pain by nitroglycerine. Form is in general similar to that of the control record. (Reproduced by permission of *American Heart Journal)*

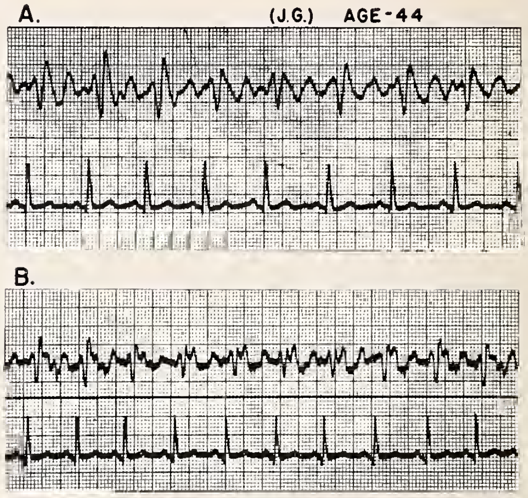

Fig. 8. The two records (high-frequency bed above, aperiodic below) from a forty-four-year-old man with coronary artery disease. The high-frequency bed record shows shortening or absence of the K wave varying with respiration. In the aperiodic bed record there appears an abnormal, headward wave (marked by arrows) in each complex, corresponding in time to the K wave in the other record, which shows similar respiratory variation. (Reproduced by permission of *Circulation).*

Here then is our impasse. On the one hand we are searching for a more sensitive method of detecting coronary artery disease, preferably one which will provide evidence of the disease early in its course, before it produces symptoms. The ballistocardiographic method gives a positive result in a very high proportion of patients with known coronary disease and also gives a positive result in about one-half of those presumably normal individuals whose ages make it likely that coronary disease is present. On the other hand, our uncertainty that the positive result is, in fact, due to coronary disease alone, demands a conservative attitude until more evidence is brought to bear.

Admitting the present inadequacies of ballistocardiography, I would like to briefly discuss research efforts being made to improve the method and to clarify our understanding of the information it supplies. There is little doubt now that imperfections of the method itself are partly to blame for its limited usefulness. Serious errors are inherent in all of the standard techniques and these are caused for the most part by the body's dorsal elastic spring interposed between the body mass and its supporting surface.[14] Recent developments[15,16,17] in instrumentation now make it possible to obtain records free of this artefact, and directly related to the force applied to the body mass by cardiovascular activity; furthermore, the range of frequencies which can be observed has been extended much higher and errors in timing due to phase shift largely eliminated.

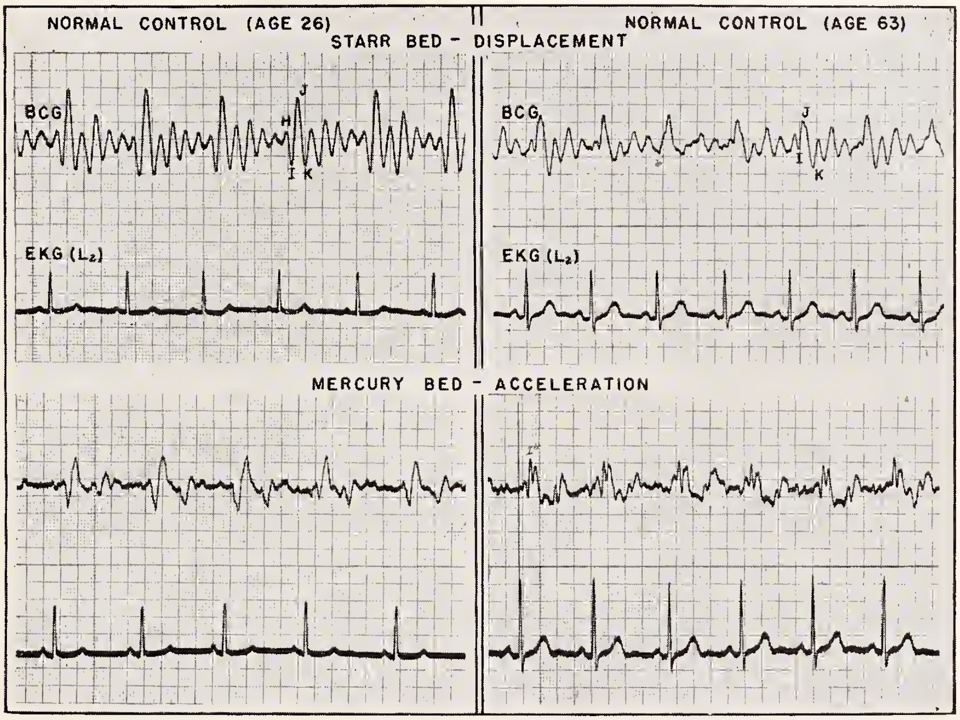

Fig. 9. Starr bed and aperiodic mercury bed records on a twenty-six-year-old normal female and a sixty-three-year-old normal male. Both records are normal in the young person, both are abnormal in the older person. In the latter, the multiple rapid forces seen in the mercury bed record are absent in the Starr record. (Reproduced by permission of *Bulletin of Johns Hopkins Hospital*).

To illustrate the differences between the newer techniques[18] and the standard ones are the records (Fig. 8) from a forty-four-year-old man with coronary disease. Record A, from the Starr high-frequency bed, is simple in waveform and shows only slurring and shortening of the J-K segment. Record B, from our "aperiodic mercury bed,"[16] is quite different; it contains more rapid detail and shows an extra abnormal headward force in the latter part of systole. This abnormality failed to show up in the Starr bed record.

Even at this early stage it seems certain that improvements in method will increase the specificity and precision of our measurements of cardiovascular force. However, there is no assurance that improved ballistocardiographic methods will permit wider separation of patients with coronary disease and older normal persons.

In Figure 9 the records on the left are from a twenty-six-year-old normal person. The Starr record is above, the mercury bed record below. Both are normal but the differences in waveform are apparent. On the right are the records from a sixty-three-year-old clinically normal subject. Both Starr and "mercury" records are quite abnormal although much of the rapid force detail in the latter failed to appear in the Starr record. Whether or not such abnormalities in the records from these older persons indicate coronary atherosclerosis is a question which remains to be answered.

Current and future research in the field of ballistocardiography must be directed toward the identification and quantitation of the numerous factors, cardiac, extra-cardiac and extra-cardiovascular, which contribute to or modify the basic internal force pattern. Further biophysical and physiological investigations both in man and in the experimental animal are in progress and it is hoped that these will lead to an improved understanding of cardiovascular mechanics in normal individuals as well as in patients with cardiovascular disease.

Aside from physiological research, time itself may provide some of the answers. In clinical follow-up studies on normal individuals, if those whose original records were abnormal, clearly develop coronary disease sooner or more frequently than those with normal records, then the abnormal ballistocardiogram would take on more significance.

In conclusion, I would like to summarize the views of the group which I represent regarding the clinical value of ballistocardiography in the study of patients with coronary atherosclerosis.[19] However promising the method may be in the future, it is at present primarily a research tool. We strongly urge that its use be confined to clinical research. The widespread application of ballistocardiography in the clinical evaluation of the individual patient could prove most dangerous for reasons which must be apparent to all.

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Discussion

Henry Longstreet Taylor, University of Minnesota: The ballistocardiograph is a new device and we are still in the process of learning how to use it. The growth of knowledge regarding a procedure of this kind can at times be irregular, occasionally misleading and not infrequently discouraging. Those of us who have been working in this field have been both enthusiastic regarding the possibilities of learning something about a new aspect of the functional state of the myocardium and at the same time have been rather fearful that the technical inadequacies of the early recording instruments would lead the field into disrepute. It seems to me that Dr. Scarborough has skillfully mixed the proper amount of caution and skepticism with an adequate proportion of hope. It should be emphasized that ballistocardiography is going through a major period of instrumental development. It appears that we are on the verge of being provided with physical instruments to record the motion of the body without artifacts. Such a development will materially reduce the uncertainties that currently exist in the interpretation of ballistocardiographic records. In addition it will make it possible for the physiologist to study, with some real hope of obtaining relatively precise results, the characteristics of the internal network of arteries, tendons and other tissues which transmit cardiac forces to the mass of the body. So we should not be unduly discouraged with the obvious failure of the current ballistocardiographs to aid a great deal in the diagnosis and management of coronary disease.

Dr. Scarborough has pointed out that one of the major problems facing ballistocardiography today is the interpretation of the record which has an abnormal wave form and/or low IJ force in an otherwise healthy individual. It has been suggested by Dr. Isaac Starr and others that such individuals are suffering from what amounts to subclinical coronary artery disease. This possibility has stimulated us to examine the usefulness of the ballistocardiogram in population comparisons as well as in long term follow up studies of initially healthy individuals. Our recent comparisons of the ballistocardiographic characteristics of American and Italian men indicate some intriguing possibilities. The basic idea in this work was that the BCG age trend found in clinically healthy American males may be different in other populations less subject to atherosclerosis and coronary heart disease. In this country samples of clinically healthy men at successively older ages from twenty to sixty show an average rise in the frequency of "abnormal" BCG records and an average decline in the I-J force. Now from such men, at some later date, the victims of coronary heart disease are recruited and we know that many of the "healthy" American males after the age of thirty-five already have marked atherosclerosis. On the other hand, men in Italy are drawn from a population in whom coronary heart disease is much less frequent than here in the United States. The fact is shown not only by vital statistics but has been verified by extensive clinical surveys and other studies by Dr. Ancel Keys and Dr. Paul D. White and their colleagues.

On some 200 Italian males in either Naples or Cagliari (Sardina) records were obtained with a high-frequency, undamped Starr Type BCG bed. These men were clinically healthy and were selected to be reasonable representative of the local population. It was found that there was no difference approaching statistical signifiance between the frequency of "abnormal" BCG wave patterns found in Italians and those found in Americans up to the age of sixty. It seems reasonable to conclude that the increasing pattern abnormality with age in both countries is more closely related to the aging process *per se* or to some processes associated with it, than it is to coronary artery disease. However, when we turn to the measurement of the IJ force a different picture appears. It was found that the IJ force, measured on those records in which the ballistocardiographic pattern was judged to be normal, was much larger in Italians than in Americans in the fourth, fifth and sixth decades. The possibility that these observed differences could occur by chance was less than 1 in 1000 as judged from application of the standard T test. This difference requires further investigation before one can hazard a conclusion as to its meaning. But the inference is there and it seems possible, at least, that the ballistocardiograph may develop into a useful tool in the study of the epidemiology of heart disease and that force measurements may have some prognostic significance.

All but a handful of Minnesota's eighty-seven counties will have Heart Fund chairmen for the 1956 drive in February. Field representatives from the Saint Paul office of the Minnesota Heart Association have been busy for several months recruiting local chairmen in all parts of the state. Upon accepting the invitation to serve, the typical county chairman in Minnesota becomes responsible for recruitment of city chairmen in each community within his county borders.

Working several months in advance, many county chairmen succeed in recruiting Heart Fund solicitors at even the township level. Complete organization of the state is the ultimate recruitment goal of the Minnesota Heart Association, and volunteer workers are the lifeblood of the movement.

Since Minnesota is recognized as a world center of heart disease research, MHA feels a strong obligation to state residents to lead the way by raising many Minnesota dollars to take advantage of the wonderful research facilities existing within the state.

Medical Emergencies in Myocardial Infarction

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MYOCARDIAL infarction is a consequence of locally impeded coronary flow of such degree and duration that necrosis of muscle results. Fundamentally, the sequence of events begins when two deficiencies occur: the supply of oxygen is locally insufficient to maintain equilibria vital to contemporary myocardial function, and the flow of fluid (blood) is inadequate to sweep away the end products of myocardial metabolism. Up to a point this sequence is reversible: as oxygen supply and blood flow improve, or as the load upon the heart muscle diminishes, normal function is restored. Beyond this more generalized reactions are evoked which distinguish the presence of injured, necrotic tissue.

The medical emergencies in myocardial infarction surely begin with the compelling need for accurate diagnosis, to distinguish between reversible changes in myocardial function manifest in transient abnormalities of the electrocardiogram and irreversible injury to heart muscle. Currently this rests principally upon the demonstration of changes remote from the heart itself: leukocytosis, accelerated erythrocyte sedimentation rate and fever—a series of reactions on the part of the body to the necrotic area in the heart.

To these indices of myocardial infarction there has lately been added elevated levels of glutamic-oxalacetic transaminase.[1,2] This enzyme is commonly present in the sera of normal adults but in concentrations less than 40 units per ml. In most patients with other signs and symptoms of myocardial infarction the serum transaminase level rises to a maximum of 100 to 300 or more units within three days after the first manifestations of pain, presumably due to release of this enzyme from damaged heart muscle, and returns to normal within a week or less. Although it requires careful preparation of reagents, and its significance as an index of myocardial necrosis is highly questionable in the presence of liver disease, the estimation of serum transaminase level may take its place with the other tests applied to the diagnosis of myocardial infarction.

A second emergency, disorders of the heart beat, may overtake the patient without warning and sometimes with disastrous consequences. Auricular arrhythmias may further embarrass the function of the injured ventricle but are rarely fatal. Auricular fibrillation or flutter calls for rapid digitalization, if ventricular rate is rapid or signs of pulmonary congestion appear—0.8 mg. lanatocide C intravenously followed by 0.2 to 0.4 mg. in four to six hours.

Ventricular arrhythmias, on the other hand, are much more serious. At the border between the injured area and the normal ventricular muscle the products of myocardial metabolism or damage accumulate as irrigation is deficient. In this zone local excitability may become peculiarly instable. Sometimes this provokes no more than distortion of the electrocardiogram; sometimes it is manifest as a focus of ventricular extrasystoles. Neither of these phenomena is significantly dangerous to life, but when these are combined with more generalized changes in myocardial excitability or conduction, ventricular tachycardia or fibrillation may supervene. Prevention or treatment of these disorders must be based upon two factors: the influence of drugs—quinidine or procaine amide—upon excitability and conductivity in the heart muscle, and improvement of the intrinsic myocardial circulation. For ventricular tachycardia quinidine may be given in doses of 0.4 gm. every two to three hours. If arrhythmia persists this may be raised to 0.6 gm. per dose. If intravenous medication seems imperative procaine amide may be injected slowly—not more than 0.1 gm. per minute—up to a total of 1 to 5 gm. Neither quinidine nor procaine amide is indicated as a routine preventive measure. Indeed, when myocardial infarction is associated with "shock" both quinidine and procaine amide must

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be used with extreme caution since each has a tendency to provoke hypotension. Otherwise, when premature beats are observed quinidine may be employed to advantage—0.2-0.3 gm. every three to four hours.

Finally, due to the injured area of muscle, myocardial contraction and therewith ventricular stroke volume may become seriously deficient. During the acute phase of myocardial infarction a moderate and transient fall in blood pressure is not uncommon. Although this requires watching it usually passes off and is not associated with symptoms and signs of collapse. On the other hand, in about one half of the fatal instances (10 per cent or less of all cases) death is preceded by a state of "shock": severe hypotension (systolic pressure 90 mm. Hg or below) with small "thready" pulse (pulse pressure 25 mm. Hg or less) plus cold moist skin, pallor, weakness, usually tachycardia, and oliguria. The most significant factor at the basis of this complication is certainly hypodynamic contraction of the injured left ventricle with consequent reduction of cardiac output and of "effective" (intra-arterial) blood volume.* With the critical fall in blood pressure, blood flow through the entire coronary arterial bed is diminished and to the locally damaging effects of occlusion there is added reduced irrigation of the remaining myocardium. If this circulatory state becomes established and persists recovery is uncommon.

The first step in preventing shock with myocardial infarction is to be on guard. It is by no means necessary to interrupt rest to measure blood pressure every few minutes but change in heart rate or in quality of the pulse should not fail of observation. If, as in some instances, acute hypotension is precipitated by some rapid arrhythmia this should be promptly, though cautiously, treated.

The general measures usually employed in treating myocardial infarction, particularly the use of oxygen, apply equally in the case complicated by "shock." However, one may express caution regarding morphine and a preference for meperidine (demerol—25 to 75 mg.) intramuscularly for relief of pain in the presence of hypotension.

One cannot sharply distinguish fundamental differences between shock with myocardial infarction and traumatic or hemorrhagic shock, but in the first, left ventricular failure is certainly a more common complication. Digitalis should be administered if congestive failure is evident; overdosage is to be avoided.

All the methods of treating hemorrhagic or traumatic shock have been applied to the condition under consideration, including transfusion—intravenous or intra-arterial—of blood or plasma. Perhaps because the total blood volume is not commonly so critically decreased in shock with myocardial infarction—indeed, systemic venous pressure may be increased—transfusions do not seem to be so dramatically useful here as in shock following hemorrhage or trauma.

Epinephrine had been occasionally tested as a vasopressor agent in this variety of shock but only with some apprehension because of its striking action upon the rate and force of the heart beat and upon myocardial irritability. However, other drugs are now being produced in rapid succession which have the property of provoking peripheral vasoconstriction without hazardous direct effect upon the myocardium. If the treatment of myocardial infarction can be said recently to have advanced it is along the line of this adjunct.

The most potent of the available substances is 1-norepinephrine (1-arterenol) but it must be administered by vein; it causes necrosis if injected or extravasated into the muscle. Dosage: 4 to 24 cc. of 1/1000 solution added to 1000 cc. 5 per cent glucose and given by slow intravenous drip. Others suitable for intramuscular or intravenous injection have a milder and more evanescent action. These include:

N-methylphenylbutylamine sulfate ("Wyamine"): dose 35 to 70 mg. in 5 per cent glucose I.V.** by slow drip or 15 to 45 mg. I.M.†

Phenylephrine hydrochloride ("Neo-synephrine"): dose 5 to 10 mg. I.M.† or I.V.** (in dilute solution) every fifteen to thirty minutes.

Hydroxyamphetamine ("Paredrine"): dose 10 to 20 mg. I.M.† or 5 to 10 mg. I.V.** in dilute solution.

B-hydroxy-B-(2,5-dimethoxyphenyl)-isopropylamine ("Methoxamine"): dose 30 to 50 mg. I.M.† or less I.V.**

Isopropylnorepinephrine ("Isuprel") hydrochloride has proved of value in cases of shock associated with heart

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*In experimental animals (dogs)[3] there is some evidence of a mechanism, probably neurogenic, which operates when coronary vessels are occluded and which appears to inhibit a compensatory rise in peripheral resistance.

**I.V. = by intravenous injection.

†I.M. = by intramuscular injection.

TABLE I.[7] 82 PATIENTS WITH SEVERE SHOCK (CORONARY OCCLUSION) (BINDER, ET AL, 1954)

No.	Therapy	Survived	Died
22	Non-specific	4	17 (82%)
35	I.V. and I.A. Transfusion + vasopressor drugs	2	33 (94%)
25	1-arterenol	8	17 (68%)

TABLE II. THERAPY OF SEVERE SHOCK (CORONARY OCCLUSION) (BINDER, ET AL[7] AND FROM LITERATURE)

No.	Therapy	Survived	Died
59	I.V. Transfusion	6	53 (90%)
42	I.A. Transfusion	8	34 (80%)
131	1-arterenol	53	78 (60%)

block. Dose: 2 to 3 mg. I.V.** or 7.5 to 15 mg. under tongue.

Levo-1 (m-hydroxyphenyl)-2-amino-1-propanol ("Aramine") recently introduced is described by Sarnoff, et al[4] as accomplishing marked rise of blood pressure from hypotensive levels, increased coronary flow and improved myocardial contraction without requiring greater coronary flow per unit of work, and without a tendency to provoke cardiac arrhythmias. These observations have been confirmed in limited clinical trial.[5,6] Dose: I.V.** infusion - 2 to 6 cc./min. - containing 50 to 200 mg./liter.

Drs. Binder, Ryan, Marcus, Mugler, Strange and Agress[7] of the Department of Medicine, University of California at Los Angeles, recently tabulated the results of various forms of therapy of shock with severe myocardial infarction as reported in the literature, adding eighty-two cases of their own.

Conclusion

In the end the outcome of any attack of myocardial infarction will be determined—barring fatal embolism—by the extent and severity of myocardial damage. By treating hypotension when it occurs one hopes to sustain the circulation to vital organs, including the remainder of the myocardium. Combined with oxygen therapy this may limit the extent of necrosis, and improve the function of the hypodynamic left ventricle. It follows that such therapy should be instituted as early as possible after the blood pressure falls. It is seldom necessary to raise the systemic pressure above 120 mm. Hg. If it is elevated too fast or too high, pulmonary edema may be precipitated. If signs of pulmonary congestion accompany shock, one must resort to rapid digitalization. Judging from one's own experience and from the results reported by others, it may be possible to rescue perhaps twenty per cent of otherwise fatal cases of myocardial infarction by therapy with vasopressor drugs.

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Dollars donated by Minnesotans make up the Heart Fund. The drive has been conducted since 1949, when the Minnesota Heart Association was founded by a group of physicians. Since 1951, the February drive has been conducted by the Minnesota Association of Life Underwriters.

Heart Fund dollars go to support a three-point program of research, education and service. Major emphasis is placed on research support. But the Heart Fund dollar also is used to bring helpful information to the heart patient, and to help keep Minnesota physicians abreast of new developments in diagnosis and care of cardiac patients.

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The conquest of heart disease is only a matter of time, the scientist knows. But will the answers come today, tomorrow, or a decade from now?

Today, a little girl whizzes down the sidewalk on her tricycle because research found a way of closing a hole in her heart. Today, a businessman is back at his desk because research discovered drugs that can lower his elevated blood pressure.

These answers came in time for some . . . too late for others. Research supported by funds from the Minnesota public is the answer to this question of time.

Evaluation of Status and Results of Management in Coronary Heart Disease

HENRY I. RUSSEK
Staten Island, New York

MORE than one hundred and fifty years ago Jenner[1] expressed the fear that the medical world may seek in vain for a remedy for coronary atherosclerosis. Today with brighter hopes but still with a meager background of fundamental knowledge, we are witnessing thrusts into the unknown which may provide sound discoveries in the treatment and prevention of atheromatosis. Until such effective measures become available, the physician can only strive to alleviate some of the consequences of this baffling disease by applying the lessons learned from his daily encounter with civilized man's greatest killer.

Throughout the history of medicine the use of a multiplicity of drugs and procedures in treating a specific illness has usually been the ear-mark of therapeutic failure. The prevailing tendency appears to be to overtreat patients with coronary disease than otherwise. It should be recognized that under proper supervision a large proportion of these cases will run their course from beginning to end with little or no medicine, and this often with significant advantage. Nevertheless, the advent of new methods for the prevention and treatment of complications arising in the course of coronary atherosclerosis has led in many quarters to the adoption of "routine treatment" for all cases. While many components of such routine programs are extremely valuable, they lose their value when indiscriminately and at times unnecessarily employed. In recognizing that judicious use of various drugs may relieve suffering and save lives, sight must not be lost of the fact that the major part in the control of this disease still lies in expert guidance in making the mental and physical adjustments which will enable these patients to carry on within their capacity without symptoms.

Angina Pectoris

In the general management of patients with angina pectoris or other forms of myocardial ischemia, a more realistic approach has been taken in recent years to insure that restrictions imposed are carefully individualized not only on the basis of coronary reserve but also in terms of the total life situation in each case. It seems reasonable to advise patients with angina to hold a normal weight. Although there are some conflicting data regarding the relationship between obesity and life expectancy,[2-4] the clinical observation that reducing the obese cardiac helps to decrease the work of the heart cannot be challenged. The apparent correlation between fat and cholesterol in the diet and atherosclerosis has led Keys[5] to advocate the avoidance of obesity and "periodic gorging," restriction of extractable fats to not over 25 to 30 per cent of total calories and perhaps restriction of cholesterol to under 1 gram a week.

Alcohol.—Alcohol has long been known to be a depressant of the central nervous system capable of decreasing anxiety and promoting a sense of well-being. It has a beneficial psychic effect upon some patients with angina pectoris and is often of value in relaxing tension at the end of a working day. In spite of strong evidence to the contrary, alcohol is still widely regarded as a potent coronary vasodilator, second only to the nitrites. The failure of alcohol to influence the electrocardiographic response to standard exercise in coronary patients[6] indicates, however, that its ability to relieve anginal pain is based solely on a sedative action (Fig. 1). Since the administration of ethyl alcohol prophylactically may not only create a false sense of physical fitness but remove the protection afforded by angina, the danger of its employment prior to contemplated exertion should be recognized. The calorie content of alcohol also cannot be disregarded (100 proof liquor contains 100 calories to the ounce),

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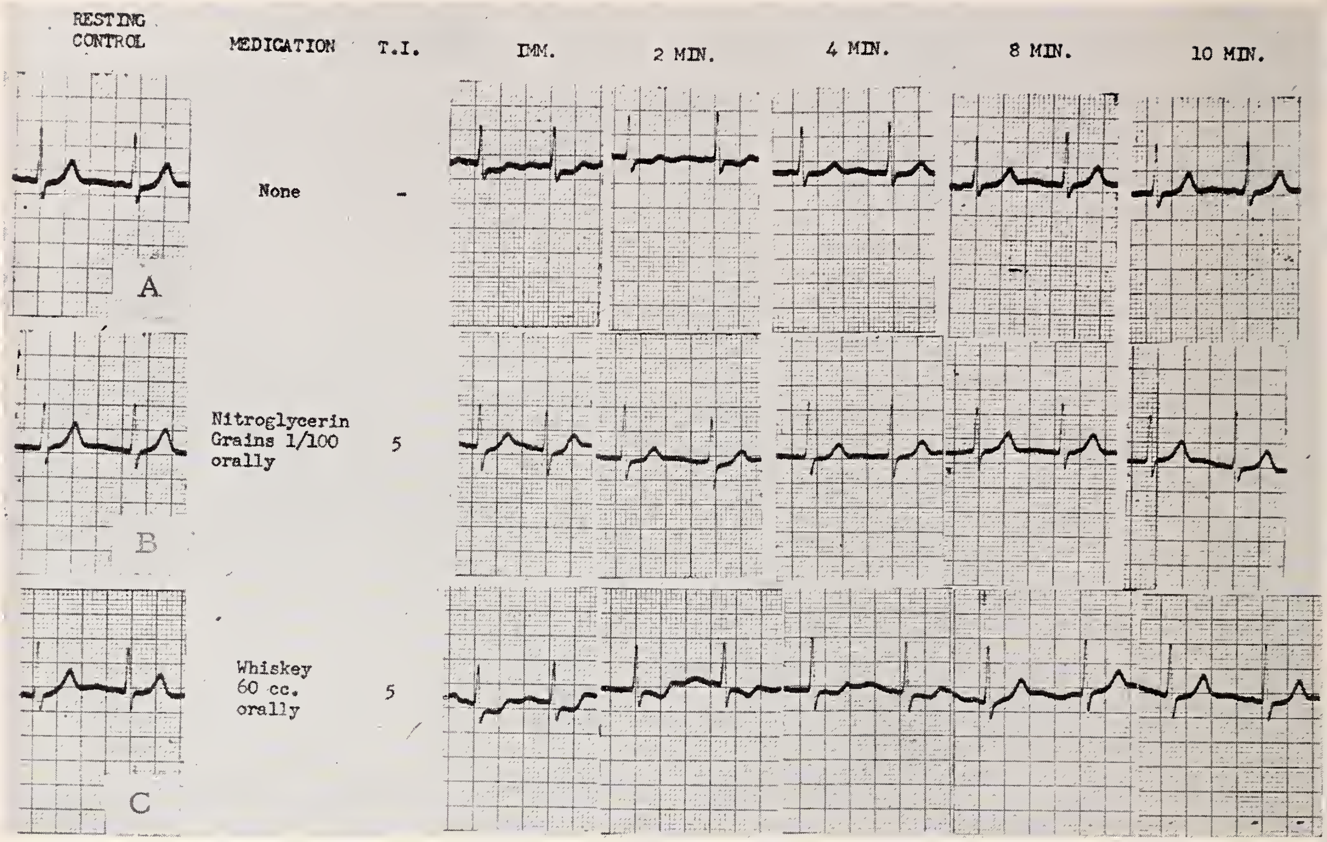

Fig. 1. Electrocardiograms (lead CF_4) showing modifying influence on resting control of standard exercise. (Master two-step test). Response A follows no medication; response B follows 1/100 grain (0.4 mg.) glyceryl trinitrate sublingually, time interval (T.I.) five minutes. Response C follows 60 cc. whiskey orally, time interval five minutes.

nor can its tendency to stimulate the appetite. One may therefore conclude that alcoholic liquor should not be a routine prescription for all patients and should no longer be regarded as possessing coronary vasodilator properties.

Tobacco.—Although there is no convincing proof that tobacco directly causes or intensifies the average case of angina pectoris, it has become common practice for physicians to advise against its use in all patients suffering from this affliction. As Sprague[7] has stated, "A doctor who cannot contemplate life without tobacco is hardly objective about the dangers of smoking." Because of the enjoyment afforded and the emotional satisfaction obtained some believe that patients with inactive forms of heart disease should be permitted to smoke in moderation.[8,9] Although there are no valid experimental data on the effect of nicotine on the coronary arteries or on the heart, numerous investigations have clearly shown an augmentation of the systolic and diastolic blood pressures, heart rate and basal metabolic rate in response to smoking. Tobacco also exerts well-known vasoconstrictive effects on the peripheral circulation even in normal subjects. These observations have led to the belief that smoking may exert an adverse influence on coronary circulation. Davis and associates[10] suggest that changes in the ballistocardiogram produced by smoking may be the result of nicotine action on the hypothalamic-posterior pituitary axis, with resultant release of vasopressin (Pitressin), which acts as a coronary vasoconstrictor. Our own studies[11,12] in patients with coronary disease have failed to confirm this mode of action of nicotine. Thus glyceryl trinitrate in almost all instances did not modify or prevent ballistocardiographic changes induced by smoking, and Pitressin, even in large dosage, failed to mimic the nicotine effect on ballistic pattern. Paradoxically, whiskey was found to block the ballistic changes in some patients who exhibited marked sensitivity to nicotine. In the light of previous evidence based on electrocardiographic tests[6] this could not be accepted as evidence of a coronary vasodilator action induced by alcohol. These observations were therefore interpreted as indicating that the ballistic response to smoking is evoked primarily by peripheral vascular constriction and not by alterations in coronary blood flow. Consequently it would appear that smoking does not present a direct danger to the patient with coronary disease through coronary vasoconstriction and that whiskey is not an effective coronary vasodilator and antidote for nicotine with respect to the heart. The antagonism between these drugs is exhibited almost entirely on the peripheral circulation. With respect to the heart, each increases the tendency to tachycardia and ectopic beats. The known cardiovascular, local and systemic effects of smoking clearly establish the inadvisability of the continuance of this habit in patients with heart disease.

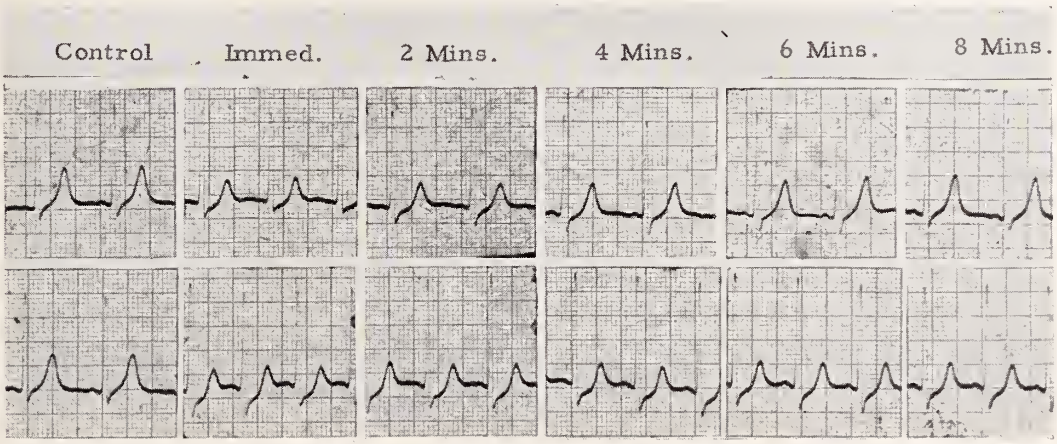

Fig. 2. Upper row, Master two-step test (lead V_4) in a patient with coronary disease. Lower row, the same test has been repeated following grain 1/150 nitroglycerine. Note more abnormal response following nitroglycerine.

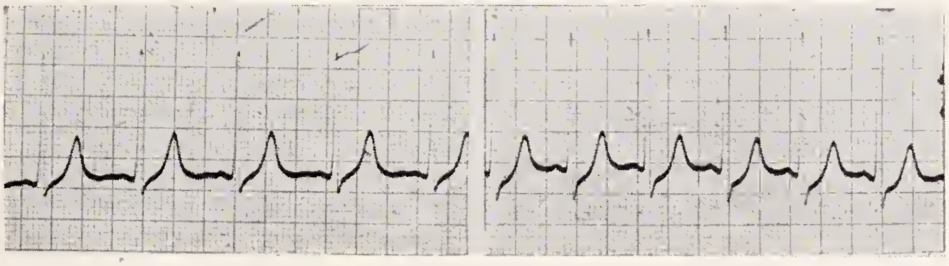

Fig. 3. (Lead V_4) in same patient as in Figure 2. At left, patient is resting supine. Tracing at right was recorded five minutes after 1/150 grain of nitroglycerine.

Medication.—Nitroglycerin still remains the most useful drug in the management of angina pectoris. Although great difficulty often exists in convincing the patient to use it as freely as necessary, it has not been adequately appreciated that the drug also possesses latent potentialities for adverse effects in this disease. A study of the modifying effect of varying amounts of nitroglycerin (1/300 to 1/50 grain) upon the electrocardiographic response to standard exercise has shown that individualization of dosage is an important prerequisite to rational therapy in angina pectoris.[13] In exercise tests 10 per cent of patients responded paradoxically to the drug as a result of venous pooling in the lower extremities, diminished venous return to the heart and reduced coronary blood flow in spite of concomitant coronary vasodilatation. These undesirable effects were prevented by reduction in the dosage of nitroglycerin and lessened by the application of elastic bandages to the lower extremities. The optimum dosage for most patients was found to be 1/300 to 1/200 grain sublingually. The precipitation of acute myocardial infarction appears to be more than a theoretic danger from overdosage during treatment of the anginal attack (Figs. 2 and 3).

Because of the relatively short duration of the action of nitroglycerin, the search has continued for a long-acting coronary vasodilator which is capable of reducing the frequency and severity

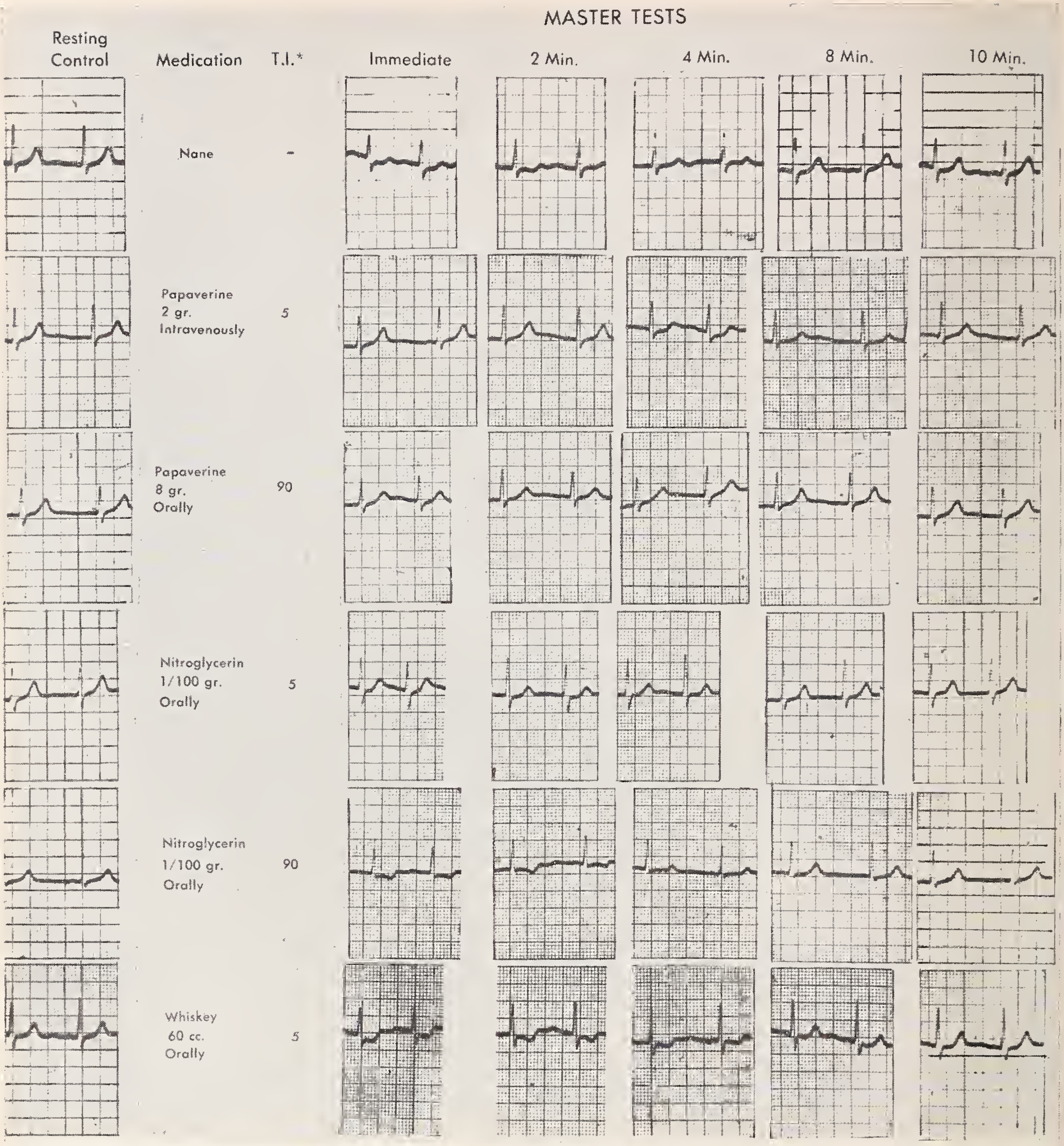
MASTER TESTS
Resting Control
Medication
T.I.*
Immediate
2 Min.
4 Min.
8 Min.
10 Min.
None
-
Papaverine 2 gr. Intravenously
5
Papaverine 8 gr. Orally
90
Nitroglycerin 1/100 gr. Orally
5
Nitroglycerin 1/100 gr. Orally
90
Whiskey 60 cc. Orally
5

Fig. 4. Master test responses (lead V_4) in a patient with coronary insufficiency with and without the administration of drugs.

*T.I., time interval (minutes) between medication and beginning of test.

of anginal attacks by routine daily administration. Many drugs have been characterized by their individual sponsors as fulfilling this need but few have been able to withstand the test of time and clinical usage. Realizing the difficulties in clinical evaluation which is dependent solely on the relief of pain, we have attempted, during the past six years, to employ an objective method based on the electrocardiographic response to the Master two-step test in carefully selected patients.[14-17] Our conclusions may be summarized as follows:

Papaverine, in spite of the disappointing results reported by others, appears to be an effective coronary vasodilator when prescribed in a dosage two to four times greater than that in common usage (Fig. 4). Dioxyline (paveril) phosphate, when administered in doses similar to those em-

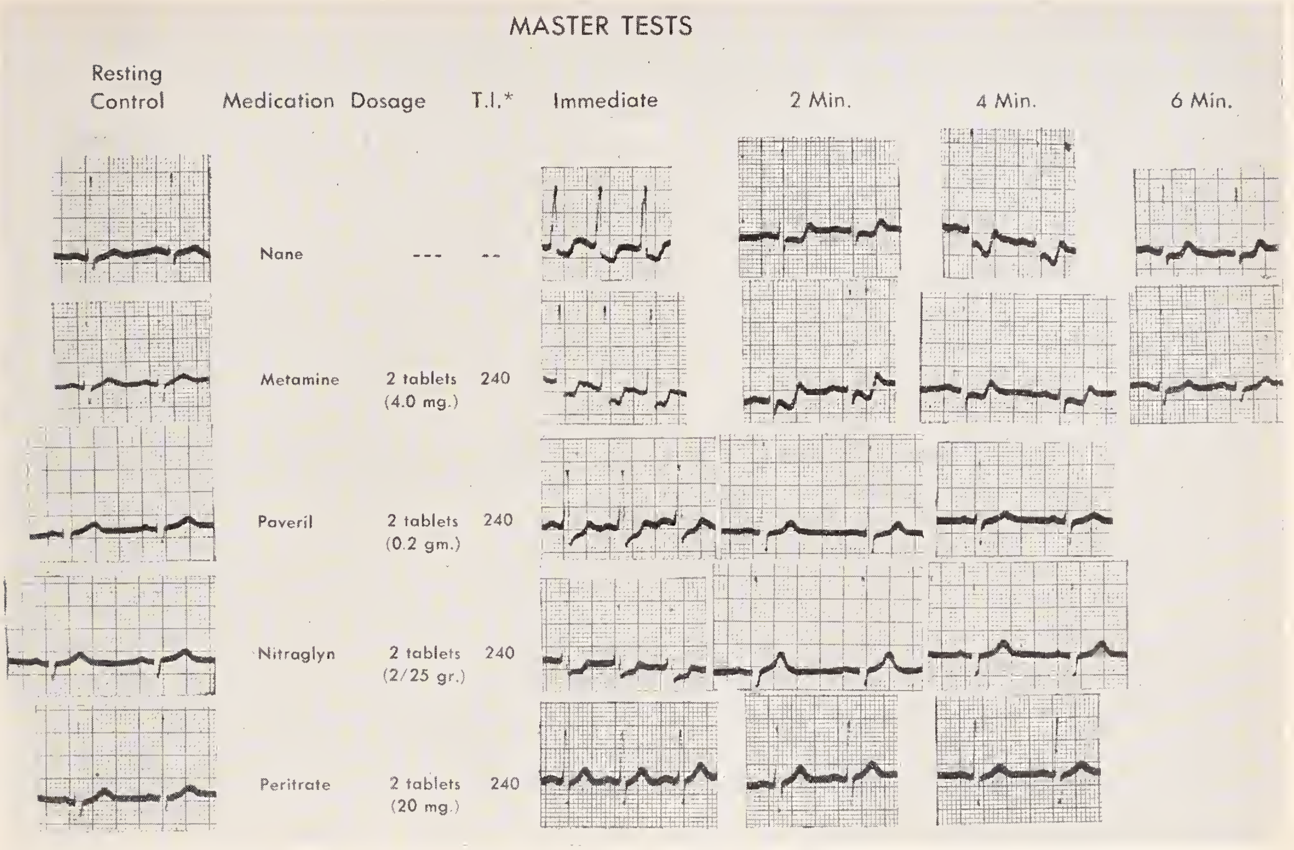

Fig. 5. Master test responses (lead V_4) obtained in a patient with angina pectoris with and without the administration of drugs.

*T.I., time interval (minutes) between medication and beginning of test.

ployed with papaverine did not give comparable results. Paveril appeared to be a much less potent drug than papaverine according to this method of testing.

Xanthine Drugs: Aminophylline in the dose of 7½ grains intravenously or 6 grains orally was found to have slight to insignificant effect. Choline theophyllinate, which has recently been acclaimed by some groups,[18] appeared to be ineffective in the recommended dosage of 200 mg. four times daily. When this dose was quadrupled, however, benefit was noted in some patients but gastric distress was common.

Khellin even in massive dosage failed to alter favorably the electrocardiographic response in all patients tested.

Octyl nitrite by inhalation was at best as effective as 1/300 grain nitroglycerin.

Heparin: Much interest has been shown recently in the influence of heparin on various cardiovascular phenomena including lipid transfer in blood, reduction of the significant Gofman ultracentrifuge flotation fractions, and anginal pain. Our own observations[19] as well as those of others [20,21] have failed to confirm any influence on anginal pain or on exercise tolerance determined electrocardiographically.

Roniacol, tetraethyl ammonium chloride and Priscoline were without demonstrable value.

Metamine (triethanolamine trinitrate biphosphate) was found to exert little or no significant effect on the electrocardiographic response to standard exercise.

Nitroglyn (glyceryl trinitrate in sustained-action tablets) gave disappointing results in spite of logic behind its use. Dosage as high as 4/25 grain was without effect in a large proportion of patients.

Peritrate in 10 to 20 mg. dosage afforded protection for four to five hours after an initial lag of 1 to 1½ hours before its action became apparent. Of all the drugs tested, Peritrate appeared to be the most effective long-acting coronary vasodilator for the clinical management of angina pectoris (Fig. 5).

Acute Myocardial Infarction

Less than twenty years ago the diagnosis of coronary occlusion occupied all the space allotted to the subject in our medical literature because therapy seemed to be so limited and futile. The role of the physician in the care of his patients was little more than that of an attentive spectator of nature's processes of healing and repair. With the advent of new concepts and methods of management this traditional regimen of "watchful waiting" has been gradually replaced by a positive therapeutic program of "active intervention." Despite the improved prognosis attending these radical innovations in therapy, no unanimity of opinion exists, even among authorities, regarding the indications, results and dangers associated with many of the specific measures now employed.

Morphine.—For the relief of pain all physicians are agreed that morphine and other opiates are without equal. In the past an initial dose of no less than half a grain of morphine was recommended routinely in the belief that smaller doses do no good and that substitutes are worthless. In spite of its wide use, little is known of the effects of morphine on the coronary arteries although the vague notion exists that the drug exerts a direct beneficial action in coronary thrombosis independent of its influence on symptoms. Experimentally, morphine given intravenously has been shown to produce increased coronary flow of brief duration in animals with acute myocardial infarction[22] but clinical confirmation of this action has not thus far been obtained. Although its characteristic influence on pain was clearly apparent, no significant effect on the electrocardiographic response to standard exercise (Master two-step test) has been observed from the use of the drug in patients with angina pectoris.[15] On the other hand, morphine sometimes complicates the course of coronary thrombosis. Its undesirable actions in producing abdominal distension, urinary retention, respiratory depression, vomiting and retching are well known. Morphine causes strong vagal stimulation, rendering the heart more susceptible to ventricular ectopic rhythms; it increases biliary pressure and exerts an antidiuretic and hypotensive effect. In doses of about 15 mg. the drug has no effect on the circulation in recumbent patients but may precipitate a hypotensive syncopal attack or collapse in patients with acute myocardial infarction who are placed with the head up and feet hanging. Consequently, the use of morphine, like the employment of bed rest, quinidine or anticoagulant drugs is associated with a calculated risk. As a result of advances in the recognition of milder cases of this disease, the old dictum calling for large doses of morphine routinely as soon as the diagnosis is made can no longer be countenanced. Large doses are occasionally necessary as is intravenous administration, but mild pain can often be controlled by demerol or codeine and in some cases even these are unnecessary. Obviously, no "rules" can ever take the place of sound clinical judgment.

Oxygen.—The value of oxygen in relieving dyspnea, cyanosis and cardiac pain in cases in which cardiac damage and circulatory derangement have resulted in a secondary generalized arterial anoxemia has been clearly established. The question as to whether or not hyperoxygenated blood is capable of influencing local myocardial anoxia in patients with uncomplicated myocardial infarction, however, has not thus far been satisfactorily answered. In normal animals which admittedly have no functional collaterals, administration of oxygen has proved valueless in experimental occlusion.[23] In other studies[24] on anesthetized dogs, however, it has been shown, using platinum electrodes, that there is a statistically significant increase in available oxygen in border zones surrounding areas of infarction after pure oxygen breathing. Fifty per cent oxygen proved greatly inferior to pure oxygen in achieving favorable results. These observations support the belief that inhalation of high concentrations of oxygen may reduce the extent of muscle necrosis resulting from coronary occlusion in man.

On the other hand, in the anesthetized animal, one does not encounter the psychogenic influences evoked by the administration of oxygen in the human subject afflicted with coronary thrombosis. Administration of oxygen to most patients carries the implication that they are critically ill. The claustrophobia which some patients develop on being placed in an oxygen tent is well documented. Administration of oxygen by nasal catheter frequently leads to irritation or inflammation of the pharynx which cause hacking and coughing, both undesirable efforts. The use of a mask often produces paradoxically a sense of suffocation and a state of anxiety. Such factors surely contribute to the total work of the heart and, therefore, are likely to nullify or even overbalance any advantages to be gained from the relatively little additional oxygen which can be added to the blood in patients with uncomplicated myocardial infarction. With the concentrations of oxygen attainable in the use of a tent only 1.6 cc. of the gas can be added to each 100 cc. of blood. Nearly twice this amount can be added if the patient inhales 100 per cent oxygen.[25] The effect of this therapy on the electrocardiographic response to standard exercise using the Master two-step test has been studied in a series of patients with angina pectoris.[26] It was found that the administration of 100 per cent oxygen did not prevent the RS-T segment and T wave changes induced by exercise and failed to hasten the disappearance of these manifestations of myocardial anoxia. The administration of 100 per cent oxygen, moreover, failed to prevent the onset of anginal pain or to influence its duration. Indeed, some observations in which exercise response actually deteriorated under oxygen therapy suggest that hyperoxygenated blood may interfere with compensatory coronary vasodilatation evoked by local myocardial anoxia.

It is concluded, therefore, that oxygen should be freely employed in acute myocardial infarction when an indication for its use is recognized or even suspected. Its continuation, however, should be based on evidence of objective or subjective improvement of the symptom for which it was administered. It would appear that the indiscriminate use of oxygen may contribute nothing to the patient's ultimate recovery and yet add considerably to his anxiety and discomfort during the most acute phase of the disease. In the uncomplicated case even the high concentrations obtainable with a mask have questionable value, but if oxygen is to be used at all only this method of administration would seem to deserve consideration.

TABLE I. MORTALITY RATE AND INCIDENCE OF THROMBO-EMBOLIC COMPLICATIONS

	No. of Cases	Mortality		Embolization	
		No.	%	No.	%
Total	1047	350	33.4	63	6.0
Good risk	489	15	3.1	4	0.8
Poor risk	558	335	60.0	59	10.6

Anticoagulants.—Although anticoagulant therapy has become an accepted form of treatment of acute myocardial infarction, there is still much disagreement, even among authorities, regarding the indications for its use. The statistical data presented by the committee on anticoagulants of the American Heart Association, as well as that recorded from numerous other sources, indicate that anticoagulant drugs exert a markedly favorable influence on both the death rate and the thromboembolic complication rate in unselected groups. In spite of this convincing evidence, none of these studies have actually shown that clinically mild or low risk groups share in the overall benefit derived from this form of therapy. There is, in fact, a growing weight of opinion in favor of withholding these agents from patients who have had a clinically mild infarction.

It has been shown that the mortality rate and incidence of thromboembolism are so low among patients sustaining an "uncomplicated" first attack[27] that anticoagulant therapy with its attendant dangers from hemorrhage could scarcely be expected to influence the prognosis favorably in this low risk group (Table I). The objection raised against flexibility of therapy relative to our judgment of the severity and anatomy of the attack, is that one cannot always be sure of what constitutes a small myocardial infarct and further that what starts as a mild attack may become severe a few hours later. In a recent study in which anticoagulants were intentionally withheld in 122 patients with acute myocardial infarction because of the absence of certain poor prognostic signs* at the time of the first examination, we have found that the preventable mortality under ideal anticoagulant therapy could not have exceeded 0.8 per cent.[28] In the entire series not a single instance of cerebral or peripheral arterial embolism was encountered. Clinical thromboembolic phenomena occurred in four patients, an incidence of 3.3 per cent. In two of these four patients moreover, the diagnosis of this complication was only presumptive. The risk of thromboembolism in such cases therefore may be equalled or exceeded by the dangers attending the use of anticoagulants. After careful consideration of all clinical and autopsy data Wright[29] recently estimated that 1.7 deaths per 100 cases were the result of anticoagulant therapy itself.

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*Patients were considered to be "good risks" when none of the following poor prognostic signs were observed on the day of admission to the hospital: (1) previous myocardial infarction, (2) intractable pain, (3) extreme degree or persistence of shock, (4) significant enlargement of the heart, (5) gallop rhythm, (6) congestive heart failure, (7) auricular fibrillation or flutter, ventricular tachycardia, or intraventricular block, and (8) diabetic acidosis or other states predisposing to thrombosis.

It is difficult to translate the carefully controlled experiments of the committee on anticoagulants, under ideal conditions in research hospitals, to the every day treatment of coronary disease in practice. The imperfections, failures, and inherent dangers in this form of therapy even in the hands of the most competent investigators however should leave little hope of improving the prognosis in "good risk" cases with the anticoagulants now available and the current method of control. In this group, the small risk of thromboembolism without such treatment does not appear to justify the use of a procedure that itself may lead, even if infrequently, to serious complications and death. The occurrence of poor prognostic signs in a "good risk" patient at any time in the course of his convalescence certainly should establish the need for intensive anticoagulant therapy, but, in our experience such a development after the first forty-eight hours following the attack is relatively infrequent. It is therefore concluded that anticoagulant therapy for "mild" attacks is a costly, burdensome, and unnecessary form of treatment. Ample evidence exists to justify "prognostic classification" as a means of selecting patients for anticoagulant therapy in acute myocardial infarction.

It has been repeatedly claimed that old age provides a special indication for the use of anticoagulant drugs in acute coronary occlusion. We have shown that no justification exists for the concept that age is an important factor in determining the prognosis in the individual patient.[30] Statistically, there is a higher incidence of serious attacks among older patients and consequently a more frequent occurrence of thromboembolism. For this reason alone, anticoagulants have shown a greater life-saving action in patients over the age of sixty than in those below this age. Our comparison of cases of similar severity in different age groups have demontrated repeatedly that the chances for surviving an attack of myocardial infarction are correlated not with age but with the severity of the clinical picture. One must therefore conclude that the initial clinical appearance of the patient, irrespective of age, constitutes the best index to his future course and is the deciding factor regarding the need for anticoagulant therapy.

Out of a total of 1,318 consecutive admissions for acute myocardial infarction, 611 (46 per cent) qualified as "good risk." When account is taken of the patients with mild attacks who are treated at home by conservative measures and the persons with serious episodes who manifest contraindications to anticoagulant therapy probably no more than 30 per cent of all patients can be considered candidates for this form of treatment. This low figure however should not detract from the value of anticoagulant therapy in properly selected patients (those with poor prognostic signs), since its administration in such instances constitutes a major advance in the treatment of this disease.

Hospitalization.—The wide employment of anticoagulant drugs in acute coronary thrombosis has been associated with a risk commonly overlooked. In order to institute treatment without delay many patients are rushed to the hospital shortly after the onset of their clinical attack despite the fact that the highest death rate in this disease prevails during the first forty-eight hours. Since these early deaths are the result of ventricular fibrillation, cardiac asystole, shock or congestive heart failure and are unrelated to thromboembolism, they almost certainly cannot be prevented by anticoagulant drugs. The desire to save the small proportion who may die from thromboembolism after the first week may therefore jeopardize the chances for survival of the much larger segment of coronary cases known to die from other causes during the first two days. For many patients, being jostled in an ambulance or admitting office, then subjected to the psychic trauma of a hospital room and bed and of being placed in an oxygen tent, often when oxygen is entirely unnecessary, and being repeatedly punctured for prothrombin and coagulation times, hardly constitutes optimum management during this early critical period. In most instances, any form of treatment deemed necessary can be initiated in the patient's own home and removal to hospital can be accomplished if indicated, when this critical period has passed.

Bed Rest.—One of the major problems in the management of patients with myocardial infarction involves the decision concerning the duration and rigidity of restriction of activity. A little more than a decade ago even the milder cases were nursed in bed for at least eight weeks and were "guarded by day and night nursing and helped in every way to avoid voluntary movement or effort." The tendency to reduce the bedfast time of patients with myocardial infarction appears to be a forward step. Since cardiac work tends to be less in the sitting than in the recumbent position the "chair" treatment advocated by Levine cannot be challenged on the grounds of failing to give maximum rest to the heart. This method obviates some of the well known harmful effects of strict bed rest, permits easier commode privileges and protects against the great bedpan hazard.

The wisdom of utilizing chair treatment during the first few days of the illness, however, should be questioned. Considerably more than one half of all the deaths in this disease occur during the first two days and, even in the mildest cases, a high proportion develop suddenly within the first forty-eight hours. Many of these early fatalities are the result of cardiac standstill, ventricular fibrillation, shock or congestive heart failure. For this reason it would seem best that the patient be disturbed as little as possible during the first few days of his illness. The hypotensive effect of the opiates when the head is elevated and the feet are dependent must also be considered when "chair" treatment is to be employed soon after the onset of the attack. Most physicians appear to share the view that chair treatment offers little advantage over bed rest when such measures as deep breathing, freedom of movement, and mild exercise of feet and legs are permitted. Dependency of the legs with the patient seated in a chair would appear to predispose to thrombotic complications rather than to prevent them. Nevertheless, in the presence of dyspnea and pulmonary congestion the venous pooling produced by lowering of the legs should not be minimized as a therapeutic measure. Some physicians advocate the use of the bedside commode while paradoxically cautioning against the risk of chair treatment. The tendency of patients nursed in this manner to take too light a view of their illness and to disregard protective restrictions has been emphasized by various authors.

Transfusions, Vasopressor Drugs and Digitalis.—If further improvement in the prognosis of acute myocardial infarction is to be obtained, the problems involved in the prevention and treatment of the severer grades of shock and congestive heart failure must be solved. It has been claimed that protracted shock, if survived predisposes to a greater or less degree of congestive failure at an early date. Evidence has been presented which suggests that the prompt and efficient treatment of cardiogenic shock may well be the means of preventing the subsequent development of congestive heart failure.[31] The latter may, therefore, be anticipated on occasions and necessary measures taken in advance. The treatment of severe shock following acute myocardial infarction is a challenging and usually frustrating experience because of the mortality rate in excess of 80 per cent of cases, the confusion of procedures advised and the futility of most management. The value of the usual non-specific measures, of transfusions and of various vasopressors has been difficult to assess because of a lack of uniformity in criteria for the classification of shock. There is little evidence that intravenous transfusion alone is of value in the treatment of severe coronary shock. In some instances patients so treated fare worse than do comparable patients who are treated non-specifically. Intra-arterial transfusion is not associated with a higher incidence of relief of shock or of survival when compared to a valid control series. To date, the most encouraging results have been reported with vasopressor drugs, notably levarterenol, which may reduce the mortality some 20 per cent.[32] Because the mechanism of coronary shock remains obscure, present day therapy continues to be largely empirical. The relative importance in the pathogenesis of coronary shock of decreased cardiac output, cardiopulmonary reflexes and peripheral vascular collapse remain to be assessed.

In summary it would appear that the maintenance of a safe, yet comprehensive therapeutic philosophy in the treatment of the patient with coronary disease demands utmost circumspection to insure that both the science and the art of medicine are being utilized wisely.

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Discussion

Howard B. Burchell, Mayo Clinic: Dr. Russek has adequately outlined the current thoughts, accepted advances, and controversies in the management of persons with coronary disease, and there is no need to rediscuss all the specific details of treatment. I would beg the indulgence of the symposium members in re-emphasizing the background, or frame of reference, into which the detailed therapeutic manipulations which Dr. Russek has described must be placed.

We still have the serious problem exemplified by the individual who may have been assured by a competent cardiologist that he had a normal heart, and yet that day even die suddenly or have an acute myocardial infarction. We may recognize that a patient, for instance an executive who might be referred to one of us for study, must have some pathologic quanta of coronary disease, but at the present time we have no way of accurately predicting its extent or its possible future manifestations. Thus the management of the adult population in general with *silent* coronary disease greatly overlaps the management of patients with *manifest* coronary disease. The importance of coronary disease as a cause of death has been repeatedly emphasized, and when we studied the medical deaths due to heart disease in our own city of Rochester in the years 1952-1953, we found that in 55 per cent coronary disease was *the* important factor, valvular and other disease affecting the heart trailing far behind. Of these individuals who died of coronary heart disease, 34 per cent died with an acute myocardial infarction.

We must suspect coronary disease in many in our adult population even when they do not give a history of, or a clue to, its presence.

As further evidence for this assertion, Dr. Achor in our Clinic, studying the records of patients who showed a healed infarct of the heart at post mortem examination, found that in somewhat over a third a presumably accurate history did not contain the information suggesting the infarction nor had the diagnosis been made. He found also, in agreement with the reports of others, that in nearly 10 per cent of all people dying and having a post mortem examination a healed gross infarct will be found.

In the past few years our approach to an attempted understanding of the clinical problem presented by coronary disease has been analysis of how individuals die with an acute infarction, and how they die if they have recovered from an acute infarction.

TABLE I. ACUTE MYOCARDIAL INFARCTION AT NECROPSY IN 133 CASES (1946-1950 Inclusive)

Apparent main feature in death		*Per Cent*
Heart failure (including shock)		52
Sudden death (arrhythmia?)		24
Rupture of heart		15
Thrombo-embolism		6
Systemic	3%	
Pulmonary	3%	
Miscellaneous		3

In Table I are the results of the work carried out with Dr. McQuay and Dr. Edwards in respect to the deaths occurring from acute infarction of the heart to indicate the therapeutic problems. One may see that there is a large number of individuals who have a persistent "coronary insufficiency" state, or a "coronary failure" manifested by angina following their acute infarction, but pathologically no further actual infarction or secondary coronary thrombosis has been found. A large number of the patients in this category die suddenly within a week or so of their major infarction. It is seen that heart failure is responsible for nearly 50 per cent of deaths if all types are combined; presenting predominantly as right heart failure in nearly one-fourth of the cases and as left heart failure, as recurrent pulmonary edema, in considerably less than that. Approximately 10 per cent died from shock which was initiated by, and persisted after, the acute infarction, and in this study 15 per cent died of rupture of the heart. Only 6 per cent died of thromboembolic disease. Implicit in this table are the therapeutic goals for the attempted control of the morbidity and mortality of acute myocardial infarction, namely the understanding and prevention of sudden death, of heart failure, shock, rupture, and thromboembolism. I believe that definite progress has been made in each of these problems, as discussed by Dr. Russek, but advance has been indeed limited.

Regarding anti-coagulant therapy, it is my practice to give anti-coagulants routinely to everyone admitted to the hospital in whom the diagnosis of an acute myocardial infarction is made. Most patients in the Rochester area are admitted to the hospital when this diagnosis is made. We are willing to give anti-coagulant therapy to 100 or more patients with the hope of preventing one death. (That is, if 20 patients were to die out of the 100, and 5 per cent of these were to die related to thromboembolism, then one, that is, 5 per cent of the 20, might be saved by anticoagulants).

I had the opportunity of looking over Dr. Russek's paper, and as a spring board for a discussion of differences, I refer to the sentence, "The risk of thromboembolism in such (low risk) cases may be equaled or exceeded by the dangers attending the use of anticoagulants." I believe that it is just in such low risk cases that the dangers of anticoagulants are minimal, its control most readily achieved, and the salvage possibilities of the patient most complete. Some evidence has been presented indicating that anticoagulants may increase the incidence of myocardial rupture, but this has not been established in our own experience. It may be pointed out that if anticoagulants do decrease the number of deaths in patients with acute myocardial infarction and if the number of ruptured hearts remained the same in the absolute scale, then the relative percentage of ruptures, as observed by the pathologist, should show a slight increase.

I believe that there is some danger from pericardial hemorrhage when a complicating pericarditis is present, but this is minimal.

I found that I was in no disagreement with Dr. Russek's discussion of what has been called the chair treatment of myocardial infarction. It is my belief that the perspective concerning this particular therapy has become distorted. The program of getting the patient out of bed for a short period of time each day involves perhaps only 5 to 10 per cent of the twenty-four-hour period. I think that the same goal may be achieved by allowing activity and postural change in a proper cardiac bed. Incidentally, I do not believe that it is particularly paradoxical to allow the patient to use the bedside commode and not go through the routine of getting him into a chair for short periods of time each day. As I have visited various hospitals and talked to patients who have been treated for acute myocardial infarction outside our own city, I do not see a great deal of difference in the way the majority of patients are treated. There are occasional ones, however, where to my mind an unnecessary period of strict bedrest has been enforced, and to have the individual *flat* in bed or to be fed by an attendant seems not only completely unnecessary but probably detrimental to his physical and psychic recovery.

TABLE II. CAUSE OF DEATH IN 250 CASES OF HEALED MYOCARDIAL INFARCTION

Etiology primarily cardiac origin	*Cases*	*Per Cent*
Congestive heart failure without acute myocardial infarction	44	17.6
Acute cardiac death ("sudden death") without congestive failure or acute infarction)	64	25.6
Deaths during episode of recurrent acute myocardial infarction	49	19.6
Total	157	62.8

If the individual survives an acute myocardial infarction, what is the future course? Dr. Achor has studied the hearts of individuals who have shown the scar of a healed myocardial infarction, and Table II

(Continued on Page 917)

Surgical Treatment of Arteriosclerosis

The Challenge of Arteriosclerosis to Surgeons

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THE challenge of arteriosclerosis, as revealed in any well considered analysis of the problem, cannot be surgical but must be medical. Certainly it is true if we accept this premise: the kind and the quantity of fat taken up from the diet by the body largely determines the intensity of the arteriosclerotic process. Therefore, a medical regimen that prophylactically manages the lipid uptake problem has the most to recommend it. Just as a vaccine is demonstrably superior to an antibiotic, so does medical prevention assume a greater role than mere surgical correction of existing and continuing disease. Although both disciplines will have important contributions to make in succeeding years, our hopes for future generations must inevitably abide largely with the former. The medical man, with an assist from the biochemist, must assume and cannot shirk from the prime obligation to establish an effective management for this problem. The magnitude of this issue, in terms of lives lost or burdened by crippling handicaps, is of the foremost order. Here, therefore, is *the* challenge: control this condition of such widespread influence that it more frequently than any other process shortens and damages the lives of adult Americans.

From poignant personal experiences I am quite aware of appetite's damaging betrayals. I am therefore convinced that an effective medical regimen must overcome the handicap of these recurring individual dietary indiscretions. Many and sympathetic discussions with those who are also overweight have established that viewpoint. Certainly, too, this objective of limiting one's fat consumption must be brought about without any very stringent regimentation of the dietary habits, —if it is to be adopted widely by all who need this help. Knowing about the disadvantages of an excessive caloric intake is no assurance that the person will forego such indulgences. Those in the ranks of the internists are no thinner than the individuals from any other specialty, at least not until we examine those hapless ones frightened by a train of symptoms which stems from a site of vascular insufficiency. That this widespread habit of overeating is more deep seated psychically than some easily maneuvered emotional quirk is also denied by the robust contour of our psychiatric associates. To summarize, there is a thin fraction spartan-minded about their diet or so naturally endowed with the gaunt habitus as to carry some protection from the degenerative effects of an excess fat intake. For the bulky remainder, until there becomes available a medical means of guarding our health in spite of one's overeating, the surgical challenges created by the arteriosclerotic remain.

The problems of arteriosclerosis exist close at hand in any general surgical practice. The frequency of arteriosclerosis is increasing; and the total number of cases is on the upgrade as well, from an enlarging percentage of our population that have lengthier lifetimes in which to acquire this affliction.

Surgeons' accomplishments in response to the arteriosclerosis challenge have, through the years, scarcely been brilliant or imaginative. They have long viewed the toes withering from vascular insufficiency only to respond by separating this unfortunate man from his leg. The relatively recent use of sympathectomy as a prophylactic measure, probably was the initial substantial benefit resulting from their thinking in terms of an extremity saving concept.

For those persons suffering from vascular insufficiency, better means are now available for providing relief—and without loss of limb. Under appropriate circumstances after an endarter-

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ectomy, or a grafting procedure which removes or shunts around a diseased area, considerable or even complete relief from the previously inadequate blood flow can be realized. However, one should not sing of this very loudly for it is but a modest triumph. Although temporary benefits can be brought to a large number of these people, the malady lingers on.

There is a considerable need for more accurate, more sensitive, means of detecting and evaluating this disease's extent. Our rate of progress in mastering this clinical problem seems sure to hinge upon finding a solution to that need.

Required too is superior vascular prosthetic equipment. Whether of artificial or natural origin, it seems reasonable to require that these devices should possess a durability, in years of good function, at least equivalent to the vascular endowment of man at birth. Degeneration, thrombogenicity, disruption, deterioration, and aneurysm formation involving and adjacent to the replacement currently crop up to limit the percentage of successes below the ideal. In connection with these above-cited problems peculiar to vascular anastomoses is the pressing need for an efficient, and preferably quick, method for uniting small vessels (2 mm. and under), and then, to be sure, have the result invariably function. Existing suture techniques in this area leave much to be desired and failure by thrombosis promptly or in the early postoperative period is disturbingly frequent. This deficiency in our knowledge looms as an especial disadvantage in contemplated dealings with direct anastomoses of systemic vessels to coronary arteries of limited initial diameter, further attenuated by arteriosclerosis.

Too, it is an inescapable conclusion that for some time to come vital organs will continue to be discovered as diseased only after they have been virtually destroyed by advanced stages of arteriosclerosis. Their replacement will be required if life is to be saved. The immunological problems of such homologous transplants appear far from insoluble even from this contemporary view of our medical future. And when we can predictably introduce homologous organs, having them survive functionally, surgery will indeed have come of age medically. Into the foreground of that bright vista projects again the critical need for reliable techniques of coupling small vessels.

The surgeon faces an arresting challenge and has an intriguing opportunity to devise means of controlling the total lipid uptake from the intestinal tract. It would appear that nature has designed an alimentary system which has proven to be unnecessarily efficient. Perhaps from an anthropological point of view we have not acquired an adequate compensatory hypertrophy of those neural centers regulating appetite. Such a neurophysiological approach to this problem should prove both exciting in its potential and rewarding in its results. In any event, since the bulk of our population now consumes excess amounts of fat beyond the actual quantity needed or even desirable for most of these individuals, catabolizing this overabundant, and ultimately hazardous, intake is impossible and hence incompletely done. From the continuing transport of, and contact with, these damaging residues arise the arteriosclerotic vascular difficulties.

Essentially the problem is to devise means that will overbalance the normal lipid metabolism in favor of the intestinal residue. The net barrier between gut and lymph or blood vessels must be upped a bit. Several possible means arise for countering this eventually disastrous state. One approach might be a reduction in the area available for intestinal absorption, as after an intestinal bypass procedure. Yet another plan might seek control of enzymatic or other chemical aids to fat absorption through a reduction in the external secretion of the pancreas or by external sequestration of bile salts from the intestine. The unique enterohepatic recircularization of the latter offers intriguing possibilities. Finally, we might modify lipid absorption through techniques designed to reduce the amount of fat reaching the blood via the major lymph channels. A limited accomplishment by any of these designs could slow down the crippling or fatal progress of arteriosclerosis. Actually, with the evidence already at hand from certain clinical observations, one is permitted the perhaps ingenuous prediction that if through one or a combination of these procedures, the dietary lipid uptake were stringently reduced, a definite, albeit limited, degree of reversal in existing arteriosclerotic processes would take place.

When one reflects for but a moment on the great loss in life induced by arteriosclerosis, it is anomalous that, proportionally, so little experimental work on this problem is currently under way in surgical laboratories.

Occlusive Arterial Disease

Management by Thromboendarterectomy

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CLINICAL investigations of the past five years have shown that the peripheral ischemic manifestations of arteriosclerosis may often be due to localized lesions in one of the major arteries. Willis has recently reviewed the evidence that the well-known localization of the early intimal lesions of arteriosclerosis to certain susceptible portions of the major arteries may be explained on an essentially mechanical basis.[1] It appears also that hemodynamic factors associated with the flow and coagulation of blood cause the subsequent arterial obstruction to become localized to only a segment of the arterial tree.[2]

Numerous reports of surgical experiences have indicated that restoration of blood flow through a segmentally occluded artery may now be successfully accomplished. Surgical efforts have been directed along two essentially parallel lines: (1) resection of the diseased arterial segment and replacement by arterial, venous, or synthetic grafts; and (2) resection of only the inner layers of the occluded arterial segment and reconstitution of a new artery using the preserved and relatively undiseased outer layers of the original artery.[2-21]

The relative merits of the two methods—resection with graft versus thromboendarterectomy, as the latter procedure is called—have not as yet been fully determined. Although the purpose of this paper is to present our experiences with thromboendarterectomy, it will be readily apparent that most of the factors contributing to the success or failure of this method also apply to any of the various techniques of resection with grafting. The primary limitations to either approach appear to be those imposed by the nature of the disease.

Between January, 1951, and July, 1955, at the University of California School of Medicine and the Veterans Administration Hospital in San Francisco, thromboendarterectomy was performed ninety-nine times in ninety-seven patients for the treatment of segmental arterial obstructions due to arteriosclerosis.* At the beginning of this period, thromboendarterectomy was a new operation and hence in an exploratory phase. Since that time, numerous modifications have been made, not only in the operative technique but also in devising more accurate diagnostic methods, in the selection of patients, and in postoperative care. A steady improvement in morbidity and mortality figures has resulted. It is believed that, owing to these improvements, a majority of the complications which occurred early in our series could now be avoided.

Full and accurate definition of the extent of arterial disease is of prime importance in the proper selection of suitable candidates for operation. In all cases, operation was performed for lesions involving a segment of the arterial tree between the upper abdominal aorta and the popliteal bifurcation. Thromboendarterectomy distal to the level of the popliteal artery has not proved to be technically feasible. Translumbar aortography, which affords roentgenographic demonstration of this entire area, has become the most useful method for arteriographic visualization. Details of the technique of aortography currently employed have been described in another publication.[22]

In patients shown by arteriography to have diffuse arterial disease and/or multiple segmental occlusions, the operative complications were increased and the circulatory improvement was less satisfactory. In selected cases, thromboendarterectomy of a high level segmental occlusion may be warranted even in the presence of a lower in-

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*Ten patients in this series are being reported through the courtesy of Louis G. Brizzolara, M.D., Chief of the Surgical Service, Veterans Administration Hospital, San Francisco, California. Dr. Brizzolara has used a technique of operation identical to that reported in this paper.

operable occlusion if, by this means, partial revitalization of a severely ischemic extremity can be accomplished.

Arteriograms may disclose stenosis of a major vessel, rather than complete obstruction. A stenotic lesion of arteriosclerosis, when it exists as the sole impediment to blood flow, is particularly amenable to endarterectomy, and inasmuch as such lesions are believed to be precursors of complete occlusion, they are a strong indication for operation.[22]

In general in our experience, the higher the level of occlusion, the more localized has been the disease and the more satisfactory the results from operation. The most favorable lesions have been those that involve the aorta and common iliac arteries. A combined lesion which obstructs the external iliac artery on one or both sides as well not only presents a greater surgical problem but also frequently indicates the presence of diffuse arteriosclerosis with spotty changes in the more distal vessels. The lower lesions causing occlusion of the superficial femoral artery have been least favorable for thromboendarterectomy. The experiences from this study have shown that femoral thromboses, though usually segmental, are commonly associated with lesions at other levels. Accordingly, the long-term value of operations at this level is lessened. Furthermore, the incidence of both immediate and late postoperative thromboses has been greater with operations at the level of the femoral artery than with operations at higher levels. Accurate preoperative assessment is based upon arteriographic demonstration of all the occluding and stenotic lesions and can rarely be made by physical examination alone.

One particular clinical finding has become important, however, in the selection of patients for operation. It has been disappointing to find that the more severe forms of peripheral ischemia were rarely associated with single segmental arterial obstruction. Patients with rest pain or gangrene were found in most cases to have multiple occlusions or other evidence of diffuse disease, and thromboendarterectomy was therefore seldom feasible. An exception has been the patient with a sudden solitary occlusion. In such a situation, sufficient collateral circulation has had no opportunity to develop, and advanced lesions and often gangrene may be precipitated. In the absence of other contraindications, arteriograms have been performed in all patients with severe ischemia in order to find the infrequent patient who may be benefited by operation.

TABLE I. THROMBOENDARTERECTOMY—SEGMENTAL LOCALIZATION. 99 OPERATIONS, 97 PATIENTS

Site of Obstructive Lesion	Thrombosis	Stenosis	Total
Aorta	2	2	4
Aorta and bilateral common iliac	29	11	40
Bilateral common iliac		6	6
Unilateral common iliac	15	7	22
Aorta, bilateral common iliac, unilateral external iliac	3	1	4
Unilateral common and external iliac	3		3
External iliac	3		3
External iliac and common femoral	2		2
Common femoral	2		2
Superficial femoral	12	1	13
Total	71	28	99

The technique of thromboendarterectomy currently employed has been described in detail in another publication.[23] The following résumé is a brief description of the method. Preliminary exploration determines the extent of gross intimal disease, particularly with reference to the lower level of sclerosis. After mobilization of the diseased segment, systemic heparinization to last one to two hours is induced. Clamps are then placed to isolate the occluded artery. The artery is opened by a continuous longitudinal incision. A subintimal cleavage plane is reached by a circumferential incision into the intima *below* the lower level of gross intimal thickening and beveled upwards to leave a smooth lower surface. By proximal dissection, the obstructing intima with the enclosed thrombus is peeled away from the outer media to a point above the proximal level of occlusion and is then excised. The residual media is then closed by a continuous suture and the clamps are released. In rare instances of suspected weakness of the media, fascia lata sleeve reinforcement is used. No anticoagulant drugs are given during or after operation, other than the single injection of heparin which is effective only during the period the obstructing clamps are in position and during a portion of the first hour of restored blood flow.

Table I indicates the level of the occluded or stenotic arterial segments upon which thromboendarterectomy was performed. Such a listing does not, however, describe the full extent of the operation. In most of the cases classified as unilateral iliac artery involvement, thickened intima was removed from all or a portion of the opposite iliac artery and the terminal aorta as well. Further, it was usually necessary to excise thickened

TABLE II. CLINICAL STATUS FOLLOWING THROMBOENDARTERECTOMY

	Aorto-iliac	Ilio-femoral	Total
Circulation restored	50	12	62
Circulation improved	16	4	20
Circulation unchanged	1	4	5
Circulation decreased	1	0	1
Amputation	0	5	5
Death	4	2	6
	72	27	99

intima where it extended distally into the proximal portion of the external iliac artery in order not to leave a projecting ledge distal to the endarterectomized zone. In cases of aortic thrombosis, the proximal level of obstruction ranged from a point just above the aortic bifurcation to as high as the level of the renal arteries. The preponderance of aorto-iliac operations in this series reflects the greater tendency for high level lesions to be segmentally localized rather than the natural incidence of all peripheral arteriosclerotic lesions. Early in the study, it became apparent that the failure rate for operations at the femoral artery level was prohibitively high. Accordingly, the latter half of the series is composed almost entirely of operations for lesions proximal to the femoral artery.

Table II indicates the clinical results from thromboendarterectomy in terms of evidence of circulatory improvement. Amplification is necessary to make the grouping meaningful.

By "circulation restored" is meant that there was clinical evidence of return of normal circulation, that a minimum of one pedal pulse was palpable in each extremity upon which operation had been performed, and that all symptoms of ischemia were relieved. (Anatomical variations are such that occasionally only one pedal pulse is normally palpable.) Successful cases in which an attempt was made to restore normal blood flow to only one extremity in a patient with bilateral disease are included in this category. Patients whose main channel was open but in whom one or more branches, i.e., the inferior mesenteric or hypogastric arteries, remained occluded, are included in this category.

By "circulation improved" is meant that there was evidence of improvement but not full restoration of peripheral blood flow. When a sympathectomy was performed at the time of thromboendarterectomy, the improvement must have been greater than would be expected from sympathectomy alone. Included in this group also are patients who were essentially asymptomatic postoperatively, but in whom neither pedal pulse was palpable. Fifteen of the patients in the improved group had occluded arterial segments distal to the operated segment prior to operation. There were three patients who developed distal thrombosis at the time of operation but who had improved peripheral circulation postoperatively even in the presence of this complication. In one patient with acute occlusion and a devitalized extremity, a locally successful thromboendarterectomy increased blood flow to the degree that amputation was successfully performed at a lower level than would normally have been considered necessary. For this reason, the patient was judged as improved. There were two patients with aorta and bilateral iliac operations who developed postoperative thrombosis of one common iliac artery with no improvement in the corresponding extremity. However since in both of these patients, patency of the aorta and the opposite common iliac artery caused restoration of normal flow to the other extremity, they are also included in the improved category.

"Circulation unimproved" implies that the signs and symptoms of peripheral ischemia were unchanged after operation. All five patients in this group developed local thrombosis at the operative area within an hour of operation.

Patients who are listed under "amputation" are those in whom the gangrene which necessitated subsequent amputation was a direct result of an unsuccessful thromboendarterectomy, or in whom there was no improvement in the level of amputation for pre-existing gangrene even though patency of the operated segment was maintained. Disruption of the suture line requiring arterial ligation in two patients and local thrombosis in two others were the factors precipitating gangrene in four of the patients in this group. In one, spreading infection from a gangrenous toe necessitated amputation.

For the purpose of operative evaluation only, we have included under the heading "deaths" those patients who were believed to have died as a direct result of operation. There were two deaths from hemorrhage—one at thirty-six days after operation from disruption of the aorta, and the other on the first postoperative day due to intraabdominal bleeding from an undiscovered source. Two patients died from renal failure on the seventh and ninth days respectively. One died from an unconfirmed coronary occlusion on the second postoperative day and one from pulmonary congestion on the third day. Five of the six patients who had died had regained normal peripheral circulation prior to death but are not so classified. Fatal cerebral vascular accidents developed in two elderly patients late in their postoperative courses (on the ninth and twentieth days respectively), but in view of their otherwise normal circulatory status, these patients are not listed in the operative death category.

TABLE III. ARTERIAL STATUS FOLLOWING THROMBOENDARTERECTOMY

	Aorto-iliac	Ilio-femoral	Total
Operated segment patent: —pedal arterial pulses palpable	54	15	69
Operated segment patent:			
—distal artery occlusion preoperatively	11	4	15
—distal artery thrombosis resulting from operation	3	1	4
Operated segment thrombosed:	4	7	11
	72	27	99

TABLE IV. OPERATIVE COMPLICATIONS

Complication	Postoperative Clinical Status					
	Circulation Improved	Circulation Unchanged	Circulation Decreased	Amputation	Death	Total
Local thrombosis:						
Aorto-iliac	2	1			1	4
Ilio-femoral		4		2	1	7
Local aneurysm formation:						
Aorto-iliac	1					1
Ilio-femoral						
Arterial disruption:						
Aorto-iliac					1	1
Ilio-femoral				2		2
Distal arterial thrombosis:						
Aorto-iliac	2					2
Ilio-femoral	1		1			2

The cases in which thromboendarterectomy was performed have been separated into two categories on the basis of anatomical position of the arterial segment. Patients listed in the "aorto-iliac" group were those in whom operation was confined to the aorta and common iliac arteries, whether or not distal lesions were present. Patients undergoing operations upon the external iliac or femoral arteries, and those who had aorto-iliac operations which included the external iliac or femoral vessels are listed in the "ilio-femoral" category.

In the aorto-iliac group, sixty-six patients (92 per cent) developed restored or improved peripheral circulation after thromboendarterectomy, compared to sixteen (59 per cent) in the ilio-femoral group. Operation was followed by decreased circulation or amputation in one (1.4 per cent) of the former group, and in five (18 per cent) of the latter.

Table III indicates the status of the major arteries following operation. Of the patients who had the aorto-iliac operation, local postoperative thrombosis occurred in four (5.5 per cent). In the ilio-femoral group, this complication occurred in seven (25 per cent). The higher incidence of local thrombosis in operations involving the external iliac or femoral arteries may be attributed to two factors. The arterial segments denuded of intima in operations upon the femoral or external iliac arteries are of greater length than in higher operations and the arterial caliber is decreased and usually more irregular, all of which are factors predisposing to thrombosis. Further, with the lower obstruction, it is usually technically difficult to create a smooth lumen at the lower end of the dissection owing to the extensive intimal thickening beyond the obstructed segment.

Apprehension on theoretical grounds has deterred many surgeons from using thromboendarterectomy as a method of restoring blood flow through thrombosed arterial segments. The potential complications from removing through long incisions all but the media of a major artery have caused many to direct their efforts toward the use of arterial or synthetic grafts as an alternative. The anticipated complications have been local thrombosis, local aneurysm formation, disruption of suture lines and distal thrombosis due to ischemia from the prolonged application of occluding clamps. The incidence and significance of these complications encountered in our series are tabulated in Table IV.

Local thrombosis in the operated segment occurred in eleven patients. (In all cases, thrombosis developed in the first hour after operation.) There are three possible mechanisms which may account for the production of local thrombosis. Animal experiments have shown that a thin layer of clotted blood lines the endarterectomized arterial wall as soon as blood flow is restored and eventually organizes to become the new intima.[24] The rapid flow of blood in the center of the artery is believed to be the factor restricting clotting to the slow-moving outer lamina of the moving column of blood. The small arterial caliber and the luminal irregularity that are encountered after femoral and most cases of external iliac thromboendarterectomy would tend to enhance the outer clot formation and are believed to be factors in the higher incidence of local thrombosis in the ilio-femoral group as compared to the aorto-iliac group. Unnecessary manipulation of a freshly operated artery, by disturbing the layered clot, could also contribute to thrombosis.

The experiences in this series have indicated that two other mechanisms of local arterial occlusion are probably more significant than this strictly local process. Slowing of blood through the new segment by occlusion or irregularity of the distal lumen has frequently been encountered. The commonest cause of thrombosis, however, appears to be that which arises from clot formation in the static column of blood immediately distal to the lower clamp during the time this clamp is in place. Unless prevented or mechanically removed, such a clot will act as a dam to blood flow and cause blood to clot in the proximal operated segment. It is now the practice to assure patency of the distal segment before the artery is closed by observing whether retrograde flow occurs upon release of the distal clamp. If such flow does not begin, the distal vessel is manually milked proximally to clear it of inlying clot. Clotted blood or intimal fragments may also accumulate above the proximal occluding clamp, causing later embolic occlusion at the first major bifurcation in the endarterectomized arterial segment. To clear the proximal segment, the upper clamp is now temporarily opened for a few seconds before final closure of the arteriotomy incision. The risk of thrombosis after thromboendarterectomy from one of these latter mechanisms is shared by those using the resection-graft technique and may explain the not uncommon early local thrombosis described in some of the homograft reports. Systemic heparinization during the time arterial clamps are in position tends to decrease this risk. The absence of postoperative thrombosis at any time other than during the first hour after operation indicates that anticoagulants are of no real value after this period.

Aneurysmal dilatation of the endarterectomized segment has apparently occurred in one patient in our series. The patient's local physician reported by letter that this complication developed asymptomatically two years after thromboendarterectomy for thrombosis of the abdominal aorta. In this case, reinforcing fascia lata was used at the time of operation. However, the patient has not been available for aortographic confirmation of this complication. It has not been the practice in our clinic to obtain routine aortograms for follow-up study, and it is possible that undiscovered arterial dilatation has occurred in other patients.

Arterial disruption adjacent to the suture line occurred in two patients in the ilio-femoral group and in one patient in the aorto-iliac category. In the latter patient, disruption of one iliac artery on the twenty-fifth postoperative day was successfully repaired by suture and local reinforcement with a fascia lata sleeve. However, this patient died on the thirty-sixth day from disruption of the aorta. The two patients with femoral disruption were those in whom the arterial wall was so friable at re-operation that ligation was necessary to control the bleeding, resulting in gangrene and amputation. Disruption occurred on the tenth and fifteenth days respectively. Early concern over this complication led us to advocate the use of reinforcing fascia lata sleeves whenever undue weakness of the media was suspected. This method was used in only fifteen patients, however, and it may be less important than originally believed.

Distal arterial thrombosis during the time of operation was surprisingly rare. A factor in the reduction of this hazard may be the increase in collateral arterial flow which occurs in the presence of chronic occlusion. Temporary systemic heparinization should further minimize this complication.

The development of thrombosis in or distal to the operative area as a result of operation is not so disastrous to the clinical status as had originally been supposed. There were fifteen patients who developed either local or distal arterial thrombosis. Two of this group died of unrelated causes. Of the remaining thirteen, peripheral circulation was improved in five, unchanged in five, and decreased in three. The decrease was to such a degree in two patients that amputation was required.

The predominant symptom for which thromboendarterectomy was performed was claudication. In all patients listed in Table II as "recovered," this symptom was completely relieved; in the "improved" group, it was lessened. Reference has already been made to the relative infrequency of operable segmental lesions in patients with manifestations of more advanced circulatory impairment. There were only eighteen patients with rest pain, gangrene, or ulceration. In thirteen of this group, arteriograms revealed extensive occlusive disease. In eleven, the operated segment was in the external iliac or femoral arteries; in two, there was distal inoperable occlusive disease in association with an operable aorto-iliac thrombosed segment. Sixteen of the eighteen patients with rest pain, gangrene, or ulceration were relieved of their ischemic manifestations following thromboendarterectomy. Two have subsequently died from cerebrovascular thrombosis, and two have had amputation performed for recurrent thrombosis in the affected extremity one year after thromboendarterectomy.

Important in the total assessment of any therapeutic procedure is the long-term evaluation. The longest follow-up period in our series now approaches five years. Of the eighty-two patients in the "recovered" and "improved" groups, seventy-five have been available for personal follow-up examination. Sixty-five are alive with no change in circulatory status of the operated extremity. The average postoperative period in this group is three years. Arterial thrombosis at or adjacent to the site of operation occurred in five patients (four were in the ilio-femoral group)—three at one year, one at three years, and one at four years after operation. Cerebrovascular thrombosis occurred in four patients—on the ninth postoperative day in one, on the twentieth day in one, two years postoperatively in one, and four years postoperatively in the fourth. The first two patients died as a result. Thrombosis occurred in arteries to another extremity in three patients. Occlusion of the coronary artery developed over two years after operation in five patients, three of whom died from this complication. One patient died from carcinoma of the kidney. A possible aneurysm has appeared in one.

The early postoperative circulatory result has been sustained in a higher percentage of patients who had aorto-iliac operation than in those who had the ilio-femoral operation. Of the original twenty-seven patients in the latter group, sixteen were improved postoperatively but only six were alive three years after operation without circulatory evidence of advancing disease in the operated extremity.

Summary

Thromboendarterectomy has been used ninety-nine times in ninety-seven patients in the treatment of segmental arterial occlusion. Seventy-two patients had obstructions in the aorta and common iliac arteries; twenty-seven had obstructions in the ilio-femoral vessels. Sixty-two patients were completely relieved of all ischemic manifestations of arterial obstruction, and twenty were significantly improved. The factors contributing to the success and failure of this operation are analyzed on the basis of selection of patients and operative technique.

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Discussion

DR. C. WALTON LILLEHEI: Arteriosclerosis is mankind's greatest killer at the present time. This has been emphasized repeatedly during this three-day symposium. Reference to the vital statistics for any recent year will emphasize this fact even more graphically. For example, in 1953 in the United States the number of deaths attributable to arteriosclerosis totaled more than twice those due to cancer, all accidents, and all infectious diseases combined. Despite the magnitude of this problem of arteriosclerosis and despite its complexities as to etiology as have been emphasized by many of the preceding speakers, there is a simple aspect to the problem also. Arteriosclerosis kills by mechanically occluding the blood flow to three or four vital areas of the body. These areas are: the brain, the heart, kidneys, and the lower extremities. Historically, surgery has always been at its best in dealing with mechanical obstructions. There are no more brilliant chapters in the history of medicine than the results of surgical relief of mechanical obstructions in the urinary tract, the biliary tract, and the intestinal tract. Thus, there exists in these mechanical obstructions in the blood vessels due to atherosclerosis a tremendous potential field for future development of surgical forms of management. I have said in the past and would like to repeat here again today my belief that if one were able to put the energies of all of the surgeons of the world to work exclusively on this problem at the present time, there would still be more work to be done than could be accomplished.

It was some of these thoughts that have occupied our attention during the past four years' time that we have utilized endarterectomy in this hospital. We have been particularly interested in: (1) the technical factors contributing to success or more pertinently to failure, (2) the long term results in patients with successful endarterectomies, (3) the application of these techniques to other organs such as the heart and the brain.

In regard to technical considerations that lead to success the following listed have been the most important in our experience: (1) At the time of surgery retrograde blood flow from the distal end of the opened artery is essential. (2) Some method of sewing down the distal intima seems necessary in many cases. Most successful in our experience has been cutting the vessel off completely and making a direct anastomosis end-to-end of the endarterectomized segment to the distal unendarterectomized but patent artery. (3) Where applicable, multiple incisions to remove the diseased intima rather than laying the entire length of the artery open are useful. (4) Sewing up the artery over a temporary splint such as a piece of appropriately sized plastic tubing is helpful in obtaining an even calibre in the endarterectomized segment. (5) Heparinizing the patient during the actual period of arterial occlusion but not in the postoperative convalescent period.

A few slides* will illustrate the clinical application of several of these principles. The first series of photographs illustrate an endarterectomy in progress from the iliac artery on the right side down to the popliteal artery. We are entirely in agreement with Dr. Wylie in his statement that by and large endarterectomies in this segment of the arterial tree over such a long length of occluded artery have been less successful. However, this particular man has done well and his postoperative arteriogram two months later is shown in this slide. He has been followed for two and one-half years now and has shown no evidence of recurrence of his obstruction, adding additional evidence to our belief that successful endarterectomies do stand up well over time.

The next series of slides are from a man who had an aortico-iliac endarterectomy. His postoperative aortogram is shown here for comparison with the preoperative picture. He too has had no evidence of recurrent obstruction in the two years since surgical removal of the thrombolic occlusion of this terminal aorta.

The next slides show the opened left common iliac artery of a patient who died eight months after a successful endarterectomy. This man returned to the hos-

*Slide presented cannot be reproduced here.

pital seven months after his endarterectomy because of a cerebral-vascular accident. He died one month later from this condition. At autopsy the opportunity was thus presented for studying this endarterectomized segment, and it was possible to see the very smooth lining that had developed to reline this endarterectomized segment. This lining undoubtedly comes from the blood cellular elements as well as endothelial cells of the capillaries in the remaining arterial wall which is of course viable.

As previously mentioned, we have been particularly interested in the possible application of these techniques to the coronary arteries. In this regard and illustrated in Figure 1 is a very favorable circumstance which has been established by past studies of coronary artery pathology. These studies have indicated that 75 per cent of the fatal or serious coronary occlusions occur in the first 5 centimeters of the left or right coronary arteries, and paradoxically the distal coronary arterial tree in many of these same patients is but little involved or even normal in its appearance. Dr. Karel Absolon, of our surgical staff, with the co-operation of Dr. James Dawson, department of pathology, has studied a number of the hearts in older patients and has routinely carried out coronary artery endarterectomies in the autopsy room in these fresh specimens to ascertain the feasibility of this procedure as well as to develop suitable surgical techniques. Here is a photograph of the coronary endarterectomy specimen from a sixty-year-old man. I would emphasize that these are not the coronary arteries themselves, but are the diseased and obstructing lining removed from this man's right and left main coronary arteries. Here is another similar specimen from a fifty-five-year-old man.

One has but to look at these specimens to know that were these obstructing sequestra removed successfully during life that these patients could not possibly have had other than an augmented coronary flow. Dr. Absolon has accumulated an immense amount of valuable information in these studies, and perhaps the most valuable point of all has been the realization from this work that the coronary arteries are large enough to work upon successfully by direct methods.

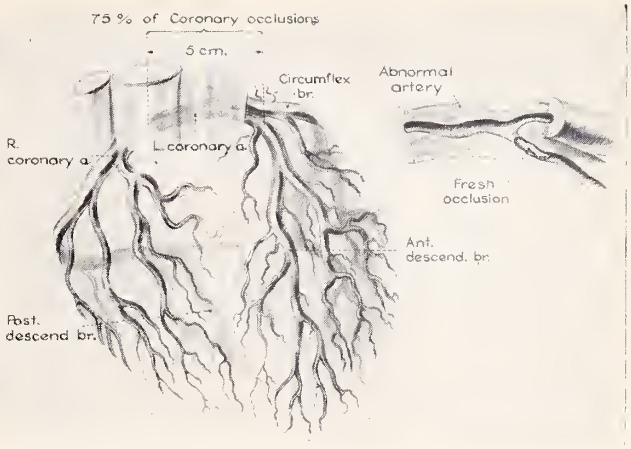

Fig. 1. Three-quarters of the serious or fatal coronary obstructions occur in the first two inches of the coronary arteries. This fact is an important circumstance prophesying future success in the removal of these obstructions by direct surgical methods.

With these facts established Dr. Absolon and Dr. Aust, of our resident staff, have in the experimental laboratory regularly been anastomosing *in vivo* the coronary arteries of dogs. Here is a photograph taken at operation of a direct end-to-end anastomosis in a dog between the internal mammary and the circumflex branch of the left coronary artery. These operations are feasible even in small dogs thus illustrating the possibility of human application. Here is another operative photograph of an end-to-end left subclavian to left circumflex coronary arterial anastomosis in a dog utilizing a synthetic segment of artery constructed of Ivalon to bridge the gap between these two arteries. In conclusion, here is a third example in the dog of an end-to-end anastomosis between the left carotid artery and the left circumflex coronary artery. These operations have indicated to us beyond any reasonable doubt the feasibility of direct surgical procedures upon the coronary arteries either by endarterectomy or by resection of the diseased segments and their replacement by suitable grafts. The main barrier at the moment to human application is the lack of good diagnostic methods to show the exact site and extent of the coronary artery pathology preoperatively. This can be solved, probably best by more precise angiocardiographic methods.

In conclusion I would like to emphasize that arteriosclerosis is one of the fields in which surgical advances are destined to contribute importantly to man's longevity. This is neither to overlook nor minimize the advances that can, must, and will come in other non-surgical forms of management or prevention of this disease. It is quite evident that the combined efforts of all fields of medicine, and in fact of anyone who has an idea, will be necessary before we will be in a position, as Oliver Wendell Holmes said, "to get ahold of nature and squeeze her until sweat breaks out over her brow and her sphincters are loosened."

Seventy-five per cent of all money raised in Minnesota for the Heart Fund remains in this state. Twenty-five per cent is sent to the American Heart Association, which in turn re-issues funds to all contributing states. American Heart Association has regularly returned more to Minnesota than it has received. Funds received by the Minnesota Heart Association are allocated almost exclusively to research projects within the state devoted to diseases of the heart and blood vessels. MHA funds helped underwrite the research which made possible the development of the cross-circulation heart surgery technique.

Occlusive Arterial Disease

Management by Use of Homografts

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THE traditional concept that occlusive arterial disease of the extremities is associated with generalized obstruction has long held sway. The evidence of its inaccuracy has also been available, but we have only recently begun to recognize that all arteriosclerotic peripheral disease is not alike and that it is necessary to evaluate again this problem which through its very familiarity has lulled us into believing that all of its clinical and anatomical features are known.

We must evolve a new dynamic concept of the pathology and physiology of atherosclerotic disease which can recognize the manifold manifestations of the arteriosclerotic process and to separate from each other the different pathological variants. At the same time we must be able to correlate all of these features to understand better the sequence of events which occur under these different circumstances. Already much progress has been made, and most of these advances in our recent knowledge have come as a result of observations made at the clinical level by exacting detail in the physical examination, by arteriography and by observations at the operating room table rather than by examination of pathological material at autopsy.

From the practical point of view occlusive arterial disease may be classified into three primary categories: (1) major arterial occlusion; (2) primarily small vessel disease; (3) a mixed form in which both large arterial occlusion and small vessel obliteration co-exist.

It is essential in the treatment of any patient to determine into which of these groups he belongs and to determine the exact site and degree of the occlusion. If there is evidence of arteriolar disease, its role in the production of signs or symptoms must be evaluated.

When the disease primarily involves the large vessels the area of complete obliteration tends to be segmental in a high percentage of cases. We have arbitrarily chosen to call all arteries proximal to the bifurcation of the popliteal artery large vessels because distal to this level direct surgical intervention is distinctly less successful at the present time than it is at higher levels, and in most cases the pathologic process which involves these vessels below this level also tends to involve the terminal arterial tree.

It is important to emphasize that high occlusions tend to be shorter than those at lower levels and that the majority of occlusions which begin above the level of the inguinal ligament end above the level of the femoral bifurcation. When major arterial obstruction is present in the absence of evidence of extensive small vessel disease, reconstruction of the major arterial trunk is the procedure of choice. Reconstruction of the major arterial obliterated segment may be extremely helpful even in the presence of small vessel disease, and the amount of benefit to be gained is dependent upon the extent of the small vessel obliteration and upon the amount of collateral which can be restored by reconstruction of the major trunk.

The natural history of the sequence of events which occur following a major segmental arterial occlusion have become more and more clear, and our current concept can be outlined briefly. It is interesting that most segmental occlusions tend to begin near or at a major bifurcation, implying that the hemodynamics of flow or anatomical structure may play a part. The process is usually one of gradual narrowing with subintimal thickening, ulceration and atheroma formation with progressive obliteration of the lumen. Ultimately, this ends in an episode of thrombosis whereby the remaining lumen is occluded. Usually, at the time that the total occlusion occurs the patient's symptoms markedly increase. This may be so sudden as to suggest embolic occlusion.

The pulsatile flow to the extremity is completely interrupted with the occlusive episode and the distal circulation then becomes completely collateral in type with a low systolic and a low pulse pressure. Proximal to the occlusion a blind

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segment now exists, and this undergoes a slow progressive thrombosis proximally to the point at which the next major branch arises. This process then usually becomes still slower and may even cease for a considerable period of time.

Distal to the occlusion there also continues a slow gradual thrombosis, again extending to the next major branch. If the vessel is still diseased at this level, the clot continues to accumulate on the distal thrombus until the next major collateral is obliterated. Relentlessly and slowly this progresses, picking off one by one the collateral blood supply to the distal extremity. The series of changes is so insidious that it may extend over a period of many years before the total arterial flow is so decreased that it jeopardizes the viability of the extremity.

On the other hand, if extensive disease is present in the major vessels, if there is co-existent small vessel disease to impair flow further or if poor collateral circulation is present, the thrombosis may extend extremely rapidly and lead to early gangrene. Similarly, the proximal extension of the thrombus, following the major arterial occlusion is variable and it may also gradually involve the proximal collateral circulation, or if the clot extends to the aorta it may then extend until it blocks the opposite iliac artery.

Thus, it is quite apparent that once a major segmental arterial occlusion has occurred a sequence of events is initiated which tends ultimately to lead to disability or destruction of the extremity, and this occurs principally as a result of the loss of the major forcible arterial pulsatile flow. Restoration of the major flow can greatly decrease the rate at which these and other changes of an obliterative nature will occur in the distal vessels which have varying amounts of disease.

For these reasons it is quite clear that arterial reconstruction with restoration of flow must be the treatment of choice in every situation in which it can be technically accomplished. These fundamental considerations are the basis of the rationale for the reconstructive approach to segmental arterial obstruction. Segmental obstructions may be long or short. Indeed, they may be treated when they extend from the level of the abdominal aorta to the knee, providing that a good patent channel is obtainable proximally and distally. The common sites for such chronic occlusions are: (1) in the abdominal aorta above the bifurcation; (2) at the bifurcation of the aorta; (3) in the common iliac artery, involving the hypogastric artery; (4) at the level of the bifurcation of the common femoral; (5) in the adductor canal above the knee.

Occlusions of the abdominal aorta below the renal arteries without extension into the aortic bifurcation are not frequent. Occlusions of the aortic bifurcation may arise as obstruction of one common iliac artery initially with progression to involve the entire bifurcation or it may occur simultaneously on both sides. It is quite usual to see a unilateral iliac occlusion which on exploration shows considerable involvement of the mouth of the iliac artery on the opposite side. Occlusion of the iliac artery at the level of its bifurcation with involvement of the hypogastric artery is probably the most common of the intra-abdominal major arterial obstructions. It is interesting to point out that the hypogastric artery is frequently among the earliest vessels to show evidence of obstruction and obliteration.

In contrast to this when the bifurcation of the common femoral artery is involved it is more usual to see the preservation of the integrity of the deep femoral artery, and indeed, even when it is occluded by thrombosis which involves the common femoral artery the thrombus frequently does not extend far into the profunda femoris.

Adductor canal occlusion is one of the more common varieties which occur below the inguinal ligament. This may be of considerable length. When the obstruction exists at this level, it is more common to see extensive distal obliteration than when occlusion occurs at a higher level.

These segmental obliterative lesions may be attacked by endarterectomy, by replacement with homologous arterial segments, by replacement with autogenous venous segments or by the use of plastic arterial prostheses. Each of these methods has certain inherent advantages and disadvantages which we well recognize, and the method to be used will depend in part upon the individual operator's experience with each of the procedures and the location of the area of obstruction. This discussion will be confined to the use of preserved arterial homografts since our experience with these have covered the longest period of time. It should be understood, however, that this does not imply that we feel preserved arterial homografts are always the best choice nor that they will necessarily continue to be the ideal method for arterial replacement in the future, for we are constantly seeking new and improved methods.

Homologous arterial segments can be preserved in a number of ways. Grafts may be taken aseptically, preserved by rapid freezing and storage at –70° C., or the rapid freezing process may be followed by dehydration and storage in a vacuum at room temperature. In the past several years it has not been necessary to take the grafts with aseptic precautions. They may be taken unsterilely and sterilized by irradiation with cathode ray emanations, beta-propio-lactone or ethylene oxide. We have adopted a simplified technique of securing the vessels without aseptic precautions at postmortem examination, sterilizing the grafts by immersion in ethylene oxide for 15 minutes, followed by rapid freezing, dehydration and storage in a high vacuum. The grafts are reconstituted at the time of use by immersion in saline solution. This method has given highly satisfactory results. It has been used in several hundred experimental grafts and in a very large number of clinical cases without evidence of graft failure due to methods of preservation.

Homografts preserved in this way have the advantage of ready availability and ease of storage at room temperature. Sealing in vacuum is helpful since one can determine immediately whether or not the seal of the tube has been broken. This can be accomplished by testing the vacuum with a high frequency spark coil. If the vacuum has been lost it must be presumed that the seal is no longer intact, and that the graft is therefore potentially contaminated and should be discarded. When such grafts are sealed in glass, they may be stored for periods of approximately two years following their processing without danger. It is of importance to realize however that all processes of dehydration are not the same, so that one must adopt a standard technique with rigid controls before one can expect to obtain uniform results. When the lyophilized grafts are reconstituted they regain their normal pliability and elasticity. This constitutes one of the major advantages of homografts. When one must replace a branching arterial segment such as an aortic bifurcation in which it is necessary to reconstruct the hypogastric and external iliac artery on each side, the adaptability of homologous grafts allows one a great deal more freedom and ease of execution of the procedure than do some other forms of prostheses.

In any consideration of obliterative arterial disease involving the circulation to the lower extremities it is worthwhile to separate those lesions which occur in the vessels which lie within the abdomen and those which lie in the extremities themselves for the more proximal lesions are distinctly more favorable. Whether this is entirely related to the size of the vessels and the more difficult technical problems associated with the anastomosis of small arteries, whether there is a fundamental difference in the arteriosclerotic process in the different areas, whether it is a matter of the length of the graft necessary to replace the diseased segment or a combination of these factors is difficult to distinguish.

It is quite clear, however, that those lesions which lie in the aorta or in the iliac vessels alone and do not extend beyond the inguinal ligament have almost uniformly satisfactory results when the diseased segment has been replaced with homografts with end-to-end anastomoses. In contrast to this the results with replacement of vessels in the leg itself have been less reliable. These results have been changing constantly as more experience has been gained. The number of successful cases has markedly increased until at the present time with well selected cases one can expect approximately 80 per cent of the results to be good after two years of follow-up. The use of end-to-side anastomoses in selected instances has been helpful, particularly when one is dealing with the smaller vessels.

It would appear that many of the early poor results with removal of diseased arterial segments were due to excessively conservative resections. At first it was believed desirable to resect only a minimum of the artery and this in general was confined to the area of occlusion. This has more recently given way to the more radical concept in which it has been felt desirable to re-establish circulation at a point in which the vessel becomes relatively good and to remove as much of the diseased vessel as may be necessary to secure an arterial wall of as good quality as can be obtained above and below the site of the occlusion. It is also desirable to restore flow distal to those parts of the vessel which are partly obstructed. This may be accomplished in some cases by excision of all of this area or in other cases by the use of end-to-side anastomoses which by-pass the area of obstruction.

A brief review of the common sites of occlusion together with the methods which are most readily applicable using preserved arterial homografts may summarize the current situation. Occlusion of the abdominal aorta without involvement of the aortic bifurcation is one of the less common obliterative lesions, but one will occasionally find this type of involvement without involvement of the aortic bifurcation, and the simple tubular aortic homograft may be used very satisfactorily to correct the situation. The arotic bifurcation thrombosis usually involves the proximal aortic segment for a considerable distance and may extend up as high as the renal arteries. The iliac involvement is variable and may not be the same on both sides. In a large number of individuals it is possible to use a bifurcation aortic graft for the single anastomosis proximally and one anastomosis in each common iliac artery just above its bifurcation. Very frequently however, on one side or the other the bifurcation of the iliac artery may be involved and an anastomosis must be accomplished between the external iliac artery and the hypogastric artery with the graft.

Occlusions of the common iliac artery are occasionally extremely short and a direct anastomosis of the segments may be accomplished after resection of the obstruction. Usually however, the area of thrombosis or of partial obstruction extends back to the aorta. If the opposite side is completely free of obstruction unilateral reconstruction is permissible. If there is evidence of serious disease on the other side however, it is optimal to use an aortic bifurcation graft with reconstruction of both sides. When unilateral reconstruction is attempted it is undesirable to attempt to use the stump of the iliac vessel if the arterial wall is of poor quality. In such cases an end-to-side anastomosis between the graft and aorta at a point above the aortic bifurcation where the aorta is less involved would appear to be the preferable procedure. The distal anastomosis can be made either end-to-end or end-to-side as the circumstances indicate.

When the obstruction extends distally beyond the pelvis and into the leg the proximal reconstructive procedures are the same as for those of the major vessels and the distal anastomosis can be made end-to-side with preservation of the major collateral in the region of the inguinal ligament. When obstruction exists between the bifurcation of the femoral artery and the bifurcation of the popliteal artery, the various procedures are helpful. If the proximal obstruction is readily ascertainable just below the bifurcation of the common femoral artery, it is frequently simple and highly satisfactory to do an end-to-end anastomosis proximally. One may then isolate the popliteal artery or the femoral artery just above its entrance into the popliteal space, and if it is patent at this level, an end-to-side anastomosis can be accomplished without isolation of the intervening vessel. This is particularly helpful if large collaterals arise at the point at which the obstruction ends or if the vessel is quite small. End-to-end anastomoses have also been highly satisfactory in our hands and it would appear to be usually quite possible to obtain a distal stump of patent vessel without sacrificing collateral. This is particularly true if one bevels the anastomosis and thus avoids the circumferential suture in a single plane.

This brief résumé of some of the problems of obliterative arteriosclerotic disease of the arteries supplying the lower extremities brings into sharp focus the dynamic concept of reconstruction of major arterial continuity. It offers the possibility of restoration of adequate circulation and retardation of the progress of the disease in a very large group of patients.

When the obstruction lies above the inguinal ligament one can expect a successful outcome with long-term good results in approximately 90 per cent of the patients. When the obstruction lies within the leg itself and can be successfully treated by restoration of pulsatile arterial flow, good results can be expected in 70 to 80 per cent of the patients. The risk for the patient in such procedures is not great and is primarily related to the risk of concomitant arteriosclerosis involving other organs in relation to a major operative procedure. In our series of all types of operations for occlusive arterial disease, including the aorta and extremities the mortality rates are under 2 per cent. On the basis of present experience we can anticipate the benefits which are currently available in selected instances will be extended to larger groups of patients, and until the great problem of prevention of arteriosclerosis can be solved we will be able to restore more and more of these individuals to a useful life.

Healing and Fate of Arterial Homografts

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RATHER than discuss Dr. Hufnagel's excellent presentation, I shall consider the general topic of the healing and fate of arterial homografts.* My comments will be based upon studies of approximately 1,000 experimental vascular grafts carried out at the University of Washington School of Medicine and Walter Reed Army Medical Center over the course of the past five years.

From these studies has come the realization that to understand the healing of arterial grafts is to understand their fate. We have gained an understanding of the healing of arterial homografts which has given sufficient insight into their fate to be worthy of consideration.

So rarely do the cells of a homograft survive that for practical purposes our consideration may be directed entirely towards the fiber framework of the graft. The fate of this framework determines the fate of the graft. After implantation, living cells die. The basic histologic fate of homografts is independent of their state of viability at the time of implantation. The fibroblasts of adjacent host tissues proliferate and invade the homograft wall to varying depths and spread from the anastomotic site along its inner surface for short distances. The anastomotic junction is healed by the ingrowth of host fibroblasts from the aortic ends and from the surrounding tissues. Fibrous tissue from the anastomotic junction grows out to cover the inner surface of the adjacent 1 to 2 cm. of graft and 2 to 3 mm. of aorta. The extent to which the intima of the host aorta contributes to this process has not been determined. Some fibrillar elements taking the elastic tissue stain extend out from the aortic intima for 1 to 2 mm. along the inner surface of the fibrous tissue at the anastomotic site.

In most instances, the inner portion of the homograft fiber framework becomes condensed to form an acellular, relatively inert, mass of foreign homologous protein which is impervious to the ingrowth of host tissue and remains so for periods of at least a year. Thus, only at and adjacent to the anastomoses (1 to 2 cm.) and to those sites where the fiber framework happens to remain pervious to the ingrowth of host tissue, or becomes so subsequently, does a layer of host tissue form along the inner surface of the homograft fiber framework. Accordingly, most homografts longer than 3 to 4 cm. are devoid centrally of an inner lining of host tissue. The condensed fiber framework of the homograft serves as an effective conduit for the passage of blood. It does not promote thrombosis. Apart from lack of perviousness, the homograft fiber framework is comparable to the plastic fiber frameworks currently being employed. However, the homograft fiber framework differs from the plastic fiber frameworks in that it is fabricated from better material in a superior manner. Because of this, the homograft is elastic, dependable, and durable. Plastic fiber frameworks have little elasticity and are not as adaptable as their homograft counterparts. The plastic fiber frameworks are best suited for bridging straight defects of large arteries where branches, angulation, and small caliber are not considerations.

In the early period following implantation, the strength of the graft resides in its homograft fiber framework. Subsequently, the autogenous fibrous tissue reaction about the homograft is sufficient of itself to serve as a suitable conduit. It is difficult to state how long the graft is dependent upon the integrity of the homograft fiber framework for its strength. As a generalization, three months is suggested. If the homograft fiber framework loses its continuity before the host tissues have been able to form an autogenous tube about it, rupture with hemorrhage occurs. If the continuity of the homograft fiber framework is lost before the host tissues have formed a *stable* autogenous tube about this framework, aneurysm formation with its attendant complications results. However, if the homograft

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fiber framework continuity persists until the host tissues have formed a stable autogenous tube about this framework, a satisfactory conduit remains whether the homograft fiber framework subsequently loses its continuity or not.

Apart from rupture or aneurysm formation, the complications of thrombosis and degenerative change must be considered. Early thrombosis is due in the main to turbulent blood flow at the anastomotic site, which circumstance is largely the result of technical error or improper size of graft. Delayed thrombosis is largely related to the development of calcific changes in the graft wall. These changes occur in the inner condensed acellular portion of the homograft fiber framework. The reason for the development of these changes is obscure, but a probable cause is deficient nutrition in the vicinity of foreign protein. Calcific changes in the homograft wall of themselves do not impair the ability of the graft to function as a conduit. However that such changes favor the development of delayed thrombosis or aneurysm formation seems certain. Longer clinical observation is required to ascertain the influence of calcific change upon ultimate graft fate.

As regards the use of homografts in the arteriosclerotic patient, these conduits with their attendant dangers are less liable to complications than is the rest of the arterial system of the patient. Accordingly, one may indorse their use in the arteriosclerotic patient with few reservations.

The results currently being obtained with plastic grafts are highly encouraging. However, for general use, the proper employment of appropriate homografts is still the recommended means for bridging arterial defects.

DISCUSSION—ARTERIOSCLEROSIS OF THE EXTREMITIES

(Continued from Page 901)

is a summary of his data. Sixty-two per cent of individuals who had healed myocardial infarction died of heart disease. Thus two-thirds died of conditions primarily related to their coronary disease. Approximately one-fifth died of congestive heart failure without further infarction, another one-fifth died of recurrent infarction, and one-fourth died a sudden death without failure, acute infarction or acute coronary occlusion. Again to focus our attention upon the therapeutic challenges, that of people dying *suddenly* is paramount, and assuming that this might be caused by ventricular fibrillation, then this is an avenue of investigation that must be further encouraged.

The therapeutic challenge of coronary disease I have thought might be epitomized by the titles of Churchill's volumes. This is a type of war, and the male sex is involved to a great extent. The *Gathering Storm* is the development of the atherosclerosis, and this may be present even when persons are in *Their Finest Hour*. The *Great Alliance* to help is the inherent capacity of the coronary circulation to develop collaterals, the subject's acceptance of restricted activities and the little help that the doctor may give. The *Hinge of Fate* is related to those factors that eventually should be elucidated, why some individuals live an active life with their disease for twenty years plus and others do not. The *Closing of the Ring* is the development of sudden coronary occlusions, and *Triumph and Tragedy* are the outcome of the acute destructive processes within the war-stricken myocardium.

Henry I. Russek: Everyone would agree with Dr. Burchell that the saving of even one patient out of every 100 stricken with acute myocardial infarction is deserving of every therapeutic endeavor. I would like to point out, however, that while I strongly favor the use of anticoagulants in severe (poor risk) cases, the statistics presented by Dr. Burchell provide the weakest support for this form of treatment that I have thus far encountered. According to these figures, only one patient out of every 100 unselected cases of acute coronary occlusion dies of a thromboembolic complication. On the other hand, careful analysis has shown that anticoagulant therapy, even in the hands of the most competent investigators, is responsible for 1.7 deaths from hemorrhage in every 100 patients treated. In weighing these respective risks, it must also be realized that present-day therapy with anticoagulants does not achieve infallible prophylaxis. Consequently, while Dr. Burchell's objective is commendable, should we not ask ourselves whether or not we are merely substituting one mode of death for another? The answer can only be found by analyzing cases within a framework of prognostic categories based on clinical signs and symptoms.

Unless so considered, crude statistics may mask important facts. Thus, in patients with shock, congestive heart failure, arrhythmias and other poor prognostic signs, the incidence of fatal thromboembolism may be 5, 10 or more per 100 cases. Certainly, in such instances, the salvage possibilities from the administration of anticoagulants justifies the risk inherent in their use. In milder cases, on the other hand, thromboembolic deaths occur so infrequently that they are encountered in only a small fraction of 1 per cent of patients in this category. The use of anticoagulant therapy in this low-risk group therefore appears to be unwarranted. Certainly, all the evidence to date can lead to no other conclusion.

Use of Greater Saphenous Vein Autographs in the Reconstruction of Segmental Arterial Occlusions

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WITH much of the current interest in arterial reconstruction being centered upon arterial homografts, plastic prostheses, thromboendarterectomy, and other similar techniques, it is felt that some mention should be made of techniques utilizing vein autographs segments. Nabatoff,[1] Julian[2], Sako,[3] and others have shown that visceral veins, when placed as a graft to bridge a defect in an artery, have a tendency to dialate into aneurysmal formations. However, the greater saphenous vein is of adequate size, has a muscular media and, as demonstrated by Julian,[2] has the strength to withstand arterial pressures. It is fortuitous that the greater saphenous vein is almost universally avilable for the sacrifice of its normal function for other purposes.

TABLE I. SAPHENOUS VEIN AUTOGRAPHS

Arterial Segment	Result
1. Abdominal aneurysm	Failure
2. Femoral aneurysm	Failure
3. Abdominal aneurysm with iliac artery occlusion	Excellent
4. External iliac	Excellent
5. Superficial femoral stenosis	Thrombosed vein
6. Superficial femoral occlusion	Excellent
7. Superficial femoral occlusion	Excellent
8. Superficial femoral occlusion	Excellent

It is felt that the techniques which utilize the replacement of an arterial segment which is occluded, by "in lying" methods, present an undue hazard to the extremity when used in distal arterial beds. Small collateral vessels must be sacrificed in the exposure and resection of the occluded arterial segment; thus, if the graft should fail, the extremity is often sacrificed. Linton[4] has described a technique whereby occluded arterial segments may be by-passed, by an end-to-side anastomosis of an arterial homograft above and below the occluded portion of the artery, thus obviating this difficulty. In our clinic, the possession of a store of arterial homografts has proven extremely difficult, and as such, we have preferred to evaluate this by-pass procedure by using the ready supply of greater saphenous vein autografts.

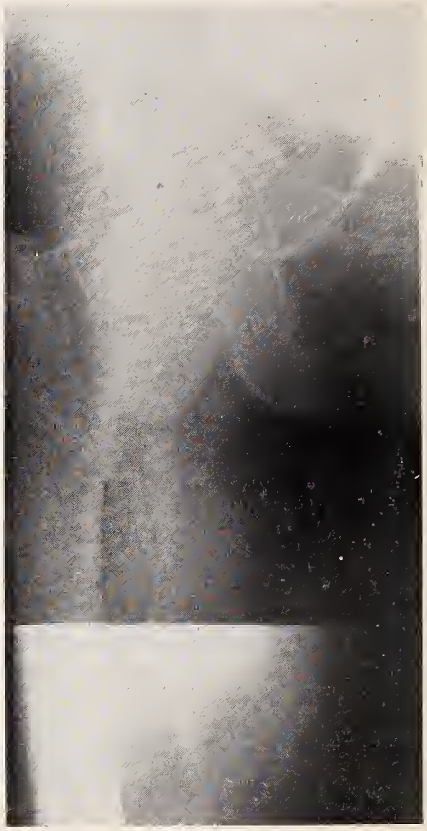

Fig. 1. Femoral angiogram of a femoral aneurysm showing contrast media filling only the distal end of a vein graft around the aneurysm. The proximal end of the graft was compressed by enlargement of the aneurysm. Case 2, Table I.

In the University of Minnesota clinics, patients with symptoms of intermittent claudication, with absent peripheral pulses, are evaluated with arteriographic studies. If segmental occlusive dis-

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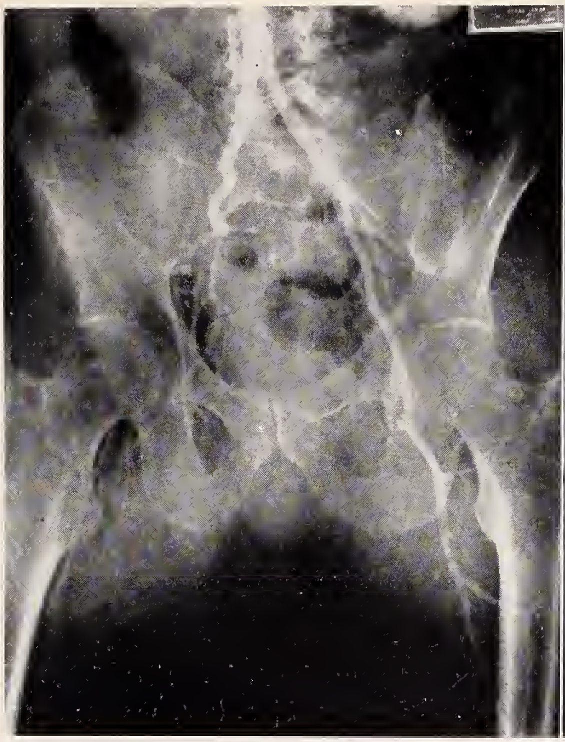

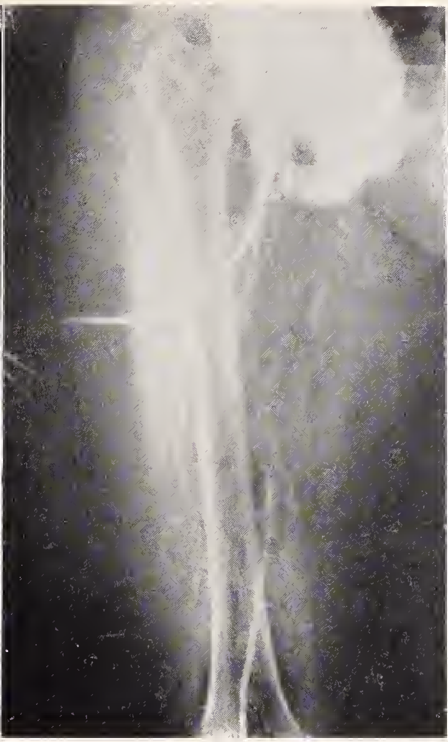

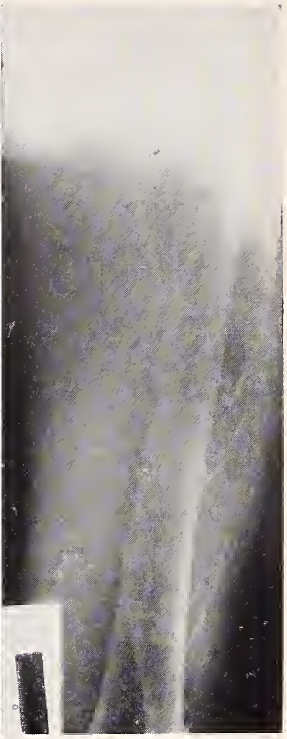

Fig. 2. *(left)* Aortogram showing segmental occlusion of the external iliac artery, with good filling of the femoral artery below the inguinal ligament. Case 4, Table I.

Fig. 3. *(center)* Femoral angiogram showing occlusion of the superficial femoral artery in Hunters canal. Case 6, Table I.

Fig. 4. *(right)* Femoral angiogram showing occlusion of the superficial femoral artery with good filling below Hunter's canal. Case 7, Table I.

ease is found above the popliteal artery, the patient is considered a candidate for an arterial reconstruction.

Technique

The patient is placed upon the operating table in the supine position, and a small sand bag inserted under the opposite hip when the popliteal space is to be entered. Using the technique described by McPheeter,[5] the saphenofemoral junction of the greater saphenous vein is dissected and all branches ligated and severed. The vein is then removed, using an external Mayo vein stripper, and all branches ligated as they are encountered. Fortunately, there are few branches to this vein above the knee, and the segment of vein from the groin to the knee is usually of sufficient length. The vein is then washed with saline to remove all blood and clots.

If the occluded arterial segment is intra-abdominal, a mid-line abdominal incision is satisfactory. With external iliac artery occlusion, we prefer a retroperitoneal approach. In occlusions of the superficial femoral artery the original incision at the sapheno-femoral junction is adequate to expose the femoral artery, and by rotating the table slightly, a curved incision at the medial edge of the popliteal space will expose the popliteal artery distal to Hunter's canal.

Heparin, 50 mg. is injected into the artery at its proximal exposure, just prior to placing the occlusive clamps during the arteriotomy. An eliptical arteriotomy is made, and with a beveled terminal end to the vein graft, an end to side anastomosis is carried out, using 5-0 arterial suture. The distal end of the vein must be anastomosed to the proximal arteriotomy site, and the proximal end of the vein anastomosed to the distal arteriotomy, to prevent the interference of blood passage through the graft by the venous valves.

If the graft is to by-pass the superficial femoral artery, we have found it advantageous to replace, but reverse, the vein in its original site, to the knee, then anterior and beneath the vastus medialus muscle, into the popliteal space.

It is further felt to be advantageous to by-pass a segment of artery from the junction of a large vessel above the occlusion, to a similar junction below the lesion. Thus, for external iliac artery

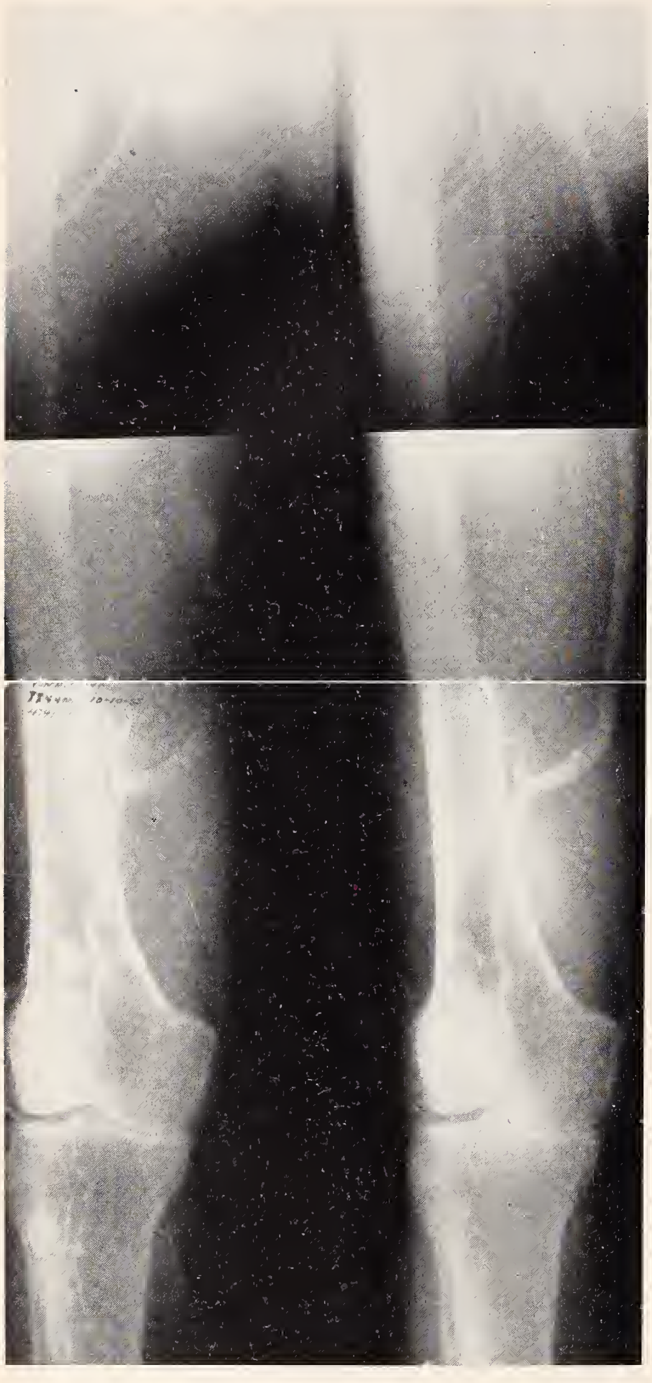

Fig. 5. Femoral angiogram, taken three months after insertion of the graft, shows filling of the vein graft and distal vascular bed, Case 7, Table I. Note the dilatation of the graft in the area of the inverted valves.

occlusion, we graft from the origin of the hypogastric and the external iliac arteries, to the origin of the deep and superficial femoral arteries. At these spots, the lumen of the artery is at its greatest, and the anastomosis may be easily carried out. Unfortunately, in the popliteal space, there are no such large branches distal to Hunter's canal.

Results

Eight procedures utilizing the above technique have been carried out (Table I). Five of these were occlusive disease, and three were attempts to bridge around aneurysms prior to their resection or with occlusion distal to the aneurysm. In one instance, the vein was found to be thrombosed when it was removed, and thus proved unsatisfactory for a graft. In the remaining seven instances, the grafts functioned well at the time of surgery.

In two of the three instances in which the graft was used to bridge around aneurysms, the procedure was unsatisfactory. In one instance, the aneurysm enlarged, to occlude the graft (Fig. 1). In the second, the graft was exposed at a second operation in an attempt to remove the lesion, and the proximal anastomosis was found occluded due to extension of clot from within the aneurysm. In the third instance the graft functioned satisfactorily for two years with complete relief of claudication during this period until the patient expired from other causes.

In the four instances where the grafts were bridged about blocked segments of artery, and were satisfactory at the time of surgery, they have remained functioning to the present time, although the follow-up period has been short (Figs. 2, 3 and 4).

Patients have been evaluated with pre-operative and post-operative plethysmographic studies using the technique of Windsor.[6] In all instances, there has been a marked change in the digital pulse volume, greatly exceeding that expected by a sympathectomy. In no instance in this series, was a sympathectomy done concomitantly with the vein graft. In all these patients, there has been relief of claudication, and in the one patient with rest pain, there has been complete relief of symptoms. In another instance, the patient had suffered from a large chronic ischemic ulcer on the anterior tibial area, for three years prior to surgery, which was refractive to conservative therapy. During the ten-day postoperative hospital stay the ulcer epithelized without special therapy, and has remained healed to this time.

Discussion

It is felt that this by-pass type of arterial reconstruction is of value for several reasons.

1. There is no sacrifice of small collateral ves-

sels as in methods requiring the removal of the diseased arterial segment.

2. In the event of failure of the graft by early or late thrombosis, the preoperative blood supply to the limb has not been impaired.

3. The operative procedure may be done through a small incision, with little blood loss, safely, even in a poor risk patient.

The simplicity of the procedure described is further exemplified since there is no need for a store of arterial homografts. Furthermore, there is no need for special instruments or procedures to make and develop plastic arterial prosthesis. The pliability of the vein wall makes it available to place across movable joints and for use in the extremity. In reality, the above technique is not an artificial reconstruction of an occluded artery, but is instead, the surgeon's attempt to increase the efficiency of nature's method of healing this lesion, by the development of a large collateral channel around the occlusion.

Summary

A technique of reconstruction of the arterial bed in segmental occlusive vascular disease, by the use of a by-pass saphenous vein autograft, has been described. The results of early follow-up in eight patients is presented. It is suggested that this technique may be used without some of the hazard of other procedures, and without the necessity of an arterial homograft bank or the use of non-vital prostheses.

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Although the Minnesota Heart Association places its greatest emphasis by far on heart disease research, it is also concerned with education and community service. Currently, the chief community service project is the Heart Council, to which MHA turns part of its attention when the annual Heart Fund drive is over.

MHA is attempting to organize permanent Heart Councils in most of Minnesota's major cities. Although the program is very young, several Heart Councils are already functioning within the state.

Composed of lay and professional persons interested in heart disease problems, the Heart Council's basic function is to inform the public about heart disease. This information program is not built upon creating fear in the minds of persons—but rather on informing them of the new hope for afflicted hearts.

Methods of informing the public are as varied as the ingenuity of individual Heart Council members. Most common are films, speakers, the press, radio, and television. Other popular means are Health Days, state and county fairs, and displays.

Twelve councils now exist in Minnesota, and a thirteenth is being formed. Minnesota Heart Association's ultimate goal is the establishment of Heart Councils in each and every Minnesota community. Funds for the activities of the Heart Councils are included in the budget of MHA. Minor expenses necessary to carrying out a Heart Council program are underwritten by the state organization. It is an investment well worth making, in the opinion of MHA's Board of Directors.

It is not intended that the Heart Council be used as a tool in the annual fund-raising campaign of the Minnesota Heart Association. Although its activities will benefit the Heart Fund drive because of increased public awareness, the council's main job is still educational. Through education will come increased understanding of the problem of heart disease.

Surgical Treatment of Aneurysms of Arteriosclerotic Origin

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ARTERIOSCLEROSIS is so frequently associated with dilatation and elongation that it is not surprising that discrete localized aneurysms not infrequently occur. More surprising and difficult to explain is the localization of these discrete aneurysms to specific regions. Aneurysms of arteriosclerotic origin may occur in any region of the body, the arm, head, thorax, lower extremity, or abdomen, but of peripheral vessels the popliteal is most commonly affected, and of the aorta itself, the region of the terminal aorta. Aneurysms of the peripheral arterial system present somewhat different problems from those of the aorta itself, and these two types of lesions will be considered separately. Cerebral aneurysms constitute a totally different condition and will not be considered.

Peripheral Aneurysms

Peripheral arteriosclerotic aneurysms may occur in almost any vessel, but since they are most common in the popliteal artery and since treatment of popliteal aneurysm is representative, this lesion will be specifically considered.

Little is known why the popliteal fossa is the site of predilection for arteriosclerotic aneurysms, though the most commonly given explanation is that of repeated flexion. Once dilatation of the artery begins, physical factors are such as to encourage enlargement of the aneurysm. Stress on the arterial wall is directly proportional to the diameter of the vessel so that as the diameter doubles the tendency for rupture doubles, and the stage is set for progressive enlargement. Males are more commonly affected, and frequently the aneurysm is bilateral. Symptoms are of two types. The mass in the popliteal fossa may cause pain and discomfort in this region, or ischemia of the lower leg may occur as manifested by ulceration, ischemic neuritis or intermittent claudication with pain in the calf. In some instances acute disability may arise from arterial occlusion.

In the past it has not been considered a particularly dangerous lesion and most surgeons have felt the danger of operation to be greater than the risk of the aneurysm itself. Hence in the absence of symptoms treatment was often not advised. A review of 100 cases by Gifford, Hines, and Janes at the Mayo Clinic, however, showed that untreated popliteal aneurysms are associated with a high incidence of complications (sixty-two of their 100 cases) in the form of arterial occlusion, rupture, or pressure on veins and nerves. Complications in 23.4 per cent of the aneurysms treated without operation led to amputation of the limb which harbored them. On the other hand, only 7.7 per cent, or two of twenty-six patients, treated operatively required amputation. Both of these aneurysms were in the early postoperative period in patients whose extremities were in peril before operation.

This observation led these authors to advise an aggressive surgical approach to popliteal aneurysms, and their preferred method of treatment at that time was sympathectomy and extirpation of the aneurysm. This can usually be done with impunity although increase in the signs of peripheral ischemia may be seen. Because of this we have recently preferred to treat this as well as aneurysms of most major vessels by excision and restoration of arterial continuity by use of a graft (Fig. 1). Our first such lesion was treated by autogenous vein graft, taken from the deep popliteal vein, after it was apparent that the saphenous would not be adequate.

More recently we have had available frozen-dried arterial homografts and have found these to be quite satisfactory in three instances for popliteal aneurysm. The fate of homografts in the peripheral arterial system does not appear

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Presented in the Symposium on Arteriosclerosis, University of Minnesota, Minneapolis, September 8, 1955.

to be as bright as in the larger vessels, the aorta and the iliac, but if occlusion occurs it is more apt to be of slow progression and concurrent with the development of collateral circulation. This then seems to be a decided advantage over simple abrupt ligation of the popliteal artery. Our three patients with four popliteal homografts continue to do satisfactorily after periods of twenty-five, fifteen and eleven months since operation. Each of these patients had open aneurysms and was operated upon in the absence of significant symptoms. When complete thrombosis has occurred I would perform an operation only for symptoms. If there is no evidence of distal ischemia, excision alone is indicated. When ischemia is present a graft should be used if possible and the lesion treated as any other such arterial occlusion.

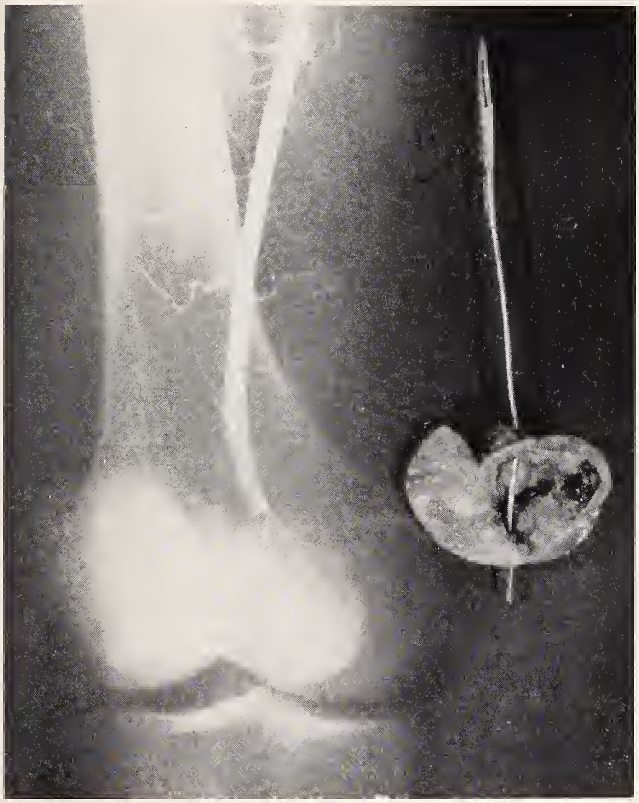

Fig. 1. Arteriogram and resected specimen from patient with bilateral popliteal aneurysms. A graft was inserted with maintenance of pulses and circulation. Patient initially seen with gangrene of contralateral limb from complication of untreated aneurysm.

Aortic Aneurysm

Aortic aneurysms due to arteriosclerosis are, with surprising uniformity, localized to the terminal abdominal aorta below the origin of the renal arteries. There is a striking difference clinically, as well as pathologically, between arteriosclerotic aneurysms and the other major types of aortic aneurysms, principally those due to syphilis. Syphilitic aneurysms are more frequently, although not invariably, saccular, most common on the ascending aorta and aortic arch, seen in younger individuals, between forty and sixty, and more frequently associated with pain or pressure on adjacent structures. Arteriosclerotic aneurysms are usually located below the renal arteries, are usually fusiform, more commonly seen in older individuals and predominently males, often associated with pain but only uncommonly with disturbance of function of the adjacent abdominal viscera. These aneurysms are becoming increasingly common in the experience of most, as was shown by Maniglia and Gregory.

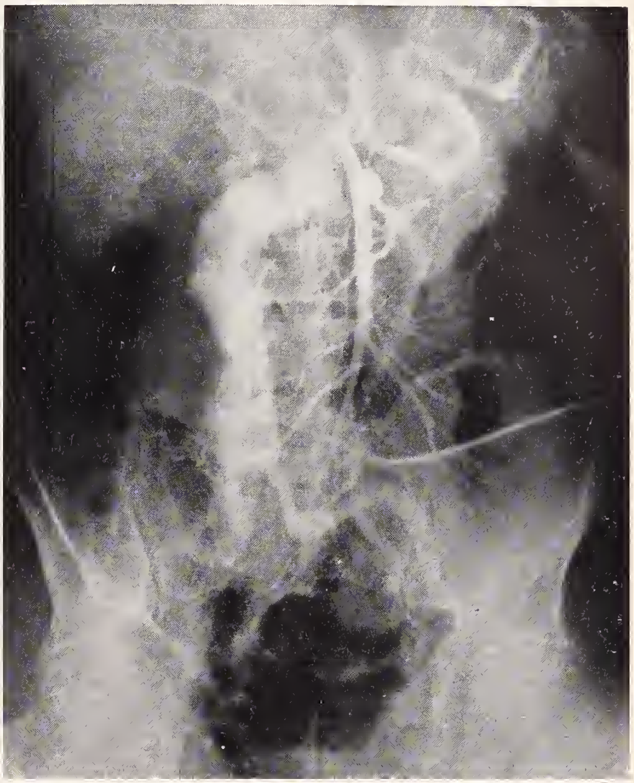

Fig. 2. Aortogram showing lumen of contrast filled aortic aneurysm. A better index of size is the calcified rim adjacent to the gas filled colon.

The localization to the distal aorta is a fortuitous and fortunate feature although the reason is unknown, other than that arteriosclerosis is most prominent in this region. From physical examination, the question often arises as to whether an aneurysm disappearing under the costal margin does not extend above the renal

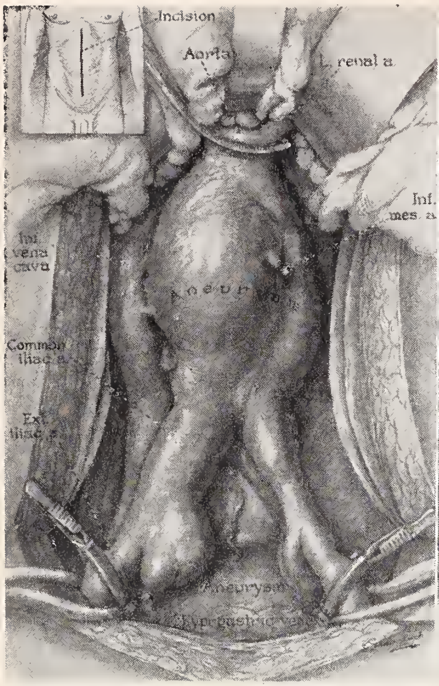

Fig. 3. Adjacent areas of the aorta and iliac vessels have been mobilized for occlusion with little dissection of the aneurysm.

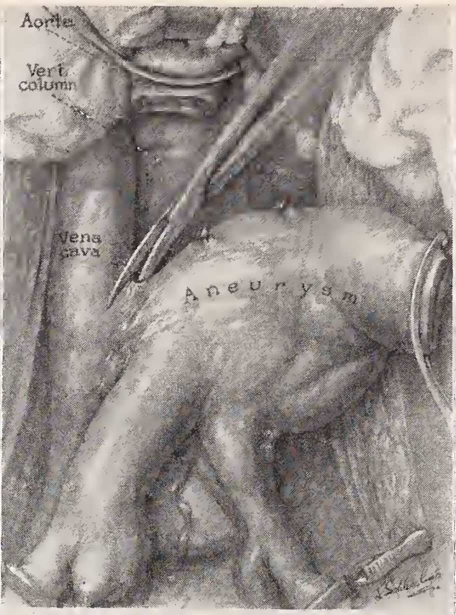

Fig. 4. After occlusion of the entering vessels the aorta adjacent to the aneurysm can be divided and the aneurysm lifted from its bed. This greatly facilitates dissection.

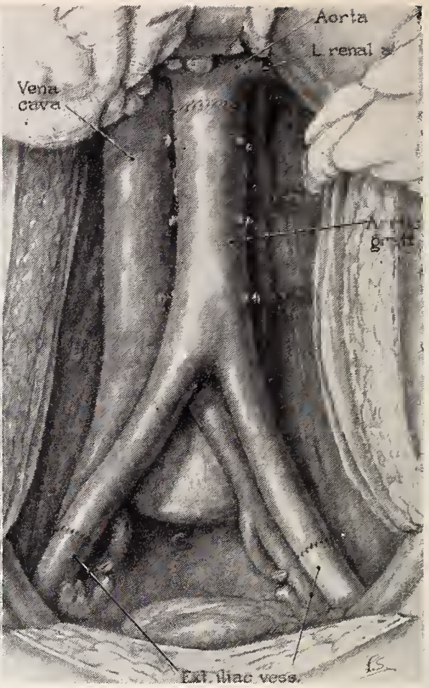

Fig. 5. Arterial homograft sutured in place to restore arterial continuity.

arteries. This can easily be reconciled with the known localization below them when one realizes that the bifurcation of the aorta is often immediately behind or even above the umbilicus and the renal arteries arise behind the xiphoid. Of the fifty-four abdominal aneurysms that we have seen in the past three years only three have extended above the renal arteries, and in one of these syphilis was also an etiological agent. This is not to say that arteriosclerotic aneurysms do not occur above the kidneys, as discussion later will show, but that when there is a discrete aneurysm in the abdominal aorta, the chances of it being localized below the renal arteries are very great.

The most frequent complaint of patients with an abdominal aortic aneurysm is the aneurysm itself, often found by physicians during routine examination or evaluation for other conditions. Some patients will complain of fullness, particularly after meals, pulsation, nausea, or other nonspecific gastrointestinal disturbances. Pain is not uncommon, and has been present at one time or another in about half of our patients and of such severity as to require opiates or the loss of weight. The severity of the pain is frequently variable and in itself is probably not a good prognostic sign. Pain, when present, has not been of a characteristic type or distribution. It is usually felt in the abdomen, often in the back, and frequently associated with tenderness of the mass itself.

We performed aortography on all of the earlier cases of abdominal aneurysm but now use it only when disease is obviously widespread, as suggested by a large thoracic aorta, when we cannot be certain clinically that the pulsating abdominal mass is not simply a slightly dilated and tortuous aorta, or when absence of pulses suggests an additional iliac stenosis. The procedure is not without danger, as we have seen sizable retroperitoneal hemorrhage sufficient to cause shock, and paraplegia from a probable thrombosis of the anterior spinal artery. Much of the lumen of the aneurysm fills with clot, and the channel filled with contrast material may be nearly normal size. The calcified rim is a better radiological index of size (Fig. 2).

The prognosis of the patient without treatment of his aneurysm is not bright. The best available data, I believe, are those of Estes of the Mayo Clinic, and from these one can estimate that about one out of five patients with arteriosclerotic aneurysms will rupture the aneurysm within one year and one-half within three to five years. It was easy for me to believe that the prognosis is even worse than this when I discovered that of the twelve patients seen in the last eighteen months whom, for one reason or another, operation was advised but not accepted, at least three are now dead because of rupture of the aneurysm. In addition one patient had a rupture and death the morning he was to be admitted to the hospital for examination.

Many forms of treatment have been used and advocated in the past such as cellophane wrapping, wiring, ligation, and aneurysmorrhaphy. None of these is associated with a sufficiently low operative risk and sufficient therapeutic benefit to justify operative treatment. The ideal treatment, and the method we are employing exclusively, is excision of the aneurysm with reconstruction by use of a graft. Wide exposure must be obtained, and we usually open the midline from symphysis to xiphoid. The aneurysm is usually densely adherent to the adjacent structures, but one may readily isolate the aorta above, between the renal arteries and the aneurysm, and the iliac arteries may be mobilized distally (Fig. 3). When these areas are occluded, bleeding is well controlled, as frequently the lumbar and inferior mesenteric arteries are thrombosed. The aneurysm can be lifted and dissected out of its bed (Fig. 4), though occasionally adherence to the adjacent vena cava is so dense that a portion of the aneurysm should be left attached to the vein. Restoration of arterial continuity can be obtained by use of an arterial homograft and anastomoses are done with standard cardiovascular techniques (Fig. 5). Grafts which we have employed have been either frozen-dried arterial homografts or, more recently, nylon bifurcation grafts. We have had no evident difficulty from occlusion of the aorta, although the time of occlusion has varied up to 150 minutes.

Since February, 1953, we have operated upon forty patients with abdominal aortic aneurysms. Thirty-one, or about three-fourths, of these are now alive and as active as their age allows. Four patients died for whom emergency excision was employed after vigorous resuscitation for a ruptured aneurysm. Three patients died either during or soon after an elective operation, which makes the operative mortality for an elective excision about 10 per cent. Two patients died four and six months following operation of coronary artery disease. These last two patients and one other lost a lower extremity because of ischemia. Embarrassment to the distal circulation was most likely due to dislodgment of emboli which form in the occluded vessels during excision or of thrombus from the aneurysm itself. Use of heparin and care in mobilization of the aneurysm has eliminated this complication in more recent cases. We have also removed several emboli from the distal circulation following operation. In one individual, extreme blanching of the leg occurred during operation and continued, with slow improvement over the subsequent several days. Later a pulse could be felt which was as vigorous as that felt prior to operation, and it was believed that almost certainly spasm alone was the cause of the difficulty. Spasm has not apparently been a problem in other patients.

Arteriosclerotic aneurysms in the more proximal aorta are less apt to be well localized, although we have seen dicrete saccular aneurysms of the lower thoracic aorta due to arteriosclerosis alone, as far as can be determined by available means (Fig. 6). Such saccular aneurysms are treated by excision and homograft during a brief period of aortic occlusion. Duration of aortic occlusion at this level must be as brief as possible, probably no more than thirty minutes.

We have seen two patients with long diffuse dilatations of the thoracic aorta, one thirty-one and the other forty-six years old. Syphilis was almost certainly excluded as an etiological agent in both individuals. In the first patient (Fig. 7) the aneurysm extended from the origin of the left subclavian artery through the region of the renal arteries. An attempt was made to excise this with aneurysmorrhaphy, an internal shunt and massive transfusions, but the effort was unsuccessful as the patient died the following day of continued shock and with a paraplegia. A second patient is being observed and, if the aneurysm continues to enlarge, will be treated probably by a staged excision.

I have borrowed from the work of DeBakey, Cooley, and Creech, a discussion of dissecting aneurysms of the aorta. These aneurysms have a particularly poor prognosis and no treatment in the past has been effective in prolonging survival and preventing further dissection and ultimate rupture. A double lumen is frequently present, often with a saccular component at some area of the second channel. This has been attacked by excision of the saccular component, if one is present, or if not, by interruption of the aorta and closure of one lumen of the aorta. In some instances the site of initial dissection has been excised and the single proximal channel anastomosed to the distal double segment, a single distal lumen being made with the anastomosis. This procedure is of only relatively recent development and ultimate results remain to be determined. To date six patients have been operated upon. Five of the six patients remain well, and one died in the early postoperative period of ventricular fibrillation perhaps related to the use of hypothermia.

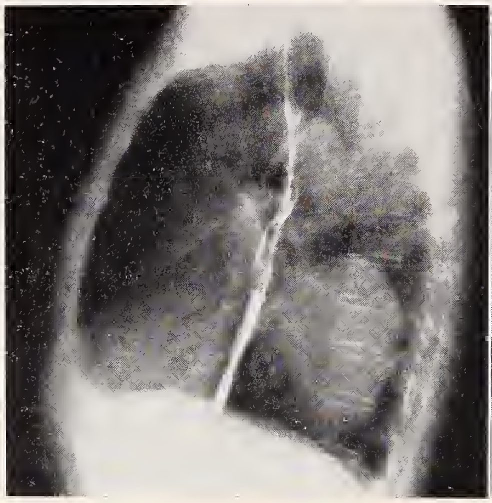

Fig. 6. Roentgenogram showing uncommon saccular arteriosclerotic aortic aneurysm just above the diaphragm.

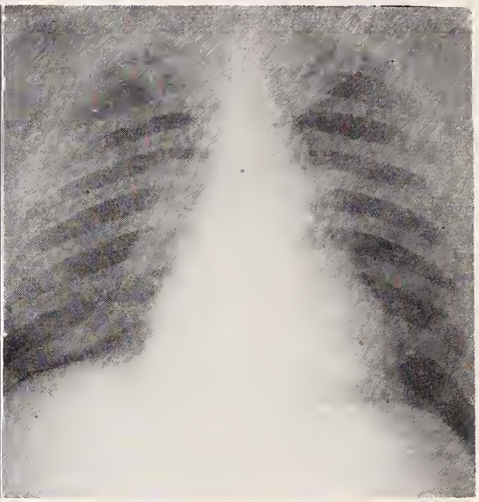

Fig. 7. Extensive aneurysm in a thirty-one-year-old man extending from the aortic arch through the upper abdominal aorta. This was apparently due to arteriosclerosis.

In instances where the aorta must be interrupted, as is true of almost all aortic aneurysms due to arteriorsclerosis, hypothermia has been suggested and used by some in order to lessen the damage to the central nervous system during the period of occlusion. We have not believed this to be indicated in patients we have treated. As mentioned above, we have no difficulty with occlusion of the terminal aorta for periods up to two and a half hours, and, as far as I know, no difficulty due to this alone has been encountered by others. Proximal to the origin of the abdominal visceral vessels a much shorter period of time, exact duration of which is unknown, is associated with irreversible damage to the abdominal viscera, particularly the liver and kidney. More proximal to this interruption of the circulation to the spinal cord is apt to be associated with paraplegia. Especially in the age group in which arteriosclerotic aneurysms occur, the risk of hypothermia seems great, and we have preferred either to use an aortic shunt or more commonly to remove the aneurysm and reanastomose the aorta as quickly as possible. Bypass shunt grafts, as temporary or permanent substitutes for long and extensive aneurysms of the thoracic aorta, have been successfully used for non-arteriosclerotic aneurysms, but the full possibilities of this method remain to be determined.

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Plastic Replacement of Diseased Arterial Segments

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CORRECTIVE surgery of diseased arterial segments entails methods for reopening or by-passing occluded and stenotic arteries and the obliteration or excision of aneurysms. For the former, two methods are available—thromboendarterectomy and the interpolation of grafts. Aneurysms of peripheral arteries can be treated by methods which involve ligation of the affected artery, but whenever a main-stem artery is affected it is highly desirable to maintain or restore its continuity. Certain saccular aneurysms of peripheral arteries and of the aorta can be excised with closure of the mouth of the sac by lateral suture. Occasionally, a peripheral aneurysm can be extirpated with approximation of the ends of the divided artery by end-to-end suture, and, rarely, this is possible in the case of an aortic aneurysm. Generally, however, a graft must be used. The need for grafts is, therefore, great.

For peripheral artery substitution autogenous veins may be employed with success. Not infrequently another type of graft is preferable for replacement of a peripheral artery and this is always true in the case of the aorta. Experimental observations from a number of laboratories suggest that venous transplants to the aorta would not be safe even if a suitable segment of autogenous vein were available, and there are no expendable appropriate veins. Arterial homografts have proved generally reliable up to the present time. To be sure, not all homografts have continued to function without complications,[1,2] and I know of several late disruptions which are not reported in the literature. Their chief disadvantage is the difficulty of acquiring them in sufficient numbers to meet the demand for their use. Over a period of years various possible substitutes have been investigated. In general, permanent intubation of arteries with rigid tubes of glass, plastics, and various metals did not prove successful. There was a high incidence of thrombosis and when they were used to replace defects in the aorta the encircling ligatures all too often resulted in necrosis of the aortic wall with fatal hemorrhage. Hufnagel and Gillespie[3] found, however, that tubes of highly polished methyl methacrylate would remain patent and that ischemic necrosis of the aortic wall could be obviated by using the principle of multiple point fixation. Using this method, Hufnagel and his colleagues[4] demonstrated that short, rigid, plastic, valve-containing tubes could be employed successfully in patients with aortic insufficiency. Obviously, the ideal plastic for general arterial substitution would be an inexpensive, readily available material obtainable in, or made without difficulty into, tubes of various sizes and shapes, sterilizable by ordinary operating room methods, pliable and easily sutured to the artery by regular vascular suture techniques. It should further be non-reactive, relatively impervious to blood, and have such properties that it would become well incorporated into the new vessel formed about it by the host. The first contribution of this sort was the demonstration by Voorhees, Jaretzki and Blakemore[5] that short tubes made of Vinyon-N cloth could be sutured to the divided ends of the aorta, served well as a conduit and became covered by a new, smoothly lined fibrous aorta. My interest and that of my associates in plastic fabrics as possible arterial grafts arose through the use of a finely woven nylon cloth* in the repair of atrial and ventricular septal defects. It was tolerated in the chambers of the heart and became firmly united with the septum and covered with a smooth endothelial-like membrane.

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*This cloth has 277 by 240 strands per square inch, obtained from Stehli Company, Inc., 1372 Broadway, New York, —241 F nylon fabric.

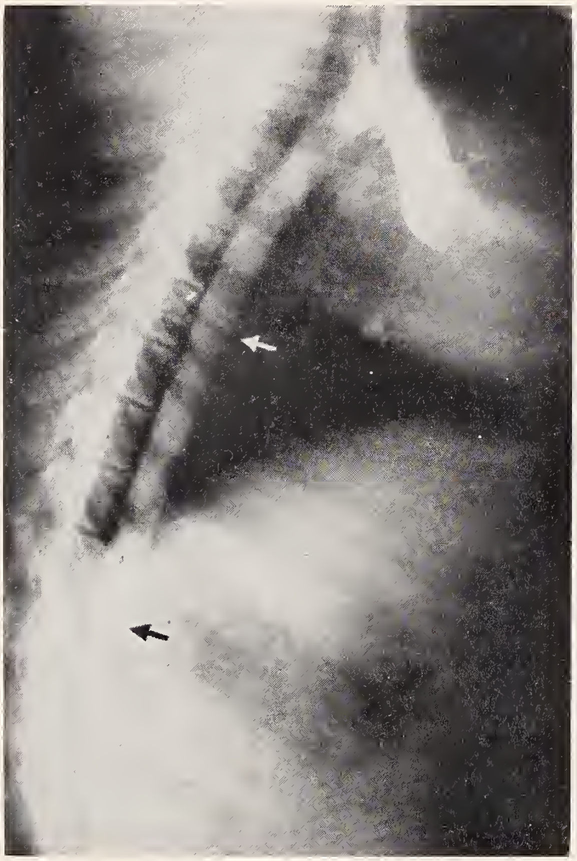

Fig. 1. Aortogram showing nylon-polythene nylon graft, 7.7 cm. in length and 230 days old. (From Shumacker, H. B., Jr., et al: Surgery, 37:80, 1955.)

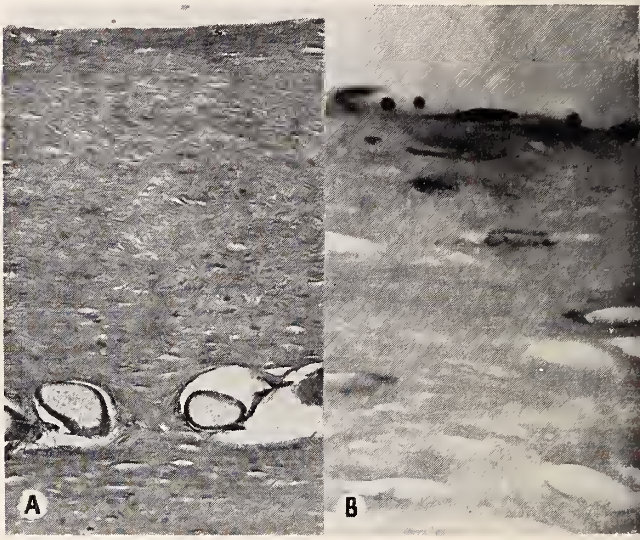

Fig 2. Photomicrographs of inner portion of graft in dog 618. Age of graft 310 days. A—low power showing growth of fibrous tissue between filaments of nylon. B—high power, showing endothelial or endothelial-like cells lining graft.

When it was first used to bridge aortic defects in dogs, this nylon fabric proved so permeable that great blood loss often occurred through the graft. For this reason, various methods for rendering it more impermeable were investigated. The fusing together, by ironing with an electric iron set to "wool," of two layers of the nylon with a thin sheet of polythene** in between served satisfactorily. In the initial study[6] seventeen grafts 1 cm. in diameter and varying in length from 5 to 10.8 cm. were observed for periods of from two to 310 days. All but one remained patent. The incidence of thrombosis was higher, however, when tubes 0.6 and 0.8 cm. in diameter were used. Of ten such grafts varying in length from 4.8 to 7.5 cm., observed for from one to 162 days, six remained patent and four clotted. For grafts of larger size the material seemed quite good. It was pliable and easily sutured to the ends of the aorta. Little or no blood loss occurred. The aortograms looked good (Fig. 1). The grafts soon became incorporated in a new fibrous aorta with a relatively smooth inner lining composed of cells which looked like endothelial cells (Fig. 2). The outer and innercoats were intimately adherent to the plastic tube and fibrous tissue grew into the pores of the nylon. Ninety-four per cent of the larger grafts remained patent. During the course of this study, it was necessary to use similar grafts for human aortic replacement because of lack of availability of homografts.[7] They seemed to work well. Concurrently, the same nylon cloth coated with a thin film of methyl methacrylate was investigated. Again, the incidence of patency was high with grafts 1 cm. (92 per cent) and lower with grafts 0.6 and 0.8 cm. in diameter (68 per cent). These grafts, however, were not as satisfactory as the others for in a number of instances narrowing occurred as the result of a thrombus laid down between the plastic tube and the outer fibrous coat.

My associates and I[8,9] have subsequently undertaken a comparative study of a variety of plastics and sufficient experience has been obtained to permit an evaluation of four types of plastic grafts and to compare them with freeze-dried aortic homografts.†

**Obtained from the Clay-Adams Company, Inc., 308 W. Washington Street, Chicago, PE 2600 Polyethylene film, 0.0015 thickness.

†The aortic homografts were removed from donor animals without aseptic precautions, cleaned of all fatty areolar tissue, covered with liquid ethylene oxide for one hour, frozen quickly in Pyrex tubes immersed in dry ice-acetone bath, lyophilized, and stored in sealed tubes filled with nitrogen.

TABLE I. RESULTS WITH HOMOGRAFTS AND A VARIETY OF PLASTIC GRAFTS

Age of Graft in Days	Excellent	Good	Satisfactory	Poor	Dog Alive with Good Pulses
Homografts					
0- 29	3	0	0	1	0
30-149	4	1	0	0	0
150-249	3	1	0	0	0
250-349	1	0	1	0	2
350-400	0	0	0	0	2
Nylon Filter Fabric					
0- 29	7	2	0	0	0
30-149	4	3	1	1	2
150-249	0	4	1	0	9
250-349	1	0	0	0	2
Braided Nylon Tubes Treated with Formic Acid and Silicone					
0- 29	2	0	1	1	0
30-149	3	0	0	0	0
150-249	0	0	0	0	9
Two Layers of Nylon Fused with One Layer of Polythene					
0- 29	2	1	0	3	0
30-149	1	1	0	0	0
150-249	1	1	0	0	0
250-349	0	0	0	0	2
350-400	0	0	0	0	3
One Layer of Nylon Fused with One Layer of Polythene					
0- 29	4	1	0	2	0
30-149	0	2	0	1	0
150-249	1	0	3	0	0
250-349	0	0	0	0	3
350-400	0	0	0	0	3
Vinyon—N					
0- 29	0	0	1	5	0
30-149	2	1	1	0	0
150-249	3	0	0	1	0
250-349	0	0	0	0	1
350-400	0	0	0	0	1

One is a nylon filter fabric,‡ one the previously described material composed of two layers of thin nylon and one of polythene fused together, one commercially fused§ single layers of the same nylon cloth and polythene film, and one a Vinyon-N cloth similar to that used by Vorhees, Jaretzki and Blackemore.†† All plastic grafts were sewn together by machine with a fine needle and fine nylon thread, 25 stitches per inch. Some, but not all, of the nylon filter fabric was washed beforehand in Tide. The rest of the plastics were unwashed. The Vinyon-N grafts were boiled in water for fifteen minutes. The other plastic tubes were autoclaved at 250° F. and at 20 pounds pressure for ten minutes. The freeze-dried homografts were reconstituted by soaking them in normal saline solution containing streptomycin and penicillin for one hour.

All the grafts were 5.4 cm. in length and all the plastic tubes were 0.78 cm. in diameter. They were sutured into thoracic aortic defects of dogs following excision of a 5 cm. segment under moderate hypothermia. An everting mattress suture of 5 zero silk was used. Only in the case of the Vinyon-N grafts was a cuff turned back at each end of the tube.

Some observations were also made with the braided nylon seamless tubes of Edwards and Tapp.[10]‡‡ This study is similar but not entirely comparable to that of grafts made from the other plastics. These tubes were of two sizes, approximately 0.4 and 0.6 cm. in diameter. They, too, were 5.4 cm. in length. They were sutured to the aorta with a continuous plain over-and-over stitch. This series of grafts was not studied over as long a period of time as the others.

The results of these investigations are tabulated in Table I. Grafts are classified as excellent when they were found to be widely patent with a thin, smooth lining and were without external constriction or clots. They were considered good when they were widely patent with an essentially smooth lining, with minimal erosion or degenerative changes, and without external constriction but with minimal external clots in some cases. If the pathway through the graft was narrowed and yet served as a satisfactory conduit or if the inner coat was roughened due to a mural thrombus or excessive intraluminal fibrinous reaction or if there was some external constriction from a large clot or fibrous reaction, the result was classified as satisfactory. It was considered poor if it failed as a conduit either from exsanguination through the graft, disruption, or an occluding thrombus. Four animals are excluded from consideration because the unsatisfactory result seemed clearly the consequence of a technical error.

Detailed data are listed in the first table. In Table II the results for each group are sum-

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‡This is an off balance taffeta, constructed of multifilament twisted yarn strands with a warp count of 232 and a filling count of 96. It is 70 denier, high tenacity nylon with 36 turns per square inch. After autoclaving, it has such a resistance that a pressure equivalent to from 4 to 5 inches of water will cause it to pass 20 C.F.M. per square foot of air. The tubular grafts were fashioned by cutting the material on the bias so the strands of the warp were at a 45° angle to the long axis of the graft. HT-5F nylon filter fabric. Equipment Development Company, Inc., 368 Bloomfield Ave., Montclair, N. J.

§Obtained through the courtesy of Dr. C. E. Iron, Ben-Mont Papers, Inc., Bennington, Vermont.

††Supplied by Mr. T. P. Mulligan, Carbide and Carbon Chemicals Company, a division of Union Carbide and Carbon Corp., 30 E. 42nd St., New York 17, N. Y.

‡‡These braided tubes have a large porosity. They were treated with formic acid to solve the problem of easy frayability, crimped in an accordion-like manner to permit bending without wrinkling, and siliconed to prevent blood loss through the wall of the graft.

TABLE II. RESULTS WITH HOMOGRAFTS AND A VARIETY OF PLASTIC GRAFTS FROM STUDY AFTER DEATH OR SACRIFICE

Type of Graft	Excellent	Good	Excellent or Good	Satisfactory	Poor
Homografts	11(73)	2(13)	13(86)	1(7)	1(7)
Nylon filter fabric	12(48)	10(40)	22(88)	2(8)	1(4)
Braided nylon tubes	5(71)	1(14)	6(85)	0	1(14)
Nylon-polythene-nylon	4(40)	3(30)	7(70)	0	3(30)
Nylon-polythene	5(36)	3(21)	8(57)	3(21)	3(21)
Vinyon-N	5(36)	1(7)	6(43)	2(14)	6(43)

The numerals indicate numbers of grafts; those in parentheses per cent of total.

TABLE III. RESULTS WITH HOMOGRAFTS AND A VARIETY OF PLASTIC GRAFTS FROM STUDY OF ALL ANIMALS*

Type of Graft	Good or Excellent	Satisfactory	Poor
Homografts	17(90)	1(5)	1(5)
Nylon filter fabric	35(92)	2(6)	1(3)
Braided nylon tubes	14(88)	1(6)	1(6)
Nylon-polythene-nylon	12(80)	0	3(20)
Nylon-polythene	14(70)	3(15)	3(15)
Vinyon-N	8(50)	2(12)	6(38)

*Animals still alive with good femoral pulses are considered to have a good result.

The numerals indicate numbers of grafts; those in parentheses per cent of total.

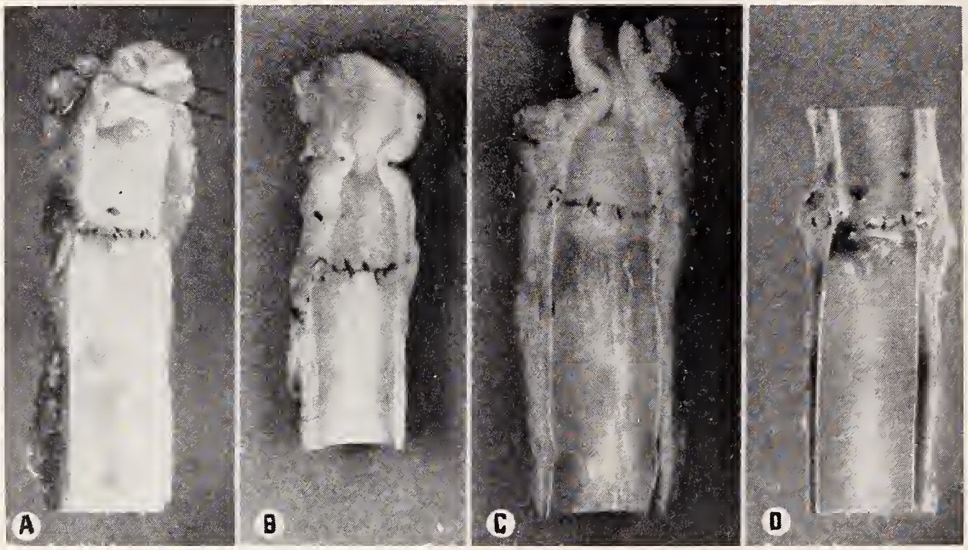

Fig. 3. Photographs of portions of four nylon filter fabric grafts and the adjacent ligated aorta, 180, 120, 60 and 30 days after implantation. (From Harris, E. J., et al: Arch. Surg., 71:449, 1955.

marized, with consideration given only to those grafts examined post mortem. It will be seen that good or excellent results were obtained in from 85 to 88 per cent of the homografts, nylon filter fabric grafts and the braided nylop tubular grafts, while poor and satisfactory results were noted in only a small percentage. The per cent of good and excellent results in the other three types of grafts ranged from 43 to 70. In these groups, the poor results varied from 21 to 43 per cent.

In Table III the outcome in all the animals is analyzed, including those which are surviving. Here grafts in living dogs with strong peripheral pulses are considered as having a good result. Approximately 90 per cent of the homografts, nylon filter fabric grafts and braided nylon tubes had good or excellent results. In roughly 5 per cent of each the outcome was satisfactory and in another 5 per cent, poor. In the other three groups, from 50 to 80 per cent had good or excellent results.

It is apparent that the results with nylon filter fabric grafts were better than those with the two sorts of fused nylon and polythene and with Vinyon-N. If the smaller series observed over a comparatively brief period is significant, the crimped braided nylon tube grafts also are quite satisfactory.

All those who engaged in these studies agreed that the freeze-dried homografts were superior to all the plastic grafts from the standpoint of ease of interpolation and the appearance of the graft up to periods of approximately one year. However, the nylon filter fabric grafts proved altogether quite satisfactory (Fig. 3). They were a bit more difficult to suture to the host aorta but were anastomosed with relative ease and rapidity. About the same percentage gave good or excellent results and the percentage of poor or only satisfactory results was correspondingly low. They became thoroughly incorporated in the new aortas built about them by the host (Fig. 4). The same seems to be true of the braided nylon tubes. They were easily affixed to the aorta and yielded a high percentage of good and excellent results (Figs. 5 and 6). When successful, the Vinyon-N grafts resulted in quite satisfactory new aortic segments (Fig. 7) but, in our hands, they gave the highest incidence of poor results. The fused nylon and polythene grafts more often yielded good results than those of Vinyon-N but were inferior to grafts of nylon filter cloth and the braided nylon tubes.

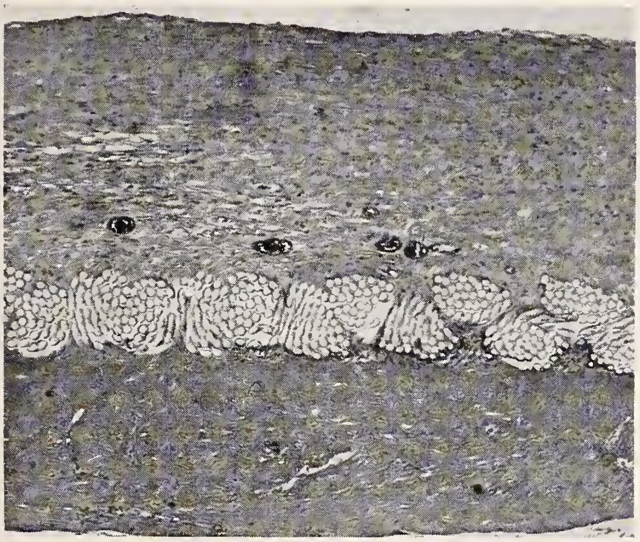

Fig. 4. Photomicrograph of nylon filter fabric graft 120 days old. (From Harris, E. J., et al: Arch. Surg., 71:449, 1955

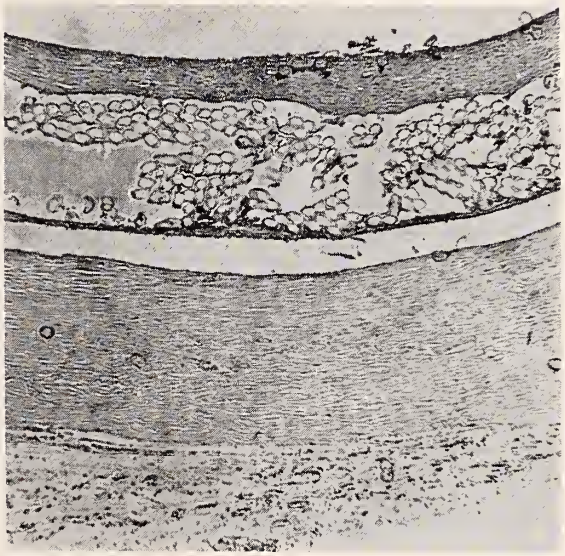

Fig. 6. Photomicrograph of braided nylon graft sixty days old. The defect in the nylon and the separation of layers is an artefact due to sectioning.

Clinical Experiences

Aortic and Aortic Bifurcation Grafts.—My experience has been limited. Altogether twenty-five plastic grafts have been used to replace segments of the aorta and the aortic bifurcation (Fig. 8). Unfortunately, not all of the patients survived. In all but two of those who died following operation a post-mortem examination was carried out with demonstration of patency of the graft. In one of the exceptional cases, a man of sixty-one with a previous history of myocardial infarction, death in circulatory collapse occurred two days after operation and permission for autopsy was not obtained. The other case will be described in more detail. The surviving patients have been observed over periods ranging from a few weeks to twenty-three months. Their grafts have remained patent. In addition,

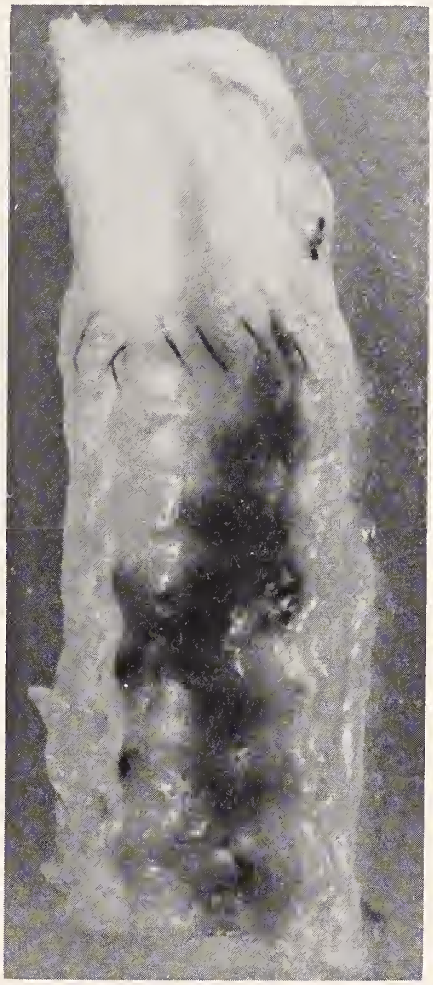

Fig. 5. Photograph of portion of chemically treated braided nylon tubular graft and adjacent aorta 112 days after implantation.

I know of a small number of cases in which other surgeons have used with success grafts of fused nylon and polythene and grafts of the nylon filter cloth.

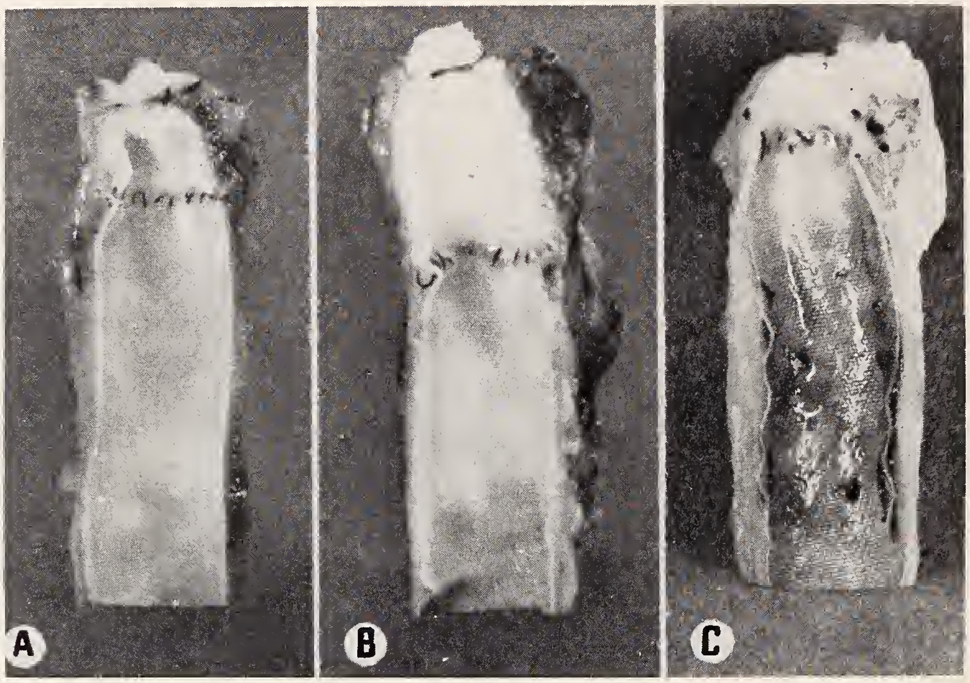

Fig. 7. Photographs of portions of three Vinyon-N grafts and adjacent ligated aorta 180, 120, and 60 days after implantation.

Heat-fused grafts of two layers of thin nylon and a single layer of polythene film were used in fourteen instances. Two were straight aortic grafts, five were anastomosed to the aorta above and the common iliac arteries below, five were interposed between the aorta above and one common iliac and one external iliac artery below, and in two cases the graft was interpolated between the aorta and both external iliac arteries. They all were sutured in place without undue difficulty and they served well as conduits. Difficulty was encountered in one case.

A man, aged sixty-three, who underwent excision of a large abdominal aneurysm on May 14, 1954. It involved the proximal portion of a very badly diseased left common iliac artery and extended down to involve the external iliac and hypogastric arteries on the right. The graft was sutured to the aorta, the left common iliac and the left external iliac artery. His immediate recovery was good and there were excellent pulses in both lower extremities. However, on May 23 he developed fever, and by June 4 it was clear that he had a retroperitoneal abscess. On that date a very large infected hematoma was evacuated and drained through a muscle-splitting extraperitoneal incision. The entire graft lay in the abscess cavity and was completely surrounded by infected material. The fever disappeared promptly and he was discharged from the hospital on June 11. There was moderate drainage at that time. When seen on July 2 he was still having slight drainage but was feeling well and had excellent circulation in his lower extremities. On July 28 he stated that he had begun to drain a small amount of bright bloody fluid from the sinus tract two days before. The pulses in both lower extremities were still excellent. He was urged to re-enter the hospital, but in spite of persistent warning he remained at home until August 19.

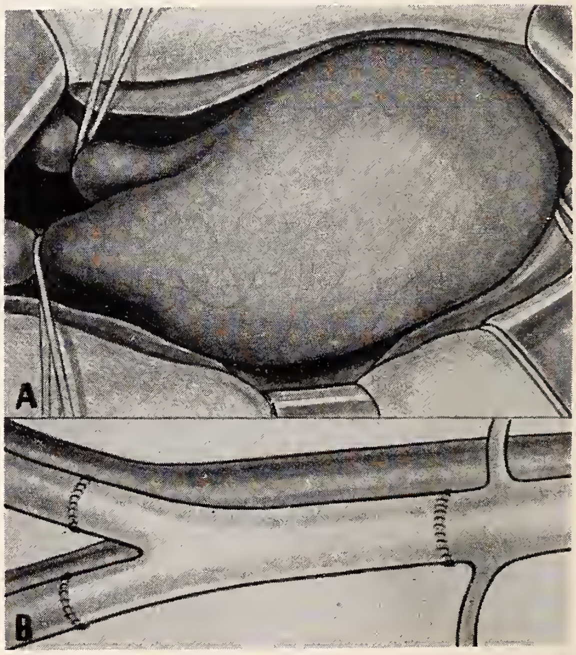

Fig. 8. Drawing of typical abdominal aortic aneurysm and interposed plastic graft after its excision. (From Shumacker, H. B., Jr., and King, H.: Surg., Gynec. & Obst., 99:287, 1954.)

He had had repeated massive hemorrhages from the sinus tract and for a few days also from the rectum. He was nearly moribund on admission. The circulation n both lower extremities was very poor. Hemoglobin was 3.3 gm. and erythrocyte count was 1.39 million. He had passed very little urine during the preceding twenty-four hours, and the urine contained albumin and had a low specific gravity. Non-protein nitrogen was 68 mg. per cent. He was not bleeding. Repeated transfusions of blood were administered and his condition mproved. Just before the time of his scheduled operation the following morning, however, he began to bleed massively both from the sinus tract and rectum and his condition rapidly deteriorated. He was taken mmediately to the operating room.

There was a large, ruptured, poorly organized aneurysm arising in the region of the left iliac artery and compressing the aorta above and the iliac vessels on the right. The open end of the common iliac artery ay in the hematoma. A small rim of iliac artery was attached to the iliac extension of the graft. There was a relatively fresh thrombus in the graft which otherwise was in good condition. A new bifurcation graft was interpolated. The aneurysm had eroded into the rectosigmoid. The opening was closed and a proximal sigmoid colostomy performed. He died in uremia four days later with a non-protein nitrogen of 155 mgm. per cent and potassium of 7.2. It had been possible to keep the serum sodium and chloride levels normal and, until the last day, the potassium level. No autopsy was permitted.

Under the circumstances which characterized this case it seems clear that any graft might have failed. Actually the graft functioned well in an infected field for over two months. At the final exploration it was imposible to tell whether rupture of the iliac artery or disruption of the graft-iliac suture line had occurred. It was clear that an aneurysm had developed with subsequent rupture through the sinus tract and into the rectosigmoid and with compression and secondary thrombosis of the aortic graft.

A methyl methacrylate coated thin nylon graft was used in a single case. It was a long bifurcation graft between the aorta and both common iliac arteries inserted after excision of a large aneurysm. The patient has done well and the graft has functioned nicely for seventeen months.

Nylon filter fabric grafts have been used for aortic replacement in ten cases (Fig. 9). Five were straight aortic grafts and four were bifurcation grafts between the aorta and both common iliac arteries. One was a curved aortic graft replacing a portion of the arch and almost all the descending thoracic aorta. Two of these patients died, both with straight thoracic aortic grafts. Their grafts were found patent on post mortem study. The remainder are well with nicely functioning grafts from a few weeks to nine months after operation.

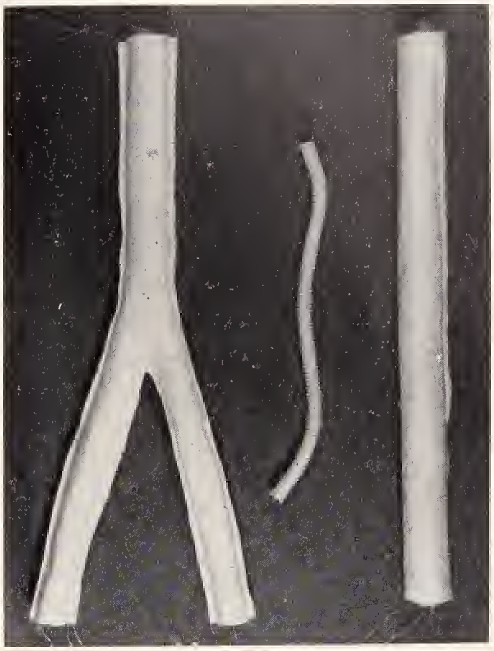

Fig. 9. Photographs of bifurcation and straight aortic grafts made of nylon filter cloth and braided chemically treated nylon tube for smaller artery replacement. The nylon filter fabric grafts have been stuffed with cotton before photographing. The crimped braided tube is seen to bend without kinking.

Smaller Grafts.—My experience with smaller grafts in patients has been even more limited. Altogether I have used eight such grafts and my associate, Dr. T. C. Moore, two additional grafts. Only in four of these cases was there a patent distal arterial tree. In the others there was distal obstruction and completely inadequate "run-off." In the four favorable cases an excellent result was obtained. All were braided nylon tubes interpolated end-to-end to the artery proximally and distally. One was a common iliac, one a subclavian, and two femoral grafts. Three other braided nylon tubes were used in relatively hopeless cases. Two femoral-popliteal grafts failed. In one case in which it seemed most unlikely that the graft would succeed it worked well. In this patient a long graft was sutured end-to-side to the proximal portion of the thromboendarterectomized common iliac artery and to the common femoral artery. Thromboendarterectomy of the entire femoral and popliteal arteries was carried out, but the terminal portion of the popliteal could not be reopened and blood flow through only small branches was restored. In one case a nylon-polythene-nylon graft was interpolated between the aorta and femoral artery. There was no retrograde flow of blood and the limb was completely ischemic. The graft did not remain patent. In two similarly unfavorable cases, nylon fabric grafts failed. One was between the aorta and femoral artery, the other between the common iliac and femoral artery. In this small series the grafts were successful in four favorable cases as well as in one in which it was thought the chances of success were very poor. The failures occurred in cases in which it is doubtful that any type of graft would have remained patent.

Discussion

A number of plastic materials have now been evaluated as arterial substitutes. Vorhees, Blakemore and Jaretzki[5,11] and D'Angelo and his associates[12] have studied Vinyon-N, as has my own group.[8,9] Though some excellent results have been obtained, the use of this material has resulted in a significant percentage of failures. The leaking of blood through the cloth is an additional handicap and pre-clotting of the cloth to obviate this difficulty may have some disadvantages.[13-15] Hufnagel and Rabil have gotten good results with orlon.[13,14] From their analysis of physico-chemical characteristics of a number of plastics, Deterling and Bhonslay[15] concluded that dacron might be the best material, with orlon and nylon as close second choices. Their small experience was good with grafts of these three materials. Shumway and his colleagues[16] from short term observations report excellent results with prostheses made of polyvinyl sponge. From the available literature and our own studies, it appears that at least nylon, orlon and dacron are suitable for plastic vascular prosthetics and undoubtedly other plastics also can be used for this purpose. Our own work leads me to prefer at the present nylon filter fabric grafts for aortic and simple aortic bifurcation replacement and the chemically treated braided nylon tubes of Edwards and Tapp for small artery substitution or by-pass.

Clinical experiences as yet recorded in the literature are small. I know that a number of surgeons have had successful cases which are not yet reported. The anastomosis of homografts is somewhat easier to perform than the union of any plastic tube with the host artery and yet the latter can be interpolated without any real difficulty. There are certain advantages of the plastic prostheses. They are readily available, easily sterilized, and can be constructed to fit the individual demands of the patient. This point is illustrated by a recent case in which a huge ruptured abdominal aortic aneurysm was excised. The abdominal aorta above and below the aneurysm was markedly dilated. A graft was quickly made to fit the proximal and distal aortic ends. It was 15 cm. in circumference at the upper end, and 12 cm. in circumference at the lower end. No homograft could have fitted so well. The plastic material is relatively indestructible and one would presume it would lend strength to the newly formed artery for an indefinite period of time.

It has proved even more difficult to obtain smaller artery homografts than aortic homografts. For this reason, I have used principally venous autografts and plastic grafts for replacement of arteries other than the aorta. At the moment I prefer grafts made of nylon filter cloth for aortic and simple aortic bifurcation replacement. Only careful follow-up studies of many patients will establish better the relative merits of homografts on the one hand and plastic grafts on the other.

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Discussion

F. John Lewis, University of Minnesota: It is now lear, through the significant work of Dr. Shumacker nd others, that plastic vessels may be used successfully o replace at least some parts of the arterial tree. The rtery ultimately obtained is certainly not a normal one ut it is usually better than the diseased one it replaces nd it is probably as good as the vessel that results rom a homograft.

A number of different fabrics have been advocated, nd so a study like Dr. Shumacker's which sets out to neasure the relative merit of various plastic arteries s an important one. We are engaged in a similar project vhich has not yet progressed as far. In our study, owever, we are investigating not only cloth arteries but lso vessels made of a different type of plastic. This lastic is not cloth, but rather a polyvinyl sponge Ivalon®).

As Dr. Shumacker began his work by repairing exerimental, septal defects with nylon, we too started vithin the heart and used molded plugs of Ivalon to lose experimental ventricular septal defects. These evices were well tolerated and we were impressed with he adaptability of the material. It could be permanenty molded into almost any shape and density simply by oiling it. Tubes that had the elasticity and consistency f arteries could be made so that they were impervious to blood but still porous enough to allow fibrous tissue in-growth. They are easy to stitch in place because they hold their form and hold stitches without tearing.

In dogs these Ivalon arteries have worked well in the abdominal and thoracic aorta. In a few animals followed for over one year abdominal grafts are still patent but not dilated. It is relatively easy to make substitutes for the aortic bifurcation with this material and more complicated vessels can be constructed, as well. We have made Ivalon aortic arches and substitutes for the abdominal aorta from the level of the diaphragm down with all the major branches. Pleated vessels similar to the woven nylon tubes of Edwards and Tapp can be made, and we have even tried to make a cusped aortic valve with the material.

Unfortunately, these plastic replacements are never transformed into normal arteries. They become, in time, firm, fibrous tubes instead. The lively elasticity of newly made Ivalon arteries is soon lost, but this is a characteristic of all arterial substitutes. Some plastic arteries may have fewer undesirable characteristics than others, and a number of them may, for various reasons, actually be better than homografts. This is a difficult evaluation to make but it can be accomplished through studies such as the excellent one that Dr. Shumacher has described.

Without the host of willing volunteers who annually omprise the Heart Fund army, the battle against heart isease could never have been joined.

In its earliest years, the Minnesota Heart Fund relied rgely upon a small group of physicians for direction f the campaign. Although their time was limited, this roup succeeded in establishing a financial "beachhead."

Then, in 1951, members of the Minnesota Association f Life Underwriters volunteered to adopt the Heart und as their state-wide service project.

Bringing their innate salesmanship and enthusiasm nto the annual February campaign, this group was instrumental in rapidly accelerating the financial growth of the Heart Fund.

Realizing that the manpower of their organization was not great enough to conduct more intensive drives, the group called upon Minnesota housewives for assistance.

In 1954, a "Heart Sunday" army of these housewives staged a one-afternoon collection of funds for heart research. The result was inspiring. In the span of just four hours, more than $110,000 was collected. Results in 1955 were even more gratifying, and the 1956 "Heart Sunday" idea will be expanded to cities throughout the state on February 26.

Current Concepts

PRESENT TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS

WENDELL H. HALL, M.D.
Minneapolis, Minnesota

It is ten years since the first patients with subacute bacterial endocarditis were successfully treated with penicillin in large doses. It is worthwhile to survey what we have learned in the past decade about the treatment of this malignant disease. The interested reader may wish to refer to two recent and extensive reviews of the subject.[1,2]

It is obvious that no chemotherapy should be given until several venous blood cultures have been obtained. In every patient with a positive culture, antibiotic susceptibility tests should be performed. The disc-plate method is not sufficiently accurate for this purpose. Lacking exact susceptibility data, the physician is deprived of the best guide to successful therapy.

Presently, penicillin seldom is given alone. Aqueous penicillin G is generally preferred, usually given intramuscularly at short intervals or by slow intravenous drip. The minimum daily dose is not less than 2,000,000 units and, with resistant infections, may reach 10 to 80 million units. The duration of effective therapy should be not less than six to eight weeks. With very susceptible strains of *Streptococcus viridans,* procaine penicillin 1,000,000 units plus dihydrostreptomycin 1 gram intramuscularly twice daily for two weeks has been successful, but it is not recommended as a routine method.[3] Anticoagulant drugs are dangerous and unnecessary.

All patients with negative blood cultures must be treated as though the infective organisms were not very susceptible to the antibiotics. With all resistant infections, streptomycin 1 gm. intramuscularly twice daily should be added. Some infections failing to respond to this treatment have been cured with oxytetracycline (Terramycin), or streptomycin plus erythromycin.

Early diagnosis and adequate therapy are important. Unfortunately, death may occur from congestive heart failure, major arterial occlusion or renal insufficiency though the infection be controlled. This is particularly true if the aortic valve is infected. The treatment is costly and uncomfortable to the patient and often difficult for the physician, but with energetic therapy 75 per cent of patients may be spared from an otherwise certain death. Because of valvular damage, those patients who recover may require continued cardiac therapy. Prophylaxis with penicillin should be given during each subsequent serious respiratory infection, and dental or surgical procedure for recurrences are not rare.

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Laboratory Aids

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The Minnesota Society
of Clinical Pathologists
George G. Stilwell, Editor

PATHOLOGY HAS EMERGED FROM THE "DEADHOUSE"

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A surprisingly large number of people, including perhaps too many physicians, still view the pathologist and his medical specialty in a light that is based on concepts founded so far in the past that their moldiness shows. It is true that pathology during the latter half of the nineteenth century, the pathology of Virchow, was somewhat static "deadhouse" pathology. However, it must be remembered that Virchow died in 1902; many changes have occurred in medical practice during the five decades that have transpired since the death of the "father of pathology."

Far from the least of these changes have been those which have taken place in laboratory medicine. A clear reflection of such changes is seen in the altered professional and economic status of the pathologist. Naturally, the economic returns from any specialty are of little basic importance in the "rating" of the various specialties but they do give some insight into the acceptance of any particular field. The story is told of the physician who went to a medical meeting one wintry day some years ago. Approaching the place of the gathering, he noted a line of Cadillacs. "I see the surgeons are here," he said. Seeing next some Buicks, he stated, "The internists are also here." A line of Fords caused him to say, "Well, the peditatricians came, too." He entered the building and noted a group of well-worn and wet overshoes. "Ah!" he exclaimed, "and here are the pathologists."

It was just thirty-five years ago that the *Journal of the American Medical Association* carried advertisements for those seeking pathologists under the heading of "Lab. Technician Wanted." The promised returns were commensurate with the obvious scientific rating of the specialty at that time. The same journal, in its issue dated October 29, 1955, carried an advertisement for a pathologist to direct the department of pathology in a hospital of medium size; the advertisement stated that the guaranteed income and percentage should net to $35,000.

Times really have changed, both professionally and economically, for the pathologist. No longer is he considered to be somewhat of a medical recluse who chose to fossilize himself with formaldehyde because he did not have the capabilities to become a surgeon or an internist. The modern clinical pathologist probably is expected to know more about the multitudinous fields of medicine both diagnostically and therapeutically than is any other specialist. Recent developments in medicine have drawn him intimately into contact with both the scientific practice and the administration of such fields as clinical chemistry, blood banking, radioactive isotopes, hematology and forensic medicine, to mention only a few. The pathologist certainly is a leader in many of the facets of postgraduate medical education, as witnessed by the fact that in hospital after hospital he is in charge of the teaching program for interns and residents and is responsible for the major part of the clinicopathologic conferences and the general staff meetings.

The extent of the ramifications of medicine with which the clinical pathologist is expected to be familiar is emphasized by perusal of the titles of the original articles listed in the index of the most recent complete volume of the *American Journal of Clinical Pathology*. A brief glance reveals such topics as basic research on new subdivisions of blood groups, heparin, components of thromboplastin, paper chromatography, techniques for determination of protein-bound iodine, spectrophotometric analyses, chemical determinations of such blood constituents as calcium, potassium, chloride, phosphatase, sulfate and

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From the Section of Publications, Mayo Clinic.

This is the thirty-fourth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

urea, bacteriologic techniques, new factors in the coagulation of blood, radioactive cobalt, cortisone, emission spectrography, Coombs' antiglobulin test, sensitivity tests for antibiotics, microhematocrit methods and various turbidity techniques for hepatic function. Many of these items were unknown fifteen or twenty years ago.

The small list of topics just given might lead one to believe that the modern pathologist has foregone the pleasure of microscopic study of the variegated blues and reds of tissue stained with hematoxylin and eosin. Such is not the case, however. The same journal also contains a wealth of articles devoted to pathologic anatomic diagnosis. Included are discussions of bone-marrow studies, cytologic examination for malignant cells in various bodily fluids, histoplasmosis, hepatitis, mumps, encephalitis, various tumors, rheumatic heart disease, nephrocalcinosis, tuberculosis and gastric granulomas.

These admittedly partial lists of the present interests of pathologists certainly indicate that the modern laboratory physician has removed himself far from the confines of the deadhouse. He plays an important role in modern diagnosis and treatment, both surgical and nonsurgical. Contrary to the older beliefs still held by some clinicians, the pathologist is deeply interested in the "living" patient. Without the services of a good pathologist and the availability of adequate laboratory facilities, it becomes increasingly difficult for the clinician to take full advantage of new diagnostic techniques.

It must be remembered constantly that the pathologist is a consultant in medical practice. He is, of course, familiar with the various techniques employed in his laboratory but the factor of ultimate importance is his broad knowledge of the application and limitation of these methods and the interpretation of their results. The pathologist must be familiar with the clinical aspects of many diseases and with their treatment. He is available as a consultant to aid in the choice of the diagnostic procedures that will give the greatest aid to the welfare of the patient at the lowest cost. His interpretation of surgically removed tissues and biopsy material guides the surgeon in the latter's decisions regarding treatment.

Yet, in some localities, the practice of pathology is not considered to be the practice of medicine. As many readers know, a suit has been brought by the hospitals of Iowa against the Iowa pathologists and the attorney general of that state as a result of the official ruling made by the attorney general that the practice of pathology was the practice of medicine.

Obviously, it does not take the ruling of an attorney general to bring out the fact that the pathologist is a practitioner of medicine. Yes, it is true that the modern pathologist still is slightly redolent of formaldehyde from his surgical and necropsy tissues. At the same time he carries a smudge of iodine from the patient whose sternal bone marrow he aspirated an hour ago, a dab of bromophenol blue from a recent test involving paper chromatography, a bit of frost from the blast of carbon dioxide used in a just-completed frozen section of tissue, and a safety badge to guard against an overdose of radioactivity.

Pathologists always have insisted, and justly so, that the practice of clinical pathology is the practice of medicine. They will welcome the internist or surgeon who seeks them out in medical consultation. Their wide experience in so many fields of modern medicine may provide just the hints needed to help solve bothersome problems, thus bringing the greatest possible good to the greatest number of patients.

THE MUSHROOM AS A PROTECTIVE AGENT

In Central Europe and in Pennsylvania, folklore credits the eating of certain species of mushrooms with protective effect against development of human cancers. Extracts from ten of eighty species examined do indeed demonstrate some inhibitory effect upon transplanted cancers in mice, report Dr. E. H. Lucas and Joseph Stevens, Michigan State College. The extracts apparently contain some cancer-retarding factor, according to the studies done in collaboration with the Sloan-Kettering Institute, New York. The factor is apparently heat resistant. None of the extracts has yet been purified sufficiently for human experiments, and any role in providing clues toward cancer control is yet unknown. —Medigrams: *GP* 12:240 B (Oct.) 1955.

Editorials

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

COMMUNICATION: AN OVERVIEW

It took World War I, with its recognition of the strategic importance of the communication front, to make us properly aware of the full significance of communication. A more efficient use of language was then imperative. Up to that time, the emphasis, both in high school and college, was on written composition. As the spotlight turned to the broader concept, we began to see that writing was only a small fraction of the process—that reading, speaking, and listening also belonged in the picture.

Early research done by Rankin, at Ohio State University, added to our realization of the importance of this concept. His survey revealed that the average individual spends *70 per cent of his waking time* in verbal communication. Of that time, 45 per cent is spent in listening, 30 per cent in speaking, 16 per cent in reading, and 9 per cent in writing.

We also began to be aware of the vital distinction between expression and communication. "Eschew polysyllabic verbal symbols" is expression, but is it communication? "Don't use big words" is more likely to be both.

Gradually, the whole process of communication assumed a clearer perspective. For every writer, we began to think of a reader; for every speaker, a listener. Communication, a sharing experience, demanded a sharing of responsibility if it were to succeed.

A nutrition consultant surveyed 100 pregnant women on their understanding of twenty words typically used in consultation. Although all were natives of Florida, only forty-six knew what was meant by citrus fruit. How often does communication break down partially or completely through failure to understand the capacity of listener or reader?

Furthermore, those of us who are professionally trained have a particular problem. It is easy for the college graduate to forget that college graduates make up less than 6 per cent of our population. Yet, in a democracy, the college-trained 6 per cent must communicate effectively with the other 94 per cent or distrust and suspicion enter to weaken the unity of our nation. Such terms as *intellectual snobs, brain trusters,* or *eggheads* suggest the presence of barriers which must be broken down through increased understanding of the communication process. Efficient communication is the very heart of the democratic process.

Within the last decade, research has begun to come to our help. It has brought new methods of teaching reading efficiency to adults faced with the problem of keeping up professionally in a print-filled world. It is providing needed help in improving listening efficiency for better human relations as well as better learning. Even writing and speaking as they relate to professional status, are being re-examined in an attempt to affect the entire 70 per cent of our communication time. The result should be of benefit to the individual and community as well as to the nation and the world at large.

James I. Brown
Professor of Rhetoric

TUBERCULOSIS CONTROL IN THE SCHOOLS OF MINNESOTA

The Committee on Tuberculosis of the American School Health Association is composed of twelve physicians located in eight states across the nation. The main project of this committee consists of certifying schools with reference to tuberculosis control work in progress. The qualifications for certification consist of testing with tuberculin at least 95 per cent of the children and 100 per cent of the personnel members, including teachers, bus drivers, cooks, and others. All adult reactors, including high school students and personnel members, must have x-ray film inspection of the chest, and those who present shadows which might be due to tuberculosis are completely examined. Those found to have active disease are promptly placed under management.

When children are found to react to tuberculin, an attempt is made to find responsible adult associates as soon as possible.

First in a series of editorials on various forms of communication.

This committee chose Minnesota as the demonstration state to determine the feasibility and effectiveness of such a program. A Minnesota sub-committee consisting of three physicians was appointed, and this committee, working in close co-operation with the state tuberculosis and health association, began work in the early 1940's. The schools of Northfield were the first to qualify and were officially certified in 1945. To date, more than 2300 certificates have been issued to Minnesota schools, and a large number in various parts of the state are about to qualify. It now appears that within the next two or three years every school in Minnesota will have a certificate displayed on its walls. These certificates are signed by the president and executive secretary of the American School Health Association, the chairman of the Minnesota state sub-committee, and the executive secretary of the state tuberculosis and health association.

In Minnesota, there have been numerous instances of teachers, bus drivers and other members of personnel, as well as high-school students, developing contagious tuberculosis without their knowledge and disseminating tubercle bacilli to large numbers of children and personnel members. The certification of schools program spells the doom of such occurrences.

Not infrequently, when this project is started in the school, so much interest has been manifested that it has extended to persons of all ages in entire communities. Usually, local physicians have participated in administering tuberculin tests, making x-ray film inspections of the reactors, and completing examinations when indicated. After the project is completed, many persons later report to their private physicians for periodic tuberculin tests, if previously negative, and periodic x-ray film inspection of the chest, if positive.

This project has been so effective that it has spread to other states. It is now in operation in both Dakotas, Iowa, Illinois, Missouri, and Wyoming. Plans are being made for its adoption in a number of other states.

Here is an opportunity for physicians everywhere to participate in a program that will not only hasten the eradication of tuberculosis in their communities, but also will increase public confidence in them.

J. Arthur Myers, M.D.

THE FAMILY AGENCY AND MARITAL COUNSELLING

Marital counselling has been one of the primary functions of the private family agency since the beginning of family casework. We prefer, however, not to use the word "counselling," because it implies that the social worker gives advice. We know that telling a couple what to do when the interaction between the two has reached a high emotional state rarely relieves the situation. Therefore, we have developed techniques of working with people so that they learn to understand themselves better in relationship to their marital partners and to their children. We use the term "family casework," because conflict between a husband and wife has its effect on the children. Sometimes a marital relationship goes along quite well until children are born and the responsibilities placed on both partners becomes greater. This added responsibility, with certain combinations of personalities, throws the interaction between husband and wife out of balance, and in extreme cases the wife may even have a postpartem psychosis.

In our work with families, we think in terms of the "alcoholic marriage." Case examples show that certain types of women tend to marry the men who drink to the extent that they may be classified as "alcoholics." The woman's personality is such that while she is concerned about her husband's excessive drinking which deprives the family of material things, she at the same time handles her husband in such a way that his drinking becomes worse. She seems to have an unconscious need to have an alcoholic husband.

We diagnose our family situations in terms of typical axes, around which family relationships revolve. The Community Research Associates, Inc., sponsored a pilot study in Saint Paul, Minnesota, using Family Service case records. The study took place in 1952 and is the beginning of a classification framework for family-oriented social diagnosis. The second phase of the Classification Project is going on at the present time. Seven family agencies, including Family Service of Saint Paul, are taking part in this research project.

We have learned from our large body of knowledge that certain types of men tend to marry certain types of women, and vice versa. With

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Fourth in a series of editorials on Family Service.

few exceptions, most marriages go along quite well until some crisis arises. With case-work help, many marriage relationships can be put back in balance. Some marriage relationships, when diagnosed, show that in reality the relationship was never a "marriage." When a marriage does end in divorce, each partner needs help in seeing what his part in the relationship was. Each man and woman tends to seek in the second marriage the same kind of personality that he sought in the first marriage. This is why we often see the same man or woman entering into more than one marriage, and with the relationship ending each time in divorce.

To date, we have tentatively formulated ten relationships. They are:

1. A dual immature dependency axis.
2. A woman—oriented anxiety axis.
3. A woman—dominated dependency axis.
4. A woman—dominated competitive axis.
5. A man-dominated competitive axis.
6. A man—oriented, self-depreciating axis.
7. A woman-oriented, self-depreciating axis.
8. A dual emotionally detached axis.
9. A man—dominated hostile axis.
10. A woman-dominated hostile axis.

These ten relationships were formulated from studying 100 families. We are assuming that there are more, and continued research is being carried out to determine whether other classifications of emotional axes and personality types may be found among families with problems. These ten descriptions are not inclusive in themselves. They are descriptive headings we are using to distinguish the relationships. The classification is made after detailed study of the personalities that make up the axis.

Case-work has developed methods of treatment in dealing with people who are having difficulty in their relationships. Their methods vary from the manipulation of the environment when the crisis that upset the marriage is primarily an environmental factor to the method of "clarification" which helps the man and woman have better understanding of themselves in relationship to each other. Each family situation may need not only one but a combination of two or more treatment methods in dealing with the problems of marriage.

(Mrs.) Lois Hoffman
Director of Case Work Services

SUBURBAN MEDICAL MIGRATION

Ever since man began practicing medicine, people have been "going to the doctor." But times have changed. Today, doctors are "going to the people."

With America's population steadily increasing, practically every major city is experiencing residential expansion. And much of this expansion is taking place in the suburban areas, as new families overflow city boundaries. As a result, physicians are locating where the people live—with offices in outlying business districts, and in more recent years, in or near major shopping centers.

An informal study of the location of physicians' offices in the city of Saint Paul, for example, shows that there are actually more general practitioners located in outlying areas than in the downtown "loop" area. A total of 171 have outlying offices, while 140 maintain downtown offices.

Specialists, however, still seem to prefer downtown. In Saint Paul, almost all of them—139 to be exact—have downtown offices. The few specialists who have offices in the outlying areas are principally pediatricians or obstetricians.

Outside of the fact that many hospitals—especially the newer ones—are located in residential areas, there are other reasons why physicians are locating in or near the major shopping centers. Convenience for the patient is probably most important. The young mother with three or four small children certainly finds it easier to get to her physician's office if it is located at or near where she does most of her shopping. She doesn't have to "dress up" as she does when going downtown. If she doesn't have the family car during the day, she can quite often walk to her physician's office. If she does have to use public transportation, the ride is usually quite short. And, certainly, an outlying office offers many conveniences for the elderly patient.

Another factor to consider is that many shopping centers now have nurseries where the mother can leave her children while she shops. This same nursery could be utilized by the mother when she visits the physician.

Another reason shopping centers are attracting physicians is that shopping centers of the future will be more than groups of stores. They are developing into civic and cultural centers with additional facilities for entertainment, recreation and other activities.

Although the physician is not primarily concerned with the "business prospects" of a shopping center, it should be noted that many of the smaller centers are doomed to failure, because for want of careful planning they are so close to each other that they are in competition. So in contemplating the location of an office in a shopping center, careful attention should be paid as to whether or not the shopping center will attract the large numbers of people it is supposed to serve.

It is also entirely possible that the physician locating his office in or near a shopping center will be placing even more stress on evening hours, to conform with the shopping habits of the people living in the area. Many large centers are open three nights a week, others as many as five nights. The reason for this is that shopping is becoming more of a family affair, and evening is about the only time Dad is free. Thus, evening hours might be more convenient for more people.

Another possible future development is that shopping center planners will be including a "Medical Center" as part of their group of buildings, with street-level entrances. At the present time, physicians are using mostly second-floor locations.

If our present population trend continues—and there seems to be every reason that it will—both the new physician and the established practitioner would do well to investigate the many advantages they could offer their patients by locating in the outlying areas.

If the developers and investors in the outlying areas do not provide suitable office facilities, then, of course, it becomes necessary for the physician to consider the construction of his own facilities.

Because, more and more, the doctors are "going to the people."

A. E. Rehnberg
Saint Paul Realtor

A TREND OR MORE?

What assembled criteria would constitute a trend, we don't know. We also don't know the noun of assembly (you remember the *herd* of sheep, the *pride* of lions, the *gaggle* of geese—and the *carton* of Camels) that would be applied to such criteria. However, when a series of related events come on us in a short space of time, we think more than coincidence is at work. For example, we can't explain, with or without statistics, these conjunctions except as at least a trend:

First, we ran into a statistical summary demonstrating an increased incidence of injuries from home tools, over weekends—to folks who were doing-it-themselves.

Second, we received a letter from a colleague who gave us advice about which tools and techniques to use in refurbishing a basement wall. His final advice was, ". . . and get a good supply of Band-Aids."

Third, we took judicial note of a look-out-for-the-do-it-yourself-accidents in a *New England Journal of Medicine* editorial.

Fourth, ditto for a do-it-yourself cartoon with a *get-well-quick* card display prominent in the foreground.

And last, another colleague showed us the almost healed amputation stump of his left thumb, the result of the ministrations of a power saw and a surgeon, in that order.

Now, what with the winter season well upon us and less chance for venting excess energy out of doors, and with the natural urge to do this or that around the house while weather-bound, or to make this or that present in some spare time, we are impelled to lay these matters before you, and suggest that some folks have spent their technical careers acquiring skills, the exercise of which you are now trying to deny them by doing-it-yourself.

Anybody know where we could get a ¼-inch power drill complete with 1,001 attachments? Cheap?

—H.G.M.

FIFTH PRINTING OF DIABETES PAMPHLET

In Januuary, a reprint of "Diabetes: How to Make It Harmless" will be issued by the Committee on Diabetes of the Minnesota State Medical Association, under the chairmanship of John R. Meade, M.D., Saint Paul. There will be revisions to bring the pamphlet completely up to date.

This pamphlet teaches all that is necessary for a patient to know to enable him to manage his condition safely. Any diabetic may have a copy if he first obtains the approval of his physician. Besides general instructions of value, there are many sample menus.

This is the fifth printing of the pamphlet. It was first published in 1934 by the Committee on Diabetes, MSMA, while Russell M. Wilder, M.D., Rochester, was chairman.

President's Letter

NO TIME FOR LITTLENESS

With the approach of the Christmas season and its age-old theme of "Peace on Earth, Good Will Toward Men," it is well that we gain new faith and encouragement in the furtherance of the tenets of our profession. Why? Because the message of the Christmas season allows no place for petty bickering, selfishness, personal jealousy, nor minority pressure groups. Our profession and the medical organizations are dedicated to the furtherance of the health of the people of our community, and anything that interferes with this objective is directly contrary to the spirit of Christmas. Good will toward all men is a challenge as well as a responsibility of our professional organization.

It has never ceased to be a source of amazement to me that small groups of our fellow citizens will seek to impose doctrines and procedures upon their fellow men by any means whatsoever, even though history, experience, common sense and professional expert knowledge all point to the fact that they are against the public interest. Is it because of ignorance, willful or otherwise? Is it because of a thirst for power, or personal aggrandizement? Is it for financial gain, or is it because, in some distorted minds, certain persons feel superior to their fellows and seek to impose their will and regulatory way of life upon them? The answer probably is a combination of all of these reasons. They are little men, small in spiritual stature. They have deviated far from the teachings of the Great Master, whose birthday we shall soon celebrate.

December also brings to a close my year as president of the Minnesota State Medical Association. It is with a sense of inadequate accomplishment and frustration that I leave this office, to which you elected me eighteen months ago.

I am grateful for the honor which you have given me, and assure you that it has been a privilege to work with the other officers, council, and executive officers of our association. They have all been most helpful, and especially is it true of the State Association office staff. To my own professional associates and staff, I am especially grateful for their understanding and aid in furthering the work of our organization.

I am sure that each president hopes that he has left some small imprint of his personality and ability upon the state organization. In the past, we have never suffered from lack of leadership, and I am sure that the future will enable dedicated men to serve the association and our profession with the same zeal that has been exhibited by my predecessors.

The work of our organization is never-ending. The "little men" and the minority pressure groups are forever seeking to hamper the work and the accomplishment of the objectives to which our association is dedicated. Eternal vigilance is the price of not only liberty but of progress, in attaining the ends toward which we all strive. The endless battle should not be a source of discouragement, but rather a challenge for us never to compromise with principle, never to succumb to the "little man's" level of personal or professional selfishness. In other words, inculcate forever into our work the Christmas spirit of "Good Will Toward Men."

It is my personal wish that each of you and your loved ones have a blessed Christmas season.

Arnold O. Swenson

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

COUNTY SOCIETY STUDIES DOCTORS' ESTATES

Revealing facts are seen in a recent small, but meaningful survey of obituaries of physicians, made by the Hartford County (Connecticut) Medical Society.

The survey showed that one out of eight of the physicians who died between 1940 and 1953 was in debt at the time of death. The survey is reported by a recent AMA Secretary's Letter, noting that the number of deaths studied was 144.

Also, of the estates studied, one out of three left net assets of less than $10,000.

According to the report, "The Hartford survey disclosed only one extremely wealthy doctor out of the 144 and that $575,915 of his estate was consumed by estate taxes and other settlement expenses. Only one doctor in eight survived his wife."

Death Rate High

The report also showed that deaths among doctors aged forty to fifty occurred in a ratio of 2:1 compared with the general population, and in the sixty to seventy age bracket, the doctors' death rate was 50 per cent higher than that given for the same ages in the insurance table. As would be expected, heart disease and cerebral hemorrhage were the chief causes of death.

The age of death of the physicians, when compared with life insurance mortality tables, showed that there were two vulnerable age periods for medical men—forty to fifty and sixty to seventy.

MEDICAL COSTS SURVEY NOTES TRENDS

Only 26 per cent of the adult population believe that the cost of medical care in America is much too high. This figure is reported in a recent survey of opinions and attitudes toward medical care just released to the pharmaceutical industry by the Health Information Foundation of New York. Other results show that 40 per cent of the adult population believe food costs are much too high; 45 per cent believe repair charges (TV, auto, et cetera) are excessive; 27 per cent are equally critical of clothing costs.

The survey notes:

> "While medical costs in general come in for less criticism than other elements of the cost of living, within the category of medical costs, the percentage believing costs 'much too high' for doctors' fees is 16 per cent; hospital charges, 39 per cent; dentists' fees, 24 per cent; prescriptions at drug stores, 38 per cent."

Another interesting fact is that 56 per cent of the population cannot give the name of one company that, they believe, manufactures a new or "wonder drug."

When asked the question, "What are some of the things that make it easier to have good health today than it was thirty years ago?", 71 per cent cited improvements in medical knowledge and facilities, and almost half the population (47 per cent) specifically referred to new drugs, medicines, and vaccines now available.

Prescription Costs

Further information shows that 38 per cent of the population believe that cost of prescriptions is much too high, and 28 per cent believe that costs are somewhat higher than they should be. The report states:

> "Of those giving reasons for high cost in this category, less than half attach blame to anyone. Twenty-six per cent of those who complain of prescription costs blame the druggists, 10 per cent mention physicians and 7 per cent pharmaceutical manufacturers. An additional 16 per cent blame some unspecified 'they.' Some individuals mention more than one person or group."

This question was also asked: "What people or groups do you think have been mainly responsible for these new 'wonder' drugs?" The answer:

> ". . . only 11 per cent of the respondents give specific credit to the drug companies, pharmaceutical houses, the chemical industry, or drug manufacturers; an additional 9 per cent mention 'laboratories'; 23 per cent credit doctors, the American Medical Association, or groups of doctors; 40 per cent credit scientists, science, research, or persons such as medical researchers and chemists."

GRADUATE MEDICAL EDUCATION SHOWS BIG GAINS

According to a recent news release from the Council on Medical Education and Hospitals of the AMA, "training programs for newly-graduated doctors have become as big—in terms of enrollment and time spent—as basic medical school education." It is believed that this situation is unique among the professions, the news release stated.

The release notes that enrollment of young physician graduates as interns and residents for the 1954-55 year passed that of students in undergraduate medical schools. It also notes that this is an indication of "the magnitude of the growth of graduate training over the past ten years."

Figures Tabulated

The Council's annual report on internships and residencies showed 9,066 graduates serving internships for 1954-55 and 20,494 serving as residents, a total of 29,560 in 1,364 hospitals. This is an increase of about 2,500 over last year. Internships offered show for 1954-55 a 250 per cent increase over the number reported in 1914, the first year approved hospitals were listed by the council.

Trend Reversed

The Council reports that "this year's internship and residency figures reverse the trend of the past several years, when unfilled positions were increasing." The report showed foreign medical school graduates in 1954-55 filling almost half of the vacancies not taken by American graduates, thus alleviating partly the demand for hospital staff appointees.

Residency Training

Another important change reported by the Council was in requirements for residency training, which follows intenship as the next step after medical school graduation. The Council reported that previously some programs offered specialist certification training which almost could be compared to allowing a medical student to receive his M.D. degree after "completing four successive years at the freshman level at four different medical schools." The new policy eliminates the possibility of taking specialty training in several separate programs and instead requires stricter continuity in an integrated training course.

Cash Stipend Higher

The Council's report stated that the average cash stipend paid to interns by hospitals affiliated with medical schools was $87 a month compared to $84 a month last year. Hospitals not affiliated with schools pay $136 a month on the average, an increase of $1 over last year. The report also said that figures indicate that the amount of stipend, as usual, has no effect on the prospective intern's choice of a hospital for training.

"Federal hospitals, such as armed services of VA institutions, and local governmental hospitals," the report concluded, "offer 36.5 per cent of all available intern positions and have the highest rate of filled positions. Their rates range from 100 per cent for the Public Health Service, 99 per cent for Army hospitals, and 97 per cent for Navy. County and city-county hospitals have rates of between 93 and 96 per cent."

SUGGESTIONS ON JOINT COMMISSION ON ACCREDITATION WANTED

At the American Medical Association convention last June, a committee was appointed to study the functions of the Joint Commission on Accreditation of Hospitals. The committee is now asking physicians for any observations or suggestions concerning the work of the Joint Commission.

The committee is interested especially in the following:

1. The general understanding by physicians of the functions of the Joint Commsision.
2. Whether the method of appeal from an adverse ruling regarding accreditation is satisfactory.
3. The effect on the individual physician's hospital connections due to actions of the Joint Commission.
4. Whether any organizations not now represented should have official representation on the Joint Commission.
5. The effect of the Joint Commission's requirements concerning such matters as staff meetings.
6. The pros and cons of separating administrative and professional accreditation functions in the inspection of hospitals.
7. Constructive suggestions for improving the hospital accreditation program.

Any comments from individual members or state and county medical societies should be addressed to: W. C. Stover, M.D., Chairman, Committee to Review Functions of Joint Commission on Accreditation of Hospitals, 535 North Dearborn Street, Chicago 10, Illinois.

Committee members will report on their findings at the next American Medical Association meeting. It is important to co-operate in this effort if the Joint Commission is to function usefully.

SOCIAL SECURITY, OASI FIGURES NOTED

Latest information from the Social Security Administration is reported in a recent AMA Washington Letter, and reveals that the proportion of people receiving old-age assistance payments (based on need) has declined 20 per cent in the last five years, but because states are increasing the amount of individual payments, the total expenditure has increased. "At the same time," the report says, "figures released by the agency indicate that in the same period the proportion of old people receiving old age and survivors payments has increased by about 150 per cent."

The report quotes the administration's comment on the drop in numbers receiving old-age assistance:

> "The economic prosperity of the nation is reflected to some extent in this decline. Another major reason is the continued growth in the number of aged persons who now receive benefits under the old-age and survivors insurance program."

Further explanation is:

> "The 150 per cent increase in proportion of OASI recipients is explained only in small part by shift of elderly assistance cases to the pension rolls; most of the increase was caused by congressional action in extending coverage to additional occupations and making it increasingly easy for older persons to qualify for pensions."

Population Noted

The report says that in June, 1950, the aged population (sixty-five and over) numbered 12,399,000, of whom 2,786,690 (225 of every 1,000) were receiving old-age assistance. Latest data (June, 1955) show in the sixty-five-and-over age group a population of 14,244,500, of whom 2,544,496 (179 in every 1,000) were receiving old-age assistance.

In June, 1950, approximately 169 in every 1,000 aged persons (about 2,094,000) were receiving OASI pensions. Five years later, 423 in every 1,000 (6,025,000) were receiving OASI pensions, a proportional increase of about 150 per cent. The month of June is used by the Social Security Administration to demonstrate what has been happening to the old-age assistance program. Five years ago, payments for that month totaled $122,350,000 (an average of $43.85 to the 2,790,000 recipients). Last June, the rolls had dropped to 2,549,000, but total payments had increased to $133,297,000 (an average of $52.30 per recipient).

New York Medicine Says:

> "It would cost an extra $2,000,000,000 per year for the next twenty years to implement H.R. 7225 and then $2,500,000,000 a year thereafter. To finance the gigantic increase, Social Security taxes over twenty years would increase from the present 2 per cent to 9 per cent . . . a jump of four and one half times.
>
> "In deciding to fight H.R. 7225 the Board of Trustees of the AMA have made a major change of policy. The AMA, it may be recalled, has never fought the Social Security laws as such but on the point that physicians should not be included in Social Security coverage of the self-employed. Now it will attack a proposed section of the law which applies . . . not to physicians alone but . . . to the rest of the population."

FOLSOM ASKS MORE FUNDS FOR MEDICAL RESEARCH

Secretary Folsom, HEW, favors more funds for medical research in crippling and fatal diseases. His address of welcome at the third annual Symposium on Antibiotics is reported in a recent Washington Letter of the AMA.

Mr. Folsom noted that Congress approved this year more than $97 million to investigate causes and treatment of major illnesses, or about twelve times the amount spent in 1946.

He stated: "In addition to these government expenditures, the pharmaceutical industry and many other private groups spend many millions of dollars for resarch yearly. We must seriously consider making even more funds available for medical research to bring even greater benefits to humanity."

Mr. Folsom described progress in antibiotic therapy during the past thirteen years as phenomenal. The antibiotic industry, non-existent in 1942, now has a capital worth of over $1 billion; antibiotics, once produced in milligram quantities, are now measured in tons, and untold numbers of people are alive today because of the drugs, he said.

MINNESOTA GRADUATES IN PUBLIC HEALTH WORK

I have just returned from the Eighty-fifth Annual Meeting of the American Public Health Association in Kansas City, and I want to share with the physicians of Minnesota my great sense of pride and satisfaction in the prominent and distiguished part that Minnesotans played in this meeting.

In the first place, the President of the Association, who presided at the meeting, was Dr. Herman E. Hilleboe, M.D., University of Minnesota, 1931, Health Commissioner of New York State. The President for next year is Dr. Ira Hiscock of Yale, and chosen as the new President-elect for 1957 is Dr. John W. Knutson, D.D.S., University of Minnesota, 1931. Incidentally, Doctor Knutson becomes the fifth out of seven consecutive presidents of the American Public Health Association whom we can claim as a Minnesotan. The first was Dr. William P. Shepard, M.D., University of Minnesota, 1922, and M.A. in 1924, President of the American Public Health Association in 1951 and currently Second Vice President of the Metropolitan Life Insurance Company; the second, was Dr. Gaylord W. Anderson, Director of our University of Minnesota School of Public Health, President of the American Public Health Association in 1952; the third was Dr. W. L. Halverson, born and raised in Litchfield, Minnesota, Commissioner of Health of California, and American Public Health Association President in 1953; the fourth was the current President, Dr. Herman E. Hilleboe; and the fifth, the new President-elect, Dr. John W. Knutson.

Several of the most distinguished awards in medicine and public health were conferred upon Minnesotans at Kansas City: Dr. Albert J. Chesley, whose name is synonymous with public health in Minnesota, was given posthumously the Sedgwick Medal for "distinguished service in public health." This was accepted by Dr. Robert N. Barr, Doctor Chesley's successor as Executive Secretary of the Minnesota State Board of Health.

Another award conferred upon Minnesotans at this meeting was the Albert Lasker Award for Medical Research, which was given to Dr. C. Walton Lillehei, Dr. Richard L. Varco, Dr. Morley Cohen, and Dr. Herbert Warden, all members of our Department of Surgery, for their "brilliant and imaginative studies which have clarified many of the problems of surgery within the heart."

Three former Minnesotans, Mrs. Lucile Petry Leone, Miss Pearl McIver, and Miss Margaret Arnstein, all of whom entered the United States Public Health Service from the University of Minnesota, also received a Lasker Award for distinguished leadership in the development of public health nursing services which "have significantly furthered the interests of public health and made a notable contribution to the advancement of the well-being of this country."

Within the Laboratory Section of the Association, the annual Kimble Award for outstanding contribution to the development of laboratory procedures was presented to Dr. Paul W. Kabler, M.D., University of Minnesota, 1938.

Another prominent Minnesotan, Senator Hubert H. Humphrey, spoke on "The United States and International Health" at a large luncheon meeting arranged by the National Citizens Committee for the World Health Organization. At this same meeting, it was my privilege to report on the Eighth World Health Assembly which was held in Mexico City last May. Also, at this meeting, Dr. James E. Perkins, M.D., University of Minnesota, 1930, Managing Director of the National Tuberculosis Association, spoke on "World Health Goals in Tuberculosis Control."

In addition to the above, many other Minnesotans made prominent contributions to the meeting as speakers on the program, as officers of sections, and chairmen or members of important committees. This was truly an important national meeting in which Minnesotans can take real and justifiable pride.

Harold S. Diehl, M.D.
Dean of Medical Sciences, University of Minnesota

In Memoriam

ALBERT JUSTUS CHESLEY

Dr. Albert J. Chesley, secretary and executive officer of the Minnesota State Board of Health, died in St. Mary's Hospital, Rochester, October 17, 1955. Born in Minneapolis in 1877, he spent a lifetime working in and for the State of Minnesota.

Dr. Chesley was a life member of the Hennepin County Medical Society and the Minnesota State Medical Association. He was a fellow of the American Medical Association and a member of the Joint Committee on Health Problems in Education. He was also a member of the Association of Military Surgeons of the United States, the American Epidemiological Society, on the board of directors of the American Child Health Association, an honorary life member of the American Social Hygiene Association. He was an honorary fellow of Britain's Royal Sanitary Institute and worked with many other groups, including the National Society for the Prevention of Blindness, the Veterans of Foreign Wars, the Order of Masons and Nu Sigma Nu fraternity.

Among many honors, Dr. Chesley was holder of the Outstanding Achievement Medal of the University of Minnesota, and the Distinguished Service Medal of the Minnesota State Medical Association.

He is survived by his wife; a daughter, Mrs. Paul Miller of Minneapolis; and two grandchildren.

Following are excerpts from the story on Dr. Chesley in *Minnesota's Health,* montly publication of the State Department of Health, November, 1955.

Dr. Chesley was appointed secretary and executive officer of the board on May 13, 1921, and in all that time he had attended every meeting of the board. Active almost to the end, the way he would have wanted it, he died October 17 at age seventy-eight.

At the outbreak of the Spanish-American War, Dr. Chesley added a year to his age to enlist as a private in the medical corps of the U. S. Army, and was sent to the Philippines. There he served as a medical aide for the wounded in the field and volunteered for duty in special hospitals, particularly those for smallpox and Hansen's disease.

It was on his return from the Philippines that he began his long public health career in Minnesota. He started in 1901 as a clerk in the state health department, and worked part time as a laboratory assistant while completing his medical education at the University of Minnesota. When he received his medical degree in 1907 he was appointed an assistant bacteriologist.

He is believed to have had the longest continuous record of public health work in the nation. With the exception of the period from May 1918 to October 1920, he served his entire public health career in his native state. When World War I broke out he was held in the United States as long as he would submit to it. He reached Europe as a public health expert for the American Red Cross, Commission to France, and later was chief of staff, American Red Cross, Commission to Poland. It was overseas that he met and married his wife, then Dr. Placida Gardner, a member of his staff.

Dr. Chesley had become director of the division of preventable diseases by the time he went overseas. Shortly after his return from Poland, Dr. Chesley found himself acting secretary and executive officer. Upon the death of Dr. Smith, then executive officer, Dr. Chesley, aged forty-four, succeeded to the office and became the state's fourth health officer, on May 3, 1921.

Dr. Chesley was always true to the obligation he had assumed to protect and preserve the health of the people of Minnesota. In his lifetime he had lived through some of the great epidemics, had seen many of these diseases brought under control, and he himself had played an active part in the battle against them. Changing conditions brought new health problems. Work ceased in some areas and expanded in other directions as advances were made in the knowledge of how to combat disease. To each problem and to each advance, Dr. Chesley gave the benefit of his knowledge, his time, and his sincere interest. At the same time he was quick to point out that the only "new" thing about many of the so-called new problems was a willingness to do something about them.

Lack of funds was an obstacle but it did not deter him. The first legislative appropriation for the department had been in the amount of $500. He had worked with men who got things done in spite of budget limitations. On more than one occasion in the early days he forfeited his own modest salary to keep a program going where he felt there was a need but for which funds were not available.

Most of the recommendations made in his first biennial report have been carried out, although some of them were slow in coming. As the result of the tuberculosis control program, in which he maintained a continuous interest, he saw the tuberculosis death rate fall from 69.95 per 100,000 in 1922, with 1,726 deaths, to a rate of 4.4 with only 138 deaths in 1954.

A section of industrial health was set up in the health department in 1943 after the adoption of a general code for environmental sanitation by the State Industrial Commission. The section of water pollution control was organized in 1945 following the enactment of the State Water Pollution Control Act. By law, the secretary and executive officer of the state board of health is secretary of that commission and the technical aspects of its work are carried out by the health department.

Dr. Chesley had a real love for children and a deep concern for their health. The results of his program of maternal and child health are indicated by the current infant and maternal mortality rates. In 1954, it was calculated that 419 Minnesota mothers and 5,650 infants lived that year who would have died had 191 mortality rates prevailed.

"Children never had a better friend nor did the Chil dren's Bureau," wrote Dr. Martha M. Eliot, chief of the Children's Bureau, on his death. In the words of Katharine F. Lenroot, former chief of the bureau, he was "a great pioneer in child health as well as general health. I always take pride especially in his service to Indian children."

One of the problems of deepest personal concern to Dr. Chesley was the improvement of health services for Indians. His ingenuity in bringing the subject of Indian health into almost any public or private health discussion was incredible. Visitors could almost measure the degree of welcome he extended to them by the length of time that passed before he made some reference to the Indians. Many of his Indian friends, knowing his concern for them and his love of children, named their sons, "Chesley" and he was proud of it.

The record is impressive. Minnesota was the first state to employ an Indian public health nurse on a reservation. In 1924 an old frame Indian school by Leech Lake was converted into the Onigum Tuberculosis Sanatorium. The state held its first general Indian health clinic in 1929 at which over 700 Chippewas who had come to harvest wild rice received physical examinations. In 1936, one year after the opening of the 125-bed Chippewa wing at the State Sanatorium, the tuberculosis death rates per 100,000 among Minnesota Indians was 385. For Dr. Chesley, 1954 was memorable—for the first time on record in Minnesota, no Indian deaths from tuberculosis were reported. The transfer this year of Indian health and medical care from the Bureau of Indian Affairs to the Public Health Service was almost entirely due to his efforts.

He saw the need for public health nurses with special training to fit them for their work with families. As early as 1917, when he was director of the division of preventable diseases, he claimed that "such nurses should themselves be teachers." Throughout his career he worked for the development of good public health nursing services for all parts of the state.

Dr. Robert N. Barr, who succeeds Dr. Chesley as secretary and executive officer, was one of his "boys." There are at least fifty health officers and others holding important posts in public health throughout the country who can proudly make that claim.

In 1937, Dr. Chesley initiated the first field training course in the middlewest for health officers, a course that was a forerunner of the school of public health at the University of Minnesota. He himself was a professor of public health in the department of preventive medicine at the University for 20 years, 1925-1945.

Dr. Chesley took both professional and personal pride in the outstanding research and training programs of the University of Minnesota medical school. Dr. Harold S. Diehl, dean of the school, had been one of the "bright young men" on his staff in Poland. Dr. Chesley's leadership, interest, and support of the program in public health at the University was a prime factor in the establishment of the school of public health. To perpetuate that personal interest and concern, friends are now contributing to what is to be the Albert J. Chesley memorial fund for a lectureship in public health, to be handled through the Greater University Fund.

Although he did his major work in Minnesota, Dr. Chesley belonged as much to the nation as to the state. When he received the distinguished service medal of the Minnesota State Medical Association in 1948, it was for "outstanding service in the field of public health and social hygiene, not only in Minnesota but elsewhere in the United States and abroad; and for his rare insight and fine understanding of the private practice of medicine to which, as one of its most ardent associates, he has given unfailing co-operation at all times."

Nearly every honor that exists in public health had been awarded to Dr. Chesley. The last was announced at the eighty-third annual meeting of the American Public Health Association when he was named the recipient of the annual Sedgwick Memorial Medal. The occasion was to have been "a special opportunity for recognition of our admired friend," wrote Dr. Reginald M. Atwater, executive secretary of the association of which Dr. Chesley had been president in 1930.

The last tribute paid to him during his lifetime came from the state in which he was born on September 12, 1877, from which he had received his education, and in which he had done his work. Only four days before his death, he was elected an honorary member of the Wabasha County Medical Society at its eighty-seventh annual meeting.

He was president of the State and Provincial Health Authorities from 1924 to 1927 and secretary from 1927 to 1945, when the group finally accepted the resignation he submitted annually.

He was equally active in the Association of State and Territorial Health Officers. "The meetings will never be the same without this wonderful friend and public servant," wrote Dr. Bruce Underwood, commissioner of health in Kentucky.

Only one who had lived public health with him could list the many accomplishments in health fields to which he gave leadership, guidance, and assistance but for which he gave the credit to others. He was a modest man and preferred not to be in the limelight. He had a terrific loyalty to his staff.

The lights burned at all hours in his office in the health department. He did not spare himself and it was hard for him to understand why others were not at their desks on holidays or over week-ends. Time was of no moment to him. Those who knew him best can recall few occasions when he took a vacation.

The long hours he spent on his job kept him in touch with what was happening in public health. Up to the very last, he knew everything that was going on in the building, in the state, and in the nation. He was a crusader and a fighter who held fast for the things in which he believed. Those who knew him best also remember his warmth and gentleness, his quiet sense of humor, and his many, many thoughtful deeds.

"Minnesota stands out among all the states in the country for its progress in public health and largely

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Reports and Announcements

MEDICAL MEETINGS

National

Congress on Industrial Health, 16th annual meeting, Sheraton-Cadillac Hotel, Detroit, Michigan, January 23-24, 1956.

American College of Surgeons, sectional meeting, Hotel Schroeder, Milwaukee, Wisconsin, February 27-29, 1956. Dr. Forrester W. Raine, Milwaukee, chairman.

Atlanta Graduate Medical Assembly, Biltmore Hotel, Atlanta, Georgia, February 20-22, 1956.

Mediclinics of Minnesota, postgraduate course **sponsored by Academy** of General Practice of Broward **County, Fort** Lauderdale, Florida, March 5-14, 1956.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination (Part 1), written examination and review of case histories, for all candidates for the American Board of Obstetrics and Gynecology will be held in various cities of the United States and Canada on February 3, 1956.

Case abstracts, totalling twenty, are to be sent by the candidate to the secretary, Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland, Ohio, as soon as possible after receiving notification of eligibility for the Part 1 written examination.

AMERICAN COLLEGE OF SURGEONS

Twelve Minnesota physicians were elected to fellowship in the American College of Surgeons at the annual meeting of the organization in Chicago during the first week of November. The new fellows include Dr. Charles D. Adkins, Dr. Maxwell M. Barr, Dr. Merrill D. Chesler, Dr. Conrad I. Karleen and Dr. D. Keith Millett, all of Minneapolis; Dr. Henry W. Dodge, Jr., Dr. F. Henry Ellis, Jr., and Dr. Edward D. Henderson, all of Rochester; Dr. William C. Downing, Crookston; Dr. Kenneth A. Storsteen, Duluth; Dr. John R. Nickerson, Fairmont, and Dr. Robert P. Meyer, Faribault.

CONTINUATION COURSES

The University of Minnesota announces a continuation course in *Emergency Surgery for General Physicians* which will be presented at the center for Continuation Study from January 30 to February 1, 1956. The management of various types of injuries will be taken up in detail, as will certain other common surgical emergencies. The course will be presented under the direction of Dr. O. H. Wangensteen, professor and chairman, Department of Surgery.

* * *

Neurology will be the subject of a continuation course to be presented by the University of Minnesota at the Center for Continuation Study next February 6 to 10, 1956. Intended primarily for physicians in general practice, the program will have appeal also for neurologists and neurosurgeons. The most commonly seen neurological symptoms and syndromes will be stressed.

Guest faculty will include Dr. William M. Meacham, associate clinical professor of surgery, Vanderbilt University School of Medicine, Nashville; Dr. Morris B. Bender, director, Neurology Service, Mount Sinai Hospital, and professor of clinical neurology, New York University College of Medicine, New York City; Dr. John F. Sullivan, associate professor and head, Department of Neurology, Tufts College Medical School, Boston, and Dr. Oliver H. Lowry, professor and head, Department of Pharmacology, Washington University School of Medicine, St. Louis.

The course will be presented under the direction of Dr. A. B. Baker, professor and director, Division of Neurology, and Dr. William T. Peyton, professor and director, Division of Neurosurgery, University of Minnesota Medical School.

* * *

The University of Minnesota announces a continuation course in *Recent Advances in Internal Medicine for Internists* which will be held at the Center for Continuation Study from February 13 to 15, 1956. This year's program will deal principally with recent advances in the fields of endocrinology and metabolism, renal disease, an cardiology.

Guest speaker will be Dr. Joseph W. Jailer, associate professor, Department of Medicine, College of Physicians and Surgeons, Columbia University, New York City, who will also give the Minnesota Pathological Society Lecture on Tuesday evening, February 14. The course will be presented under the direction of Dr. C. J. Watson, professor and head, Department of Medicine.

* * *

Cancer Detection will be the subject of a continuation course for general physicians which will be presented at the Center for Continuation Study by the University of Minnesota and the Minnesota Division of the American Cancer Society from February 16 to 18, 1956. Cancer detection techniques which can be used effectively in general practice will be stressed. The course will be presented under the direction of Dr. O. H. Wangensteen, professor and chairman, Department of Surgery, and Dr. W. A. Sullivan, director, Cancer Detection Center.

WABASHA COUNTY SOCIETY

At the eighty-seventh annual meeting of the Wabasha County Medical Society at Plainview on October 6, Dr. B. J. Bouquet, Wabasha, was elected president of the organization. He succeeds Dr. E. W. Ellis of Elgin.

Other officers elected were Dr. D. G. Mahle, Plainview, vice president; Dr. B. A. Flesche, Lake City, secretary-treasurer; Dr. E. C. Bayley, Lake City, delegate to the state association, and Dr. Ellis, alternate.

Woman's Auxiliary

STATE AUXILIARY HOLDS FALL MEETING

Mrs. L. J. Leonard, Minneapolis

The pattern of the Fall School of Instruction, held at the Curtis Hotel on Tuesday, October 4, was somewhat different this year. Mrs. H. H. Fesler opened the session with a short business meeting and this was followed by a panel discussion, with Mrs. Peter Rudie as moderator. Members on the panel were the officers and committee chairmen who served last year, and the information and advice they supplied to the new chairmen were invaluable. Mrs. M. I. Hauge spurred each member on to greater achievement by saying, "When you stop being better, you stop being good!"

Mr. Leo Brown, Chicago, Director of the Department of Public Relations, American Medical Association, spoke on the subject, "The Doctor's Wife and Public Relations." Several examples of extremely unfavorable public relations shown by doctors' wives were cited by Mr. Brown, who admitted these were rarities. However, the strongest factor influencing public relations between the doctor and the people in his community is the behavior of the doctor and his wife. He stated that improved patient relationship is the first step toward improved public relations: if improved services are stressed, improved public relations will follow automatically.

He stated that less than 30 per cent of our doctors read the valuable material sent them by the AMA, and suggested that the wives could do some of this reading for their husbands. A new booklet, "To All My Patients," scheduled for mailing to doctors' offices after October 20, Mr. Brown said should be read and not be delegated to the wastebasket. "The Human Side and the Business Side of Medicine" is another AMA pamphlet he recommended.

In closing, Mr. Brown gave the following quotation from Charles Dickens:

> "I never walk out with my husband, but I hear the people bless him. I never go into a house of any degree, but I hear his praises, or see them in grateful eyes. I never lie down at night, but I know that in the course of that day he has alleviated pain and soothed some fellow creature in the time of need. I know that from the beds of those who were past recovery, thanks have often, often gone up, in the last hour, for his patient administration. Is not this to be rich? The people even praise *ME* as the doctor's wife."

Dr. F. W. Behmler, Morris, Minnesota, State Senator and vice president of the Minnesota State Medical Association, gave an inspiring and challenging talk on the dangers lurking ahead through legislative channels: granting osteopaths the right to administer drugs and to perform surgery. He referred to the article on pages 736-742 in the July 2, 1955, issue of *The Journal of the American Medical Association* which gives a clear, concise picture of the situation as it exists today. He advised any women living in communities where the League of Women Voters is an active group, to join them and participate.

The meeting recessed at 12:30. At 1:00 P. M., a delightful luncheon was served in the Cardinal Room. Mrs. Fesler introduced the guests and also the past state presidents who were present. Dr. H. B. Sweetser brought greetings from the State Medical Association.

Mrs. Mason G. Lawson, Little Rock, Arkansas, National President, spoke in a very sincere and informal manner, with all the charm of the South. She stressed the importance of the individual county member, placing her achievement over that of the national member who sets the policy but does not produce the ultimate results. She is much interested in the promotion of the American Medical Education Foundation and described a way in which her own county is helping this cause. Her county auxiliary has had a card printed conveying a Christmas greeting and stating that a contribution to the AMEF has been made in the purchase of the card. This has been done in the belief that the recipient will appreciate the dual purpose which has prompted the sender to use this type of Christmas card.

Mrs. Fesler adjourned the meeting after reading a short poem entitled "It's up to You," which should have inspired everyone there to do her share of auxiliary work. From general comments heard after the session, it was a most successful and worthwhile day, and thanks are due Mrs. Harvey Beek, who had charge of plans.

NATIONAL AUXILIARY HOLDS CONFERENCE

Mrs. H. H. Fesler, State President

Mrs. L. P. Howell, Rochester, president-elect, and your state president attended the 12th Annual Conference of State Presidents, Presidents-elect and National Committee Chairmen, November 1-3, 1955, at the Drake Hotel in Chicago.

The meeting was called to order by the national president, Mrs. Mason G. Lawson. The theme of the conference was "Active Leadership in Community Health." The purpose was to exchange information, to pool resources of Auxiliary experience, and to become better acquainted with one another. Members came seeking new ideas and trading information. This was accomplished by small groups getting together for Dutch treat breakfasts.

The presiding officer of the conference was Mrs. Robert Flanders, national president-elect. Panel discussions were held on the nine various Auxiliary activities, followed by a question-and-answer period. Your president was assigned to the Organization Panel on "What would you do to include future members in our program now?"

Guest speakers from the AMA appeared on several panels giving many helpful suggestions and valuable information. Dr. Elmer Hess, AMA President, was a luncheon guest speaker and gave an inspiring message on our American heritage and freedom of thought and power to vote, which is so often neglected.

Dr. Ernest B. Howard, Assistant Secretary of the AMA, discussed social security disability benefits. He said that House file H.R. 7225 amendment, which will come up in the spring, will involve millions of people and its impact on the medical profession in the future is serious. Private insurance companies are 100 per cent against this bill.

The best investment we can make is in safety precautions. Medical research shows the national pattern of cause of injury; 50 per cent of injuries are due to doors on cars popping open and persons falling out. Safety precautions on cars will not develop until the public demands them.

The American Medical Education Foundation is planning an eighty dimes campaign this spring—a dime for each of our eighty-one medical schools in the nation. All over the country, people are calling for more doctors, more research and more health services, but the medical schools are handicapped by lack of funds. They need help now. Contributing to the national fund is a sound way to keep all the schools with a single gift. The need to keep our medical schools free, solvent and progressive, is $10,000,000 a year.

The final day of the conference, a tour of the AMA, headquarters was held. The program included greetings from Dr. George F. Lull, Secretary and General Manager, and a talk by Dr. Austin Smith, chairman of U. S. Committee, World Medical Association. Dr. Smith said that in the past Europe was the medical center; now the trend is reversed and people are flocking to our country for information and training.

Four excellent health movies were shown, followed by a luncheon at the AMA headquarters, and adjournment.

The reports in full will be printed in the January issue of *The Bulletin*. Please don't fail to read them. It was a most successful and inspiring conference.

IN MEMORIAM

ALBERT JUSTUS CHESLEY

(Continued from Page 949)

because of his work. His old friends will remember his wisdom, his dedication, and his fine kindness and gaiety as long as they live," wrote R. R. Rosell, executive secretary of the Minnesota State Medical Association.

It is hard to remember how young the state health department is in terms of history or how old it is in terms of public health. It is even harder to believe that one man, in his life span, has personally experienced so much of it. The words of Dr. Hewitt, when he summed up his own career in public health in the annual report of the Minnesota State Board of Health for 1896 apply equally to Dr. Chesley: "The best years of my life and effort have gone into this work. I have spared neither time, labor, nor thought, to make it what it ought to be. Such as it is, the record is made and closed." Dr. Chesley gave his entire life to public health. Few men have done so much so unselfishly for his fellow men.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

MINNEAPOLIS MIDWIFE SENTENCED FOR WILLFULLY MAKING FALSE BIRTH CERTIFICATE

Re: State of Minnesota vs. Lillian Twedt

On November 14, 1955, the above-named defendant, sixty-seven, 2246 Benjamin Street, N.E., Minneapolis, a licensed midwife, was sentenced by the Hon. Irving R. Brand, Judge of the District Court of Hennepin County, to a term of six months in the Minneapolis Women's Detention Home on a charge of willfully making a false birth certificate. However, Judge Brand stayed sentence for a period of one year and placed the defendant on probation during that period of time. Mrs. Twedt had originally been charged with a felony, offering false instruments for filing or record, to which she had entered a plea of not guilty in Hennepin County District Court on September 20, 1955, but the charge against her was later reduced to a misdemeanor, the crime of willfully making a false birth certificate, to which she entered a plea of guilty on October 3, 1955 before Judge Brand. At that time, Mrs. Twedt surrendered her midwifery license in open court and also signed an agreement with the Minnesota State Board of Medical Examiners authorizing the Board to permanently cancel her license. She also agreed not to make application to the Board in the future for a license to practice midwifery.

The criminal charge against Mrs. Twedt was based upon a birth certificate which was filed by her in connection with the birth of a baby on March 7,1955, to an unmarried Minneapolis mother, the defendant having been in attendance at the birth. The birth certificate, which was filed with the Clerk of District Court of Anoka County, Minnesota, contained false information in reference to the place of birth, the names of the parents and other data concerning them, and also in regard to the legitimacy of the child. After the birth of the baby, it was placed in the Richfield, Minnesota, home of the persons who were listed on the birth certificate by Mrs. Twedt as being the father and mother of the child.

Mrs. Twedt, who was born in Saint Paul, Minnesota, on March 4, 1888, was licensed to practice midwifery in Minnesota on March 1, 1932. She holds a diploma dated June 3, 1928, from the Chicago School of Practical Nursing. The Minnesota State Board of Medical Examiners on November 5, 1955, made an order revoking the license to practice midwifery held by Mrs. Twedt.

The best thing to give your enemy is foregiveness; to an opponent, tolerance; to a friend, your heart; to your child, a good example; to a father, deference; to your mother, conduct that will make her proud of you; to yourself, respect; to all men, charity.

—Balfour

General Interest

A drive for funds for a million-dollar **Masonic Memorial Cancer Hospital** at the University of Minnesota was launched by 68,000 Masons on November 9. The hospital, which will care primarily for those suffering with advanced stages of cancer, is planned to be located in the grouping of the Mayo Memorial and Variety Club Heart institutions at the University.

* * *

Dr. Erling W. Hansen, professor and head of the department of ophthalmology at the University of Minnesota, was named president-elect of the American Academy of Ophthalmology and Otolaryngology at its annual meeting in Chicago on October 13. He will take office in January, 1957, succeeding **Dr. A. C. Furstenberg,** dean of the University of Michigan Medical School, president for 1956.

Elected as a vice president of the organization was **Dr. Henry L. Williams,** Rochester.

* * *

Dr. Heinz H. Bruhl, staff member of the Faribault State School and Hospital, was the principal speaker at a meeting of the Faribault Rotary Club on October 19. Dr. Bruhl described his impressions of Europe following a two-month tour of the continent this past summer.

* * *

Dr. Wesley W. Spink, professor of medicine at the University of Minnesota, left for Tunisia on November 18, the start of a three-week trip to Africa and Europe, during which he planned to study and lecture on brucellosis.

* * *

Dr. and Mrs. Merrill D. Chesler, Minneapolis, returned in October from a two-week vacation in New York and New Jersey, during which Dr. Chesler attended the annual meeting of the American Society of Plastic and Reconstructive Surgery at Atlantic City. On November 8, Dr. Chesler conducted a regional seminar at Union Hospital, New Ulm, speaking on "The Treatment of Burns and Birthmarks in Children."

* * *

At the second annual Veterans of Foreign Wars Cancer Day at the University of Minnesota on October 15, the VFW reported that it has raised $150,000 of the $450,000 it needs to found a cancer research clinic at the University. At a luncheon the organization honored **Dr. Owen H. Wangensteen,** chief of the department of surgery at the University.

* * *

Dr. Wallace R. Anderson, Austin, spoke on accident prevention at a meeting of the Modern Matrons in Austin on November 3.

* * *

Dr. John W. Henderson, Rochester, has been named a section editor for the *Survey of Ophthalmology,* a new bi-monthly journal which will make its debut in February, 1956. The magazine, which will be published by Williams & Wilkins Company, Baltimore, is under the editorial direction of **Dr. Frank W. Newell,** chairman of the section on ophthalmology at the University of Chicago.

* * *

Dr. David A. Sher, Virginia, has been named a deputy county coroner by **Dr. Cyril Smith,** Duluth, coroner of St. Louis County.

* * *

Principal speakers at a meeting of the Minnesota Society of Neurology and Psychiatry in St. Paul on November 8 were **Dr. G. Wendell Hopkins** and **Dr. Maynard M. Cohen,** both of Minneapolis. Dr. Hopkins presented a paper on "Carbon Dioxide Therapy in Psychiatric Patients," while Dr. Cohen spoke on "The Neurologic Research Program at the University of Minnesota." Each presentation was an inaugural thesis.

* * *

At a State Medical Journal Conference at A.M.A. Headquarters in Chicago on November 7 and 8, MINNESOTA MEDICINE was represented on the program by **Dr. Arthur H. Wells,** Duluth, editor-in-chief, and **Olive V. Seibert,** Minneapolis, member of the board of editors of the publication. Dr. Wells gave a talk on "Editorial Boards and Editorial Advisory Committees." Miss Seibert conducted a workshop on editing material for publication in medical journals. The conference was held under the auspices of the Advisory Committee of the State Journal Advertising Bureau.

* * *

Dr. Thomas B. Magath, Rochester, has been advanced to the rank of rear admiral in the United States Naval Reserve.

* * *

Dr. Carl W. Waldron, Minneapolis, was the subject of a biographical sketch in the Town Toppers column of the *Minneapolis Star* on October 24. Dr. Waldron, who holds both medical and dental degrees, is now in semi-retirement after practicing in Minneapolis since 1919.

* * *

Dr. Owen G. McDonald, Duluth, has been elected a member of the board of governors of the American College of Surgeons, to represent Minnesota for the term 1955-1957. He will fill the vacancy caused by the death of **Dr. Howard K. Gray,** Rochester.

* * *

Principal speaker at a meeting of the St. Thomas College and Academy Mothers Club in St. Paul on October 18 was **Dr. Philip K. Arzt,** St. Paul. His topic was "The Psychiatric Aspects of Behavior Problems in Children."

* * *

Dr. A. B. Hunt, Rochester, was named president of the Central Association of Obstetricians and Gynecologists at a meeting of the organization in Columbus, Ohio, October 6 to 8.

* * *

Dr. Fred W. Wittich, Minneapolis, president of the International Association of Allergists, left on October 31, for Rio de Janeiro, Brazil, to preside at the second congress of the organization. The meeting, attended by 600 members, was held November 6 through 12.

* * *

Principal speaker at a Parent-Teachers Association meeting in Brainerd on October 18 was **Dr. E. R. Kanne**, Brainerd, who spoke on "Child Health."

* * *

The seventeenth annual award of the American Pharmaceutical Manufacturers Association has been bestowed upon **Dr. Charles W. Mayo**, Rochester, for his "outstanding contributions to both medicine and world understanding." Presentation of the award was scheduled for a dinner in New York on December 13.

Earlier, on October 23, it was announced that Dr. Mayo had been elected a trustee of the University of Pennsylvania. An alumnus of Pennsylvania's School of Medicine, Dr. Mayo is also a member of the board of regents of the University of Minnesota.

* * *

Dr. David W. Francis, Morristown, was given a special attendance award at the fortieth meeting of the Interstate Postgraduate Medical Association at Milwaukee, Wisconsin, November 14 to 17. The award is given to physicians who have contributed to their medical education by attending ten or more meetings of the organization since 1940.

* * *

Dr. Owen H. Wangensteen, chief of surgery at the University of Minnesota, has been elected first vice president of the American College of Surgeons.

* * *

Dr. Robert N. Barr, deputy executive officer of the Minnesota Health Department since 1949, was named secretary and executive officer of the Minnesota State Board of Health on November 1. He succeeds **Dr. A. J. Chesley**, who died on October 17. Dr. Barr has been with the health department since 1934.

* * *

Dr. N. L. Gault, Jr., was named assistant dean of the College of Medical Sciences at the University of Minnesota by the board of regents on October 22. Dr. Gault, who has been a member of the medical faculty since 1953, will take over his new duties on January 1, succeeding **Dr. Howard L. Horns**, who has resigned. Also an assistant dean of the medical school is **Dr. William F. Maloney**.

* * *

Principal speaker at a meeting of the West Duluth Women's Club on November 4, was **Dr. George M. Cowan**, Duluth. He discussed the subject, "What Makes Women Nervous?"

An Albert Lasker Award for outstanding achievement in medical research has been given to four University of Minnesota surgeons: **Dr. C. Walton Lillehei, Dr. Richard L. Varco, Dr. Morley Cohen** and **Dr. Herbert E. Warden.** The award was presented for their numerous advances in cardiac surgery.

* * *

Dr. Edgar V. Allen, Rochester, was elected president of the American Heart Association at its annual meeting in New Orleans, Louisiana, recently. Dr. Allen has been a member of the board of directors of the organization since 1944.

* * *

Dr. Harold S. Diehl, dean of the University of Minnesota College of Medical Sciences, has been elected vice president of the Association of American Medical Colleges for 1956.

* * *

"Pleural Effusion" was the title of a talk given by **Dr. W. R. Schmidt**, Minneapolis, at a dinner meeting of the Southwestern Minnesota Medical Society in Worthington on October 10.

* * *

Dr. Emil J. Fogelberg, St. Paul, was named president-elect of the Minnesota Academy of General Practice at the annual meeting of the organization in Minneapolis on October 18. He will succeed **Dr. Raymond Page**, St. Charles, who will serve as president during 1956. Other officers named at the meeting were **Dr. E. J. Tanquist**, Alexandria, vice president, and **Dr. John Butler**, Cloquet, secretary.

Named as new directors of the organization were **Dr. Herbert L. Stolpestad**, St. Paul; **Dr. Paul M. Smith**, Lake Crystal, and **Dr. H. A. Korda**, Pelican Rapids.

* * *

Grants from the American Cancer Society to the University of Minnesota have now exceeded two million dollars for cancer research and training. The announcement was made by **Dr. David P. Anderson**, Austin, retiring president of the society's Minnesota Division, at its annual meeting in Minneapolis on October 14.

Among the new officers elected by the division are **Dr. Lyle J. Hay**, Minneapolis, second vice president, and **Dr. Philip F. Eckman**, Duluth, third vice president.

* * *

Dr. C. S. Donaldson, St. Cloud, was named head of the medical services of Civil Defense in St. Cloud at a meeting on October 12. He succeeds **Dr. R. N. Jones** and will have charge of organizing physicians, dentists and nurses.

* * *

Dr. Miles Griffin, Oakland, California, was named president of the Alumni Association of the Mayo Foundation at the organization's thirty-first annual meeting in Rochester on October 28. He succeeds **Dr. Shirley C. Lyons** of New Orleans, Louisiana.

Other new officers include **Dr. George E. Rice**,

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GROUP DISABILITY INSURANCE

For several years the Insurance Liaison Committee of the Minnesota State Medical Association has been actively studying the question of group disability insurance for the membership. Insurance administrators were requested to submit proposals, and five administrators submitted ten different plans for six insurance companies.

After numerous meetings, the Committee, on April 15, 1955, voted:

1. To recommend that the Minnesota State Medical Association adopt a group disability program for members.
2. To recommend the plan submitted by the North American Accident Insurance Company and administered by the Charles O. Finley Company of Chicago, Illinois, for consideration by the Council.

Reasons for a Group Disability Plan

Any of our members under fifty and physically acceptable can obtain disability insurance from any casualty company at regular rates. They do so, however, without the advantages of group action. These advantages are:

1. The disability program of any individual may not be cancelled by the company so long as the group plan remains in force, and the member is under 70, remains a member of the organization, is actively engaged in his profession or business and continues to pay premiums.

2. The coverage under the group plan is much better than most individual policies.

3. There is a substantial savings in cost (30 to 50 per cent).

4. If 50 per cent of members enroll during the enrollment period, policies will be issued to all applicants, thus making the insurance available

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Report of the Insurance Liaison Committee of the Minnesota State Medical Association, made and adopted at the 1955 House of Delegates session at the annual meeting, May 24, 1955.

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to many who might otherwise be considered unacceptable risks.

It is the opinion of the Committee that the Association should provide, encourage, and protect a group disability insurance program for its members. An influential organization like the Minnesota State Medical Association would give excellent protection against cancellation of such a group plan, though it could not guarantee noncancellation.

Many members of the Minnesota State Medical Association do not have such desirable group disability insurance available to them. Others have similar plans offered by the specialty organizations and may wish to take this proposed plan in addition to or in place of what they now have.

Requirements of a Plan as Imposed by the Committee

The committee decided the following features were necessary in any disability plan it might recommend:

1. It must include all members actively in practice regardless of state of health if more than 50 per cent enroll during the initial enrollment period. If a smaller per cent enroll, it is poor underwriting to include poor risks as the plan then becomes overloaded with uninsurables.
2. It must be noncancellable to the individual unless the entire group plan is cancelled.
3. It must pay for lifetime for accident and at least five years for sickness.
4. It must have waiver of premium provision.
5. It must have no exclusions other than standard ones for military service, war, suicide, pregnancy or miscarriage.
6. House confinement must never be required to collect on a claim.
7. There must be no "accidental means" clause.
8. Coverage must be world-wide.
9. There must be a grace period of thirty-one days.
10. The Company must issue a Master Policy to the Minnesota State Medical Association and a certificate to the individual, rather than indi-

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vidual policies to members only. This gives a closer tie between the Company and the Association, and adds security.

11. It must require only regular attendance of a physician rather than attendance at any specified time interval.

12. Benefits must be payable in full without any pro-rating with other contracts the insured may be carrying.

13. Premiums must be returned, pro-rata, for those leaving the Minnesota State Medical Association, including those entering military service.

14. It should be possible for a member to apply during the enrollment period, but to postdate his policy by as long as six months, his enrollment to count toward the 50 per cent participation anyway.

15. Hospitalization benefits should be provided for ninety days.

16. The Hennepin County and Mayo Clinic groups, now more than 80 per cent insured, should be excluded from the initial enrollment in order to facilitate obtaining the necessary 50 percent participation.

17. The company must be licensed to do business in Minnesota.

We are happy to announce that the above features are all included in the plan which the Committee is recommending for your approval.

The Administrator and the Company

The Charles O. Finley Company is the largest administrator of professional group insurance programs in the United States and currently administers a similar group disability plan of the Hennepin County Medical Society. The Committee met with the Executive Secretary of the Hennepin County Medical Society and was informed that the program, the insurance company, and the administrator are highly satisfactory. Eighty-two per cent of the members of the Hennepin County Medical Society are now enrolled in their group disability program. It is conceivable that at some future time the Minne-

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sota State Medical Association and Hennepin County group plans might be merged into one group if both of these organizations should so agree.

The Charles O. Finley Company is also administrator for a large number of other disability insurance programs, among which are: The American Academy of Pediatrics, the Chicago Medical Society, the Southern Medical Association, the California State Medical Association, the Massachusetts Medical Society, the American Urological Society, the American Proctological Society and the American College of Surgeons.

The Committee has letters from numerous associations recommending the Charles O. Finley Company and stating that their experience with group disability programs has been very satisfactory.

Members of the Minnesota State Medical Association will be provided with enrollment material and the charter enrollment campaign is conducted over a sixty-day or ninety-day period. All members of the Minnesota State Medical Association, excluding the Hennepin County group and fellows of the Mayo Clinic, will be eligible to apply and all acceptable applicants who have applied will be issued coverage at the close of the enrollment period. In addition, some coverage will be issued to those applicants who have a history of physical impairment if the majority of the eligible individuals enroll during the charter enrollment period.

The success of this disability program is entirely dependent on its remaining in force as a group plan. The company may cancel the entire program if it so desires. It would undoubtedly do so if the experience with our group were unsatisfactory. The best way to insure its success is by a large member-participation at all times. This committee believes it is vital to the group disability program that the Minnesota State Medical Association maintain a close liaison with the administrator through the executive secretary's office, in order to assist the administrator in obtaining new applicants for the plan and to guard against an unfavorable loss experience

(Continued on page xxx)

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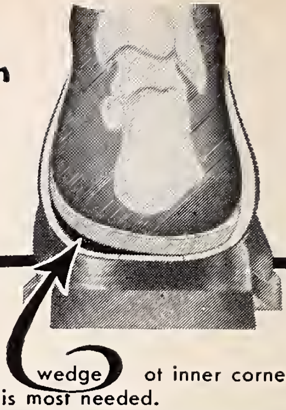

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J. Pediat. 44:386, 1954.

White, R. H. R., and Standen, O. D.:
Brit. M. J. 2:755, 1953.

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J. Pediat. 45:419, 1954.

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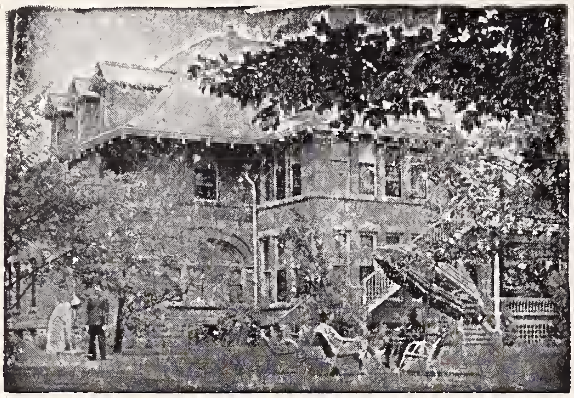

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Dr. L. R. Gowan, M.D., M.S., Medical Director

(Continued from page xxvii)

which might cause the insurance company to cancel the entire program.

The Insurance Department of the State of Minnesota reported that the North American Accident Insurance Company commenced business in 1886 and was admitted in the State of Minnesota in 1903, that the company is currently licensed in this state and is therefore considered reputable.

The Insurance Department, also, states that Mr. Charles O. Finley, 2100 W. Sixth Avenue, Gary, Indiana, is duly licensed to sell accident and health insurance in this state for the above company. He carries a nonresident license, No. 249.

In the opinion of the Insurance Liaison Committee, the Group Disability Insurance Program, offered by the North American Accident Insurance Company, best fits the needs of the Minnesota State Medical Association. We are confident the members will find it very satisfactory. We also have the utmost confidence in the ability of the Charles O. Finley Company to administer this program. We urge its approval by the Council, and if so approved, we hope it will be adopted by the House of Delegates so that it can be installed at an early date.

Respectfully submitted,
INSURANCE LIAISON COMMITTEE
CORRIN H. HODGSON, M.D. *Chairman*
R. P. BUCKLEY, M.D.
JOHN DORDAL, M.D.
P. W. HARRISON, M.D.
ROBERT MCGANDY, M.D.
HORATIO VAN CLEVE, M.D.
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Some chronic conditions are preventable; some are deferable; some are curable; and some are modifiable. We know how they are spread and how to prevent their spread. Some conditions, such as diabetes or rheumatic heart disease, are deferable even where there is a tendency toward it, if the appropriate medical care is provided in time. Some conditions, such as pernicious anemia or cerebral hemorrhage, are modifiable.—DANIEL BERGSMA, M.D., New Jersey, *Pub. Health News,* April 1955.

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X-RAY MOTION PICTURES AID IN STUDY OF SWALLOWING PROCESS

Cinefluorographic examination of the swallowing process, using fluoroscopic, x-ray motion pictures, now makes it possible to verify accurately what physicians heretofore have merely been able to infer and generalize.

This is the report from a University of Rochester radiologist, Dr. G. H. Ramsey.

The new information, recorded on 35 millimeter movie film at camera speeds of 30 and 60 frames per second, is expected eventually to provide a reliable x-ray motion test for normal swallowing.

Assisting Dr. Ramsey in the research at the University of Rochester Medical School were Doctors J. S. Watson and R. Gramiak, from the department of radiology, and Mr. S. A. Weinberg. Their conclusions have been presented in the April, 1955, issue of *Radiology*.

Their findings are based on cinefluorographic studies of 300 persons, half of whom had "normal" swallowing processes.

"We do not believe that this account of the cinefluorographic appearances of normal swallowing is in any sense complete, but rather that additional understanding of the many details can be gained by further study of normal subjects and animals, and by analysis of various pathological conditions that occur in the area," Dr. Ramsey reported.

Other conclusions advanced in the *Radiology* article:

The act of swallowing, in the view of the authors, ends in the hypopharynx when the cricopharyngeus contracts on the tail of the bolus, thereby signaling to the proximal swallowing apparatus that it can either return to the resting stage or begin a new swallow.

With the exception of a few constant reflex relationships, both the timing and order of the swallowing events vary considerably in different individuals, and in the same individual under different conditions. Other variations develop with age.

Some persons, by habit or training, deviate markedly from the usual mechanism—in particular, the "competitive beer-drinker." He merely opens his mouth, and never closing it during the act, pours the beer down his throat. All this must be taken into account when trying to distinguish between the unusual and the abnormal.

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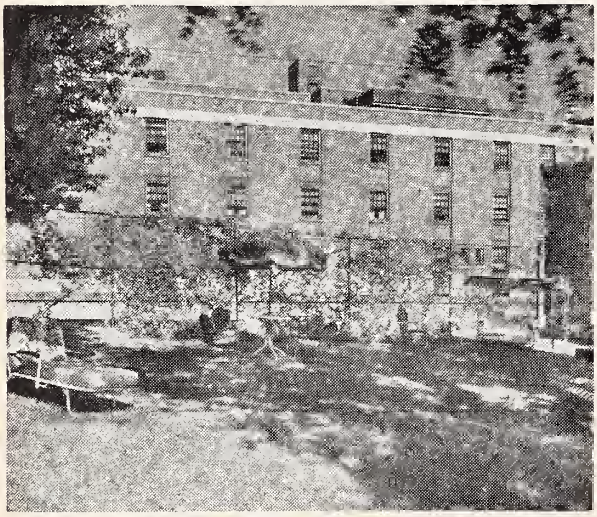

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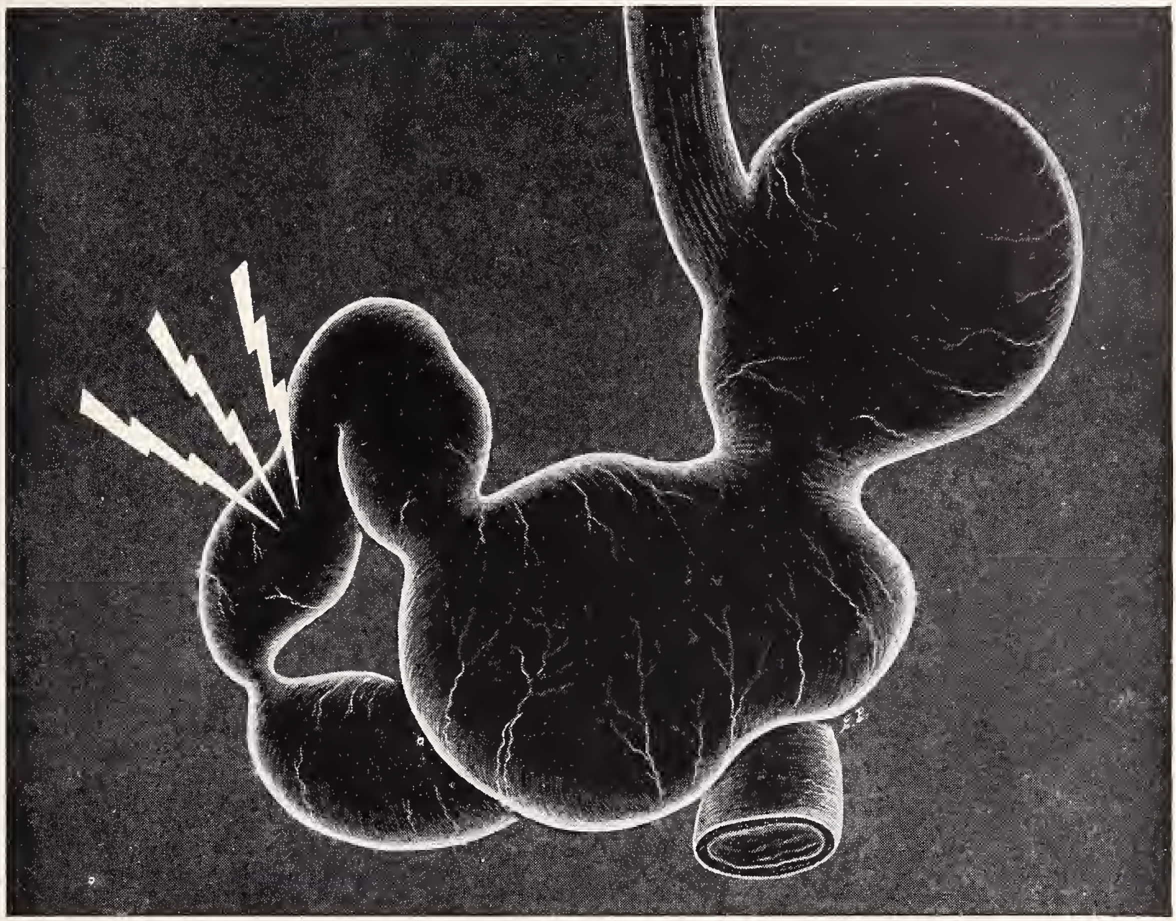

Abnormal Motility as the Cause of Ulcer Pain

Until recently the general opinion was held that ulcer pain was primarily caused by the presence of hydrochloric acid on the surface of the ulcer.

Present investigations[1,2] on the relationship of acidity and muscular activity to ulcer pain have led to the following concept of its etiologic factor:

> "... abnormal motility[2] is the fundamental mechanism through which ulcer pain is produced. For the production and perception of ulcer pain there must be, one, a stimulus, HCl or others less well understood; two, an intact motor nerve supply to the stomach and duodenum; three, altered gastro-duodenal motility; and four, an intact sensory pathway to the cerebral cortex."

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---

[1] Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthīne, Gastroenterology *25*:416 (Nov.) 1953.

[2] Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.

SEARLE

MINNESOTA STATE MEDICAL ASSOCIATION
House of Delegates—Summary of Proceedings
Minneapolis Session—May 22 and 23, 1955

First Meeting, Sunday, May 22, 1955

Gold Room, Hotel Radisson
Minneapolis, Minnesota

The 102nd Annual Session of the House of Delegates of the Minnesota State Medical Association opened in the Gold Room of the Hotel Radisson, Minneapolis, Minnesota, at 2:00 p.m., Sunday, May 22, 1955, Dr. C. G. Sheppard, Hutchinson, Speaker of the House of Delegates, presiding.

After an invocation by Father Louis W. Forrey, Pastor of Our Lady of Grace Church of Edina, the minutes of the 1954 meeting were accepted, a quorum being present.

Dr. Arnold O. Swenson of Duluth, president, spoke on the work done during the year, especially commending the work of the Insurance Liaison Committee and the Committee on Malpractice Insurance. He said that during the past few months he had received letters from several county societies criticizing the state officers and the Council for the way they handled the polio vaccine situation in the state. Dr. Swenson pointed out that the Association has been co-operating with the State Board of Health completely, and assured the delegates that it was through no fault of the officers or of the Council that confusion has existed. It is necessary to abide by the decisions of the United States Public Health Service and the State Department of Health, he said.

After introducing Dr. E. L. Bernhart, president of the State Medical Society of Wisconsin, Dr. Sheppard called on Dr. C. L. Oppegaard, chairman of the Council, to report on Council meetings held Saturday afternoon and evening, May 21, and Sunday morning, May 22, at the Hotel Radisson.

Report of the Council

Dr. Oppegaard: The Council held its first meeting of the annual session Saturday at 2 p.m. and continued sessions Saturday evening and this morning at 8:30.

The principal business, after the opening of the meeting, was extended consideration of the report of the Finance Committee of which Dr. J. M. Stickney is chairman. He will present to you this afternoon the matter of exchange journals sent to Minnesota in exchange for Minnesota Medicine.

This practice is customary in the case of all medical

(Continued on Page xxvi)

In accordance with action by the Council of the Minnesota State Medical Association, taken in 1944, publication of the proceedings of the House of Delegates is limited to summary.

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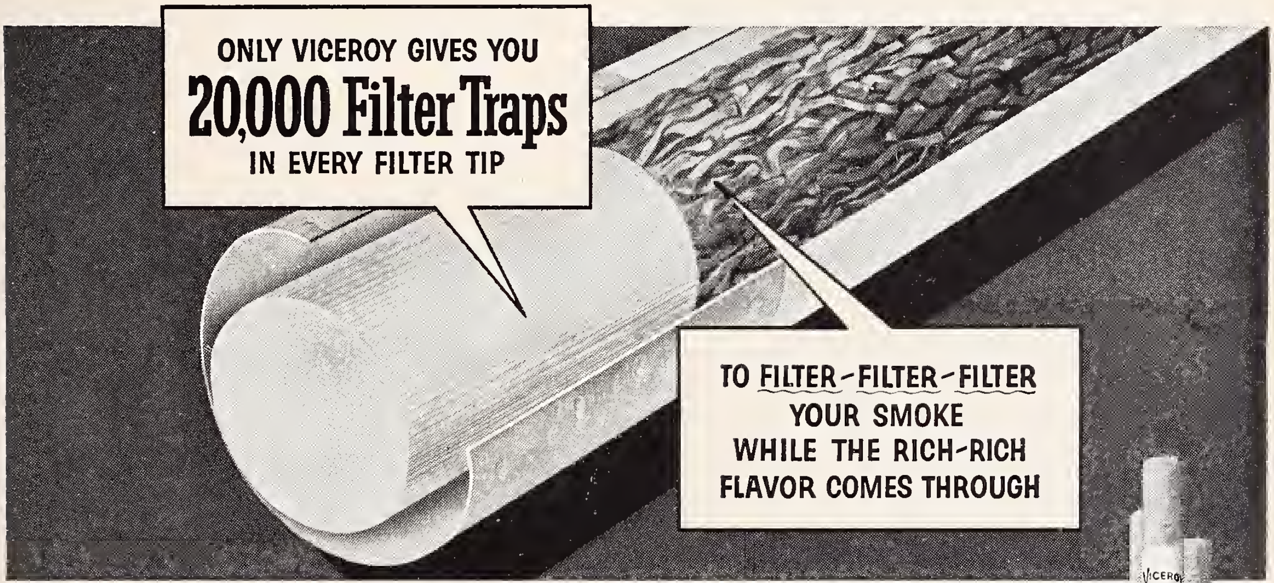
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(Continued from Page xxiv)

journals and it was agreed that a specific effort should be made to see that the journals are divided evenly between the three larger medical societies of the state, St. Louis, Ramsey and Hennepin counties, and also that the Board of Editors would have a better chance to study the exchanges in other states before being distributed to the libraries.

The Council considered several matters which will come up for action by the American Medical Association House of Delegates at Atlantic City for the purpose of instructing delegates to the AMA, as follows:

Questions on fees. This is the subject of an extensive report to be submitted by the Committee on Medical Practices of the AMA and the Council unanimously instructed the delegates to endorse the AMA effort to eliminate unethical and objectionable practices.

Resolutions have been circulated by the Texas and Louisiana Medical Associations asking that joint accreditation of hospitals be limited in that only the American Medical Association be permitted to accredit medical services in the hospitals. It was decided after some discussion to leave the action on that matter at Atlantic City to the judgment of the delegates themselves.

Ethical questions involved in dispensing drugs by physicians and in ownership of pharmacies by physicians will also be brought to the attention of the AMA House of Delegates. Once again, this was considered by your Council. It was generally agreed that dispensing, in the absence of a good pharmacy, or at nights or otherwise when the welfare of the patient demands it, cannot be regarded as unethical. On the other hand, ownership of a pharmacy, especially where the physician channels trade to his pharmacy, was regarded as bad ethics and bad public relations. No specific direction was given, but it was the general feeling that the AMA should be supported in its stand on the subject which was adopted by the AMA House of Delegates in San Francisco at its June, 1954, meeting.

The report made at that time was as follows from Section Eight of the Principles of Medical Ethics: (1) it was unethical for a physician to participate in an ownership of a drug store in his medical practice area unless adequate drug store facilities are otherwise unavailable; (2) this inadequacy must be confirmed by his component medical society: (3) the same principle applies to physicians who dispense drugs or appliances; (4) in both instances the practice is unethical if secrecy and coercion are employed or if financial interest is placed above the quality of medical care; and, finally (5) on the other hand, sometimes it may be advisable and even necessary for physicians to provide certain appliances or remedies without profit, which patients cannot procure from other sources.

The Council considered a supplementary report of the Insurance Liaison Committee presented by the chairman, Dr. Corrin H. Hodgson of Rochester. The committee recommended that The Council ask for House of Delegates approval of a group health and accident insurance plan for members of the Minnesota State Medical Association, written by the North American Accident Insurance Company and administered by the Charles O. Finley Company of Chicago.

Dr. Hodgson outlined in detail many of the advantages of the plan, which is already in force in a separate group plan for members of the Hennepin County Medical Society, and also noted the advantages of a group disability plan over individual plans.

The plan briefly provides that a master policy would be written for the Minnesota State Medical Association. The company offers an enrollment period of sixty or ninety days in which any member of the Association may apply for the group policy. The state association must acquire at least fifty per cent enrollment in the plan, exclusive of the Hennepin County Medical Society and the Mayo Clinic members.

Mr. Charles O. Finley, administrator of the proposed plan, appeared before The Council and presented many mechanical details for The Council's consideration.

After much discussion of many of the details involved in accepting such a plan, the Council recommended the House of Delegates approval. The Council also expressed thanks to Dr. Hodgson and his committee and to the assistant executive secretary for the great amount of work involved in preparing this material and presenting the recommendations.

The Council also considered a request from the Division of Vocational Rehabilitation of the Minnesota State Department of Education for the Association to appoint an advisory committee to the Division which would consist of representatives of the several specialties. The Council empowered the chairman to appoint such a committee after requesting suggestions from the organizations involved.

The Council approved the report on the requirements for nurses' aide training which was submitted by the Minnesota League of Nurses. The Council approved a new guide for "Emergency Treatment in Industry" requested by the Committee on Industrial Health, which committee has already approved the revised guide.

The Council considered nomination for election of Delegates to the House of Delegates of the American Medical Association. The Council's recommendation will be presented to the House of Delegates tomorrow when officers, councilors, and delegates are elected.

Endorsement of policies for the Cancer Detection Center of the University of Minnesota for the three-year period from March 1, 1956 to March 1, 1959, was considered at today's session. After listening to an impressive account of the accomplishments from Dr. Hitchcock, the former director, and his successor, Dr. Sullivan, the Council unanimously approved and commended the work of the center.

The question of the polio vaccination program and its current status here and elsewhere also received attention. As indicated by your president, Dr. Swenson, all aspects of the confused situation were considered and the following statement of policy unanimously agreed upon:

The Council and members of the Minnesota State Medical Association depend solely upon the National Institute of Health and the Minnesota Department of Health for technical information and advice on the Salk vaccine and the vaccination program, having no other source of such information.

They feel that the greatest possible credit should be given to the National Foundation for Infantile Paralysis and other agencies for their great contribution to public health.

At the same time they feel that the value of following the traditional conservatism of medicine in matters which involve the introduction of new treatments and affording added protections to these new procedures has been well demonstrated and the wisdom of the Minnesota State Medical Association and the Minnesota Department of Health in refusing to take part in the vaccination program on an experimental basis last year, has also been well demonstrated.

The Council has complete confidence in the Minnesota State Department of Health and will co-operate fully with that department in carrying on the program.

The report was unanimously accepted by the delegates, leaving the report of the Insurance Liaison Committee for further consideration later, and Dr. Sheppard called for the report of Dr. J. M. Stickney, chairman of the Finance Committee.

Report of the Finance Committee

Dr. Stickney said that the paid membership in the Minnesota State Medical Association as of May 1 of this year is 2,787, which is 94 more than a year ago.

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J. Pediat. 44:386, 1954.

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It is expected that there will be approximately 100 more paid memberships before the end of this year. He pointed out that expenses for the year 1954 exceeded income by $3,026.00. The net loss was due to the increase in expenses, said Dr. Stickney, and he predicted that the deficit for the year 1955 will be even greater. Association dues compare favorably with those of some of the surrounding states, but for the present, the committee has recommended to the Council that the deficits for 1954 and 1955 be taken out of the surplus and that a decision regarding future policies be made in 1956. The charge against the Veterans Medical Service stands at $3,010 and will increase at the rate of $70 per month until the end of the lease, a year from now. When this claim can be settled, the net loss to the Association will not exceed $1,000, which is a smaller loss than anticipated. During 1954, aid granted from the Physicians' Assistance Fund amounted to $2,060. There is still a considerable sum left in that account.

Dr. Sheppard interrupted the proceedings to allow Dr. Swenson to introduce Dr. Elmer Hess, president-elect of the American Medical Association. Dr. Hess spoke briefly, saying that the AMA is the one organization that takes its orders from the bottom and gives none from the top. He wished the delegates a very successful meeting, and said he was happy to be with them.

It was moved, seconded and carried that Dr. Stickney's report be accepted and Dr. Sheppard then called on Dr. R. N. Barr of the State Board of Health. Dr. Barr first discussed the polio vaccine program. He said that the vaccine being used by Minnesota physicians was as safe as it could possibly be made. He urged physicians to co-operate in the surveillance of every case of polio in the state: to report the case by phone; to send the State Board of Health a sample of the stool and a blood specimen from the patient; ten days later send another blood. Authentic information on all phases of the polio situation will be released by the State Board of Health as soon as it is available. Dr. Barr asked doctors to fill out and return forms which they would be sent, the information to be used in obtaining and spending Hill-Burton money to the best advantage.

Dr. Sheppard then called for reports of the reference committees. The first was by Dr. R. F. Hedin, Red Wing, Chairman of the Reference Committee on Medical Education Reports:

Medical Education Reports

Dr. Hedin: The committee considered the following reports:

Committee on First Aid and Red Cross—John S. Lundy, M.D., Rochester, Chairman

Committee on Blood and Blood Banks—John S. Lundy, M.D., Rochester, Chairman

Committee on Cancer—David P. Anderson, Jr., M.D., Austin, Chairman

Committee on Conservation of Hearing—K. M. Simonton, M.D., Rochester, Chairman

Heart Committee—F. J. Hirschboeck, M.D., Duluth, Chairman

Committee on Hospitals and Medical Education—H. S. Diehl, M.D., Minneapolis, Chairman

Committee on Public Health Nursing—M. McC. Fischer, M.D., Duluth, Chairman

Committee on Syphilis and Social Diseases—H. G. Irvine, M.D., Minneapolis, Chairman

Committee on Tuberculosis—J. F. Briggs, M.D., St. Paul, Chairman
Committee on Vaccination and Immunization—R. N. Barr, M.D., Minneapolis, Chairman

All the above reports were accepted by the reference committee, with the following observations: the committee noted with pleasure the co-operation between the University and physicians of the state, as reported by the Committee on Cancer; the Heart Committee is doing an excellent job in furthering research in heart disease; the committee wished to call attention to the dire need for nurses and technicians and suggested a short preceptorship be given for technicians who have been trained in short courses so that they will be able to do a more thorough job when they are employed, and commended the excellent report of the Committee on Hospitals and Medical Education; on going over the report of the Committee on Syphilis and Social Diseases, the committee questions the advisability of the further use of silver nitrate in the eyes of the newborn in the light of other effective drugs; the committee commended the Committee on Tuberculosis on the excellent job it is doing in combatting tuberculosis in the state; the Committee on Conservation of Hearing was congratulated on the excellent job it is doing in determining those having hearing defects in Minnesota; the committee recognized the present state of confusion regarding the vaccination for poliomyelitis and felt that uniform safe policies would soon be established.

In answer to a question on this report, Dr. Barr said that the short courses offered to office help, technologists and others were not sponsored by the Department of Health, though the department was interested in them and wanted to see them continued. These courses are still operating and an additional course is offered at the University in the Extension Division for two quarters. Twenty-four persons took the course last year.

The House unanimously accepted Dr. Hedin's report and the Speaker called for the report of the Reference Committee on Miscellaneous Scientific Reports, Dr. W. E. Wellman, Rochester, Chairman.

Miscellaneous Scientific Reports

Dr. Wellman: The committee read and approved the reports of the following committees:

Committee on Anesthesiology—R. C. Adams, M.D., Rochester, Chairman
Committee on Child Health—G. B. Logan, M.D., Rochester, Chairman
Committee on Chronic Illness—E. L. Tuohy, M.D., Duluth, Chairman
Committee on Diabetes—John R. Meade, M.D., St. Paul, Chairman
Fracture Committee—E. T. Evans, M.D., Minneapolis, Chairman
Committee on General Practice—James Blake, M.D., Hopkins, Chairman
Historical Committee—Robert Rosenthal, M.D., St. Paul, Chairman
Committee on Industrial Health—L. S. Arling, M.D., Minneapolis, Chairman
Committee on Maternal Health— J. J. Swendson, M.D., St. Paul, Chairman
Committee on Medical Testimony—E. M. Hammes, Sr., M.D., St. Paul, Chairman
Committee on Military Affairs—J. H. Tillisch, M.D., Rochester, Chairman

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Committee on Nervous and Mental Diseases—W. P. Gardner, M.D., St. Paul, Chairman
Committee on Ophthalmology—T. R. Fritsche, M.D., New Ulm, Chairman
Committee on Public Health Education—F. J. Elias, M.D., Duluth, Chairman
Editorial Committee—A. H. Wells, M.D., Duluth, Chairman
Radio and Television Committee—R. M. Burns, M.D. St. Paul, Chairman
Speakers' Bureau—H. M. Carryer, M.D., Rochester, Chairman

Special comment was made on the fine work of the Historical Committee in the preparation and publication of historical data in MINNESOTA MEDICINE; attention was called to the fact that the Committee on Industrial Health had assisted in the preparation of a publication, "Emergency Treatment in Industry," a guide to proper practice when a worker is injured; the Committee on Military Affairs had made a study of the question of reimbursement for examination of National Guard personnel and referred the matter back to the Council; further clarification of the relationship between ophthalmologists and optometrists, considered by the Committee on Ophthalmology, was expected at the next AMA meeting; the committee recommended that secretaries of county medical societies call attention of members to conferences on child health and urge at least one member to attend.

The delegates unanimously accepted the report and the Speaker called on Dr. H. E. Drill of Hopkins to present the report of the Reference Committee on Medical Economics Reports.

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Medical Economics Reports

Dr. Drill: The following reports were considered:

Committee on Malpractice Insurance—A. E. Ritt, M.D., St. Paul, Chairman

Committee on Medical Economics—George Earl, M.D., St. Paul, Chairman

Committee on University Relations—O. J. Campbell, M.D., Minneapolis, Chairman

Committee on Hospitals and Professional Relations—J. M. Stickney, M.D., Rochester, Chairman

Scholarship Selection Committee—F. J. Elias, M.D., Duluth, Chairman

Editing and Publishing Committee—E. M. Hammes, Sr., M.D., St. Paul, Chairman

Editor's Annual Report—A. H. Wells, M.D., Duluth, Chairman

Committee on Medical Ethics—Harry Klein, M.D., Duluth, Chairman

Committee on Interprofessional Relations—W. P. Gardner, M.D., St. Paul, Chairman

Medical Advisory Committee—W. H. Hengstler, M.D., St. Paul, Chairman

All reports were commended and accepted by the committee.

After Dr. Ritt gave a detailed explanation of the malpractice insurance policy recommended by the Committee on Malpractice Insurance, and answered many questions from the floor, Dr. Drill moved that the report of the reference committee be voted upon, omitting the Report on Malpractice Insurance and the matters pertaining to catastrophic illness. The House unanimously approved that section of the report.

The report of the Committee on Malpractice Insurance was then unanimously approved, after much discussion. Dr. Ritt and his committee were commended for the tremendous amount of work they had done. Further discussion followed on the question of Minnesota Medical Service offering a major medical care endorsement to their Plans A and B. Dr. A. O. Swenson, Mr. F. Manley Brist, legal counsel, and Dr. E. Covell Bayley, member of the Blue Shield Board, made comments and answered questions. The House then passed a motion to accept the report of the committee, recommending that a committee of five to fifteen be appointed to study with the Board the contract, and to clarify the problem, the committee to report to the Council, with the Council's decision to be tantamount to that of the House on approval or disapproval of the major medical care endorsement proposed by Blue Shield.

Dr. Sheppard called on Dr. O. M. Heiberg, Worthington, Chairman of the Reference Committee on Miscellaneous Medical Economics Reports, to give his report.

Miscellaneous Medical Economics Reports

Dr. Heiberg: The Reference Committee reviewed the following reports:

Insurance Liaison Committee—Corrin Hodgson, M.D., Rochester, Chairman

Committee on Medical Service—J. A. Bargen, M.D., Rochester, Chairman

Committee on Rural Medical Service—Paul C. Leck, M.D., Austin, Chairman

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Committee on State Health Relations—C. E. Proshek, M.D., Minneapolis, Chairman
Committee on Physicians' Assistance—W. R. Humphrey, M.D., Stillwater, Chairman
Committee on Veterans Medical Service—R. H. Creighton, M.D., Minneapolis, Chairman
Membership Committee—H. F. R. Plass, M.D., Minneapolis, Chairman

The report of the Insurance Liaison Committee will be given in a separate report to the House. The committee recommended acceptance of the other reports and made special mention of the report of the Committee on Medical Service, commending its work in the improvement of blanks clarifying responsibility of the athlete and his parents. Dr. William E. Proffitt was commended for his article in MINNESOTA MEDICINE; the committee concurred with the Committee on State Health Relations in recommending that the salaries of superintendents of mental and other hospitals be set by the Commissioner of Public Welfare, and that certain personnel be changed to an unclassified service; the reference committee concurred with Council action in disbanding the Veterans Medical Service Committee and for establishment of a new Committee on Veterans Affairs to handle all general problems on medical care for veterans; it was the consensus of the committee that major emphasis of the membership program be placed at the county society level.

Dr. Heiberg's report having been approved, Dr. Corrin Hodgson was called upon for further information on the report of the Insurance Liaison Committee. After discussion of the group disability program the committee had recommended for members of the Association, Dr. Hodgson answered questions from the floor, after which his report was unanimously approved. Dr. Hodgson introduced Mr. Charles O. Finley who will administer the plan. The Speaker called on Dr. R. P. Buckley for the Officers and Councilors Reports.

Officers and Councilors Reports

DR. BUCKLEY: The first two reports dealt with the plan for group professional liability insurance, the second was the report of the Insurance Liaison Committee on a proposed group health and accident policy. Both were special reports of the Council. Another special report of the Council on the proposed plan for major medical expense coverage was reviewed and recommended in principle to the House for adoption. Reports of the Chairman of the Council, the councilors, the treasurer, the secretary and executive secretary were approved.

The House voted acceptance of these reports and at 5:00 p.m. the Speaker adjourned the afternoon session until 8:00 p.m.

Second Meeting, Sunday, May 22, 1955 (8:15 p.m.)

After the Speaker called the evening session to order, he called on Mrs. Peter S. Rudie, Duluth, president of the Woman's Auxiliary to the Minnesota State Medical Association. Mrs. Rudie reported on the status, activities and aims of the Auxiliary.

Dr. Sheppard then asked Dr. H. E. Drill, Hopkins, to speak on the American Medical Education Foundation.

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AMEF Fund

Dr. Drill: Minnesota has done very poorly in raising money for the Foundation. Therefore, I would like to make a motion that this House of Delegates take action in placing the solicitation for the AMEF Fund on a county society level and that each county society assume the responsibility for its successful execution. (See Proposed Plan of Insured Endowment for the AMEF, page 586).

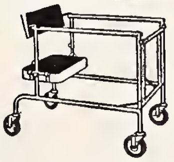
The motion was carried. Dr. F. H. Magney, Duluth, then gave the report of the Minnesota State Board of Medical Examiners.

State Board of Medical Examiners

Dr. Magney: During the last year, the State Board of Medical Examiners licensed 409 doctors to practice in the state. Of this number 141 were graduates of the University of Minnesota; 259 were admitted through examination; 85 by reciprocity; and 69 by the National Board. The Board has made two changes in the ruling on foreign graduates. During the past year we required two years of internship and only the graduates of schools approved by the AMA are considered. There was no revocation of license last year. Only one osteopath was licensed in 1954, and the total number of osteopaths is slowly decreasing. Eight new chiropractors were licensed, all on the basis of examination in the basic sciences.

There were no questions about Dr. Magney's report and Dr. R. N. Bieter was called on to give the report of the Minnesota State Board of Examiners in the Basic Sciences. He reported on the applicants to take examinations and on certifications that the Board sub-

(Continued on Page xxxv)



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Surgical Technique, Surgical Anatomy and Clinical Surgery, four weeks, October 17
Surgical Anatomy and Clinical Surgery, two weeks, August 22
Surgery of Colon and Rectum, one week, September 19
General Surgery, two weeks, October 3; one week, October 17
Gallbladder Surgery, ten hours, October 24
Thoracic Surgery, one week, October 3
Esophageal Surgery, one week, October 10
Basic Principles in General Surgery, two weeks, September 26
Fractures and Traumatic Surgery, two weeks, October 17
GYNECOLOGY & OBSTETRICS—Vaginal Approach to Pelvic Surgery, one week, November 7
Three-week Combined Course Gynecology and Obstetrics, September 12
MEDICINE—Two-week Course, September 26
Electrocardiography and Heart Disease, two weeks, October 10
Gastroscopy, One Week Advanced Course, September 12
Gastroenterology, two weeks, October 24
Dermatology, two weeks, October 17
RADIOLOGY—Clinical and Didactic Course, two weeks, October 3
Clinical Uses of Radioisotopes, two weeks, October 10
PEDIATRICS—Clinical Course, two weeks, by appointment
Pediatric Cardiology, one week, October 10 and 17
UROLOGY—Two-week Course October 10

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(Continued from Page xxxiii)

mitted to other states. There were 478 persons examined.

Dr. R. H. Wilson was called on for the report of the Statewide Advisory Committee to the Department of Public Welfare. He said that at the last session of the Legislature a law was passed that says the Commissioner of Public Welfare shall establish maximum fees which shall be paid to physicians, hospitals and nursing homes for welfare patients. However, he noted that we have assurance that the program will be worked out as before, on a level between the counties, the Welfare Boards and the committee of the Association.

Dr. Robert Rosenthal, chairman of the Historical Committee, gave the Necrology Report. A moment of silence was observed. The meeting was then adjourned until 12:15 p.m. Monday, May 23, 1955.

Third Meeting, Monday, May 23, 1955 (1:00 p.m.)

The House reconvened at 1:00 p.m., called to order by the Speaker. He asked for the report of the Resolutions Committee, Dr. C. W. Moberg, Detroit Lakes, Chairman. Dr. Moberg reported that resolutions regarding the major medical care plan had been withdrawn, the matter being resolved at yesterday's session. Resolutions of thanks to the Hennepin County Medical Society, the newspapers, the radio and television stations, the Minneapolis hotels and the Minneapolis Chamber of

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Commerce for their respective contributions to the success of the meeting were proposed by Dr. Moberg and unanimously adopted by the Delegates. Dr. Sheppard asked for the report of the Nominating Committee, Dr. A. E. Ritt, Saint Paul, Chairman.

Report of the Nominating Committee

Following recommendation made by Dr. Ritt and his committee, these officers and councilors were unanimously elected by the delegates:

President-Elect—R. H. Wilson, M.D., Winona
First Vice President—Gordon MacRae, M.D., Duluth
Second Vice President—Mancel T. Mitchell, M.D., Minneapolis
Secretary—B. B. Souster, M.D., St. Paul
Treasurer—W. H. Condit, M.D., Minneapolis
Speaker, House of Delegates—C. G. Sheppard, M.D., Hutchinson
Vice Speaker, House of Delegates—H. M. Carryer, M.D., Rochester
Councilor, Third District—Peter E. Hermanson, M.D., Hendricks
Councilor, Fifth District—L. R. Critchfield, M.D., St. Paul
Councilor, Seventh District—W. W. Will, M.D., Bertha

The following nominees, whose names were submitted by The Council for delegates and alternates to the American Medical Association, were unanimously elected: O. J. Campbell, M.D., Minneapolis; George Earl, M.D., St. Paul; and C. L. Oppegaard, M.D., Crookston and E. M. Hammes, Sr., M.D., St. Paul, alternates, respectively.

The invitation of the Olmsted-Houston-Fillmore-Dodge County Medical Society to hold the 1956 meeting in Rochester was unanimously accepted.

There being no further business, the House of Delegates of the Minnesota State Medical Association adjourned its 102nd annual session at 1:50 p.m., May 23, 1955.

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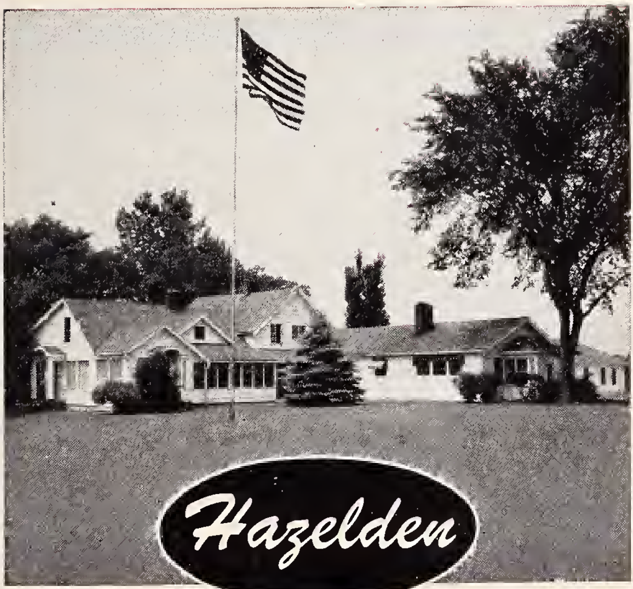

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Book Reviews

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THE DIABETIC'S COOKBOOK. By Clarice B. Strachan, Tulsa, Oklahoma. Introduction by Charles H. Best, M.D. Houston, Texas: Medical Arts Publishing Foundation, 1955. Price, $6.50.

The Diabetic's Cookbook, containing 250 recipes for preparing beverages, pastries, desserts, fruits, meats, salads, sandwiches, and vegetables, will be published this fall. American Diabetic Association values and exchange lists are used throughout the book. Also, pages are included for the diabetic to fill in his own diet prescription. Every recipe shows total amounts of carbohydrates, proteins, and fats as well as the caloric value of each serving. With this information plus the diet prescription, all in one book, the diabetic can readily prepare 250 delicious and tested dishes that fit in his diet.

Dr. Charles H. Best, one of the discoverers of insulin, has written the introduction. In it, he says, "I have no hesitation in recommending this book most highly and I feel sure that a very large number of diabetics—and their families—will welcome its publication and will profit by reading it."

The author of The Diabetic's Cookbook, Mrs. Clarice B. Strachan, of Tulsa, Oklahoma, developed and proved the recipes over a period of fourteen years, during which she cooked for her diabetic son. Mrs. Strachan has combined a strong scientific background with the delicate artistry of the kitchen to produce a volume which will add variety and zest to the diabetic's menu.

RADIATION THERAPY IN ORAL CAVITY CANCER

A report on the results from radiation treatment in cancer of the buccal mucosa and lower gingiva has been published by a University of Michigan Medical School radiologist.

He is Dr. Isadore Lampe, and his results were presented in the April, 1955, issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

A summary of his results shows:

From 1940 to 1951, fifty untreated carcinomas of the buccal mucosa in all stages of the disease were irradiated. Of thirty eligible for five-year survival rate computation, 50 per cent were alive without neoplasm at five years. For the entire group of fifty, an estimated five-year survival of 46.4 per cent has been computed.

During the same period, thirty-nine previously untreated carcinomas of the lower gingiva were irradiated. Of twenty-seven eligible, a five-year survival percentage of 29.6 was obtained. For the entire group of thirty-nine, the computed estimated five-year survival rate was 33.4 per cent. Of fourteen cases showing unequivocal bone destruction, four (28.6 per cent) were without recurrence at five years.

Abstracts

THE USE OF RADIOIODINE IN THE STUDY OF THYROID DISORDERS. Nicholson, J. P., Wilson, C. W., Newton, K. A. and MacLaren, R. G. C.: Radiology, 64: 567-571 (April) 1955.

A plaster-of-Paris cast and radioactive iodine have been combined by some British medical researchers to gain helpful information in the diagnosis and treatment of thyroid disorders.

Two physicists, J. P. Nicholson and C. W. Wilson, and two radiation therapists, Drs. K. A. Newton and R. G. C. MacLaren, all from London's Westminster Hospital, reported the findings.

"The first step in the investigation consists in making a plaster cast of the thyroid region and any other areas of interest. Several anatomical landmarks are painted on the cast and the surface is ruled into two-centimeter squares. Radioactive iodine is then given by mouth," the British research team reported.

"After an interval of about three hours, the plaster cast is again fitted to the patient and count-rate readings are taken at each two-centimeter interval. The readings are recorded on a count-rate meter," they continued.

From graphs which are prepared, they are able to compute the distribution of the radioactive iodine lying beneath the cast, relative to the anatomical landmarks.

From this information, certain diagnostic conclusions can then be drawn.

In one patient, on whom a thyroidectomy had been performed, six months after completion of irradiation following the operation, a tracer dose of 300 microcuries of radioactive iodine was given.

"The thyroid remnant was found to possess good concentrating power in spite of the heavy irradiation, and administration of thyroid extract was thus shown to be unnecessary," the authors pointed out.

In another patient, with lingual thyroid, a tracer test showed no iodine concentration below the thyrohyoid membrane. "Since the mass in the tongue was thus apparently the only functioning thyroid tissue present, its surgical removal was considered contraindicated," added the research team.

Other conclusions advanced by the British scientists: From the clinical point of view, the method offers a rapid means of investigating the iodine uptake in a particular region, though care must be taken in assessing cases in which there is no evidence of radioactive iodine uptake.

The patient suffers little inconvenience, attending in the morning to have the plaster cast made and to be given the radioactive drink. The afternoon attendance, when the readings are taken, consumes about half an hour.

Caution must be observed in the interpretation of radioactive iodine uptake studies of an isolated mass. Evidence of uptake indicates that the mass contains functioning thyroid tissue, but in the absence of uptake the result is equivocal. Loss of function may be the

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Bumbalo, T. S., Gustina, F. J., and Oleksiak, R. E.:
J. Pediat. 44:386, 1954.

White, R. H. R., and Standen, O. D.:
Brit. M. J. 2:755, 1953.

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Brown, H. W.:
J. Pediat. 45:419, 1954.

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result of thyroid disease or possibly of a lesion unassociated with the thyroid, and a high proportion of thyroid carcinomas show no pick-up.

A CLINICAL COMPARISON OF CARBINOXAMINE MALEATE, TRIPELENNAMINE HYDROCHLORIDE, AND BROMODIPHENHYDRAMINE HYDROCHLORIDE IN TREATING ALLERGIC SYMPTOMS. MacLaren, W. R.; Bruff, W. C.; Eisenberg, B. C.; Weiner, H., and Martin, W. H.: *Annals of Allergy,* 13:307 (May-June) 1955.

Although presently available antihistamines are reasonably efficient in counteracting histamine, complete relief of symptoms does not always follow their use and the numerous side effects, particularly somnolence, impair their usefulness in many patients. The new antihistamine, Clistin® Maleate (Carbinoxamine Maleate, McNeil) was developed with the aim of overcoming these objections. This antihistamine is compared in clinical effectiveness and incidence of side effects with two previously established antihistamine compounds—bromodiphenhydramine hydrochloride and tripelennamine hydrochloride.

Of the seventy patients in the series, forty-one had allergic rhinitis, twenty-six allergic rhinitis and asthma, 3 allergic rhinitis and eczema.

The amounts by which actual symptoms were reduced were, respectively, tripelennamine hydrochloride, 24.3 per cent, carbinoxamine maleate 18.9 per cent, and bromodiphenhydramine hydrochloride 14.3 per cent.

Subjective relief as reported by patients showed order of effectiveness: tripelennamine hydrochloride, bromodiphenhydramine and carbinoxamine maleate.

Sedation was estimated as follows: 1 plus if spontaneously noticed by the patient but causing no difficulty; 2 plus, if conscious effort was required to keep fully alert; and 3 plus, if effort was necessary to keep awake.

It is noteworthy that the order of subjective relief is the same as the frequency of producing somnolence. The sedative effect may make patients feel better than their symptom count indicates them to be.

Carbinoxamine maleate produced the fewest complaints of drowsiness as well as the lowest incidence of all side effects of the three antihistamines. Somnolence in particular was encountered only half as often. This suggests that larger doses than the recommended 4 mg. could be used, undoubtedly producing greater symptom control without raising side effects beyond an acceptable level.

PRACTICAL CONSIDERATIONS IN DIGITALIS THERAPY. Ehrlich, J. C.: Arizona Med., 12:239 (June) 1955.

In the 180 years that have passed since Withering, digitalis remains as the only drug we have which is effective in congestive heart failure by acting at the source of the difficulty, namely the failing myocardium. There are many different preparations of digitalis which are available to the physician today.

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but is used almost exclusively by the oral route. Approximately 20 per cent of it is absorbed from the gastrointestinal tract. The old tincture of digitalis is little if at all used today, and its chief faults lie firstly in the fact that it deteriorates with age and secondly in the fact that accurate dosage is not always possible because it is usually dispensed in drops. A similar whole leaf preparation exists from the lanata plant and is known as digilanid. It is little used today and offers no particular advantages.

Digitoxin is probably as widely if not more widely used than any of the other preparations of digitalis today. The reason for this, in the light of its pharmacology and physiology, is a bit difficult to understand. It was originally very popular because the medical profession was told that 1.2 milligrams of this drug would suffice to digitalize any and every cardiac patient. Actually, the digitalizing dose of this preparation varies from 1.2 to 2 milligrams, and the rate excretion will vary from individual to individual. Probably no more than forty per cent of the drug is excreted within two week after its use is discontinued, and it may take anywhere from four to six weeks to rid the patient completely of this preparation.

A good present-day all-purpose digitalis for general use is Gitaligin. This is completely absorbed from the gastrointestinal tract, and following discontinuance of its use it is almost completely excreted in anywhere from four to seven days. It has one particular advantage which is unique and places it apart from all other digitalis preparations. The digitalizing dose is approximately one-third of the toxic dose. With all other preparations which are available to us, digitalizing dose is two-thirds of the toxic dose.

We have a preparation which has a wider margin of safety and which will do anything therapeutically that any other preparation of digitalis will. It may be said to be the choice except in advanced myocardial disease with much fibrosis where excessive irritability is feared. In this particular case, the use of digoxin is recommended.

AEROSOL ADMINISTRATION OF ALEVAIRE. II. Clinical Evaluation, Smessaert, A.; Collins, V. I., and Kracum, V. D. (St. Vincent's Hospital, N.Y.C.) *New York State Journal of Medicine,* 55:1587, (June 1) 1955.

The rationale of therapy with surface-active compounds, their chemistry and properties are discussed. Alevaire (Triton WR-1339) is described as non-ionic, neutral, chemically stable, water and oil soluble. ". . . unlike many other detergents it is not hydrolyzed by tissue enzymes and thus retains its surface-active properties for a long time after administration. Its inertness makes it compatible with a wide range of

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INTENSIVE POSTGRADUATE COURSES
STARTING DATES—1955

SURGERY—Surgical Technic, two weeks, September 26, October 10
Surgical Technic, Surgical Anatomy and Clinical Surgery, four weeks, October 10
Surgical Anatomy and Clinical Surgery, two weeks, October 24
Surgery of Colon and Rectum, one week, October 17
General Surgery, two weeks, October 3; one week, October 17
Gallbladder Surgery, ten hours, October 24
Thoracic Surgery, one week, October 3
Esophageal Surgery, one week, October 10
Basic Principles in General Surgery, two weeks, September 26
Fractures and Traumatic Surgery, two weeks, October 17
GYNECOLOGY—Office and Operative Gynecology, two weeks, November 28
Vaginal Approach to Pelvic Surgery, one week, November 7
OBSTETRICS—General and Surgical Obstetrics, two weeks, November 7
MEDICINE—Two-week Course, September 26
Electrocardiography, one week advanced course, September 19
Electrocardiography and Heart Disease, two-week basic course, October 10
Gastroscopy, forty-hour basic course, November 7
Dermatology, two weeks, October 17
RADIOLOGY—Clinical and Didactic Course, two weeks, October 3
Clinical Uses of Radioisotopes, two weeks, October 10
PEDIATRICS—Clinical Course, two weeks, by Appointment
Pediatric Cardiology, one week, October 10 and 17
UROLOGY—Two-week course, October 10

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antibiotics and other therapeutic agents. The glycerin in the solution acts as a stabilizer of the aerosol droplets. The sodium bicarbonate creates an alkaline medium favorable for the liquefaction of the mucus and enhances the antibacterial activity of added antibiotics."

Untoward effects were never observed and there was no change in blood sodium levels although Alevaire was used in several patients for as long as six weeks. Administrations techniques are reviewed.

In a group of 300 patients with pulmonary congestion representing various age groups and clinical conditions, Alevarie aerosol inhalation gave "significant benefit" in 70 per cent (204) of the cases. In only 15 per cent of the cases was the Alevaire therapy considered a failure.

"In neonatal atelectasis results are impressive. This dreaded condition usually improved in a few hours, and it was really striking to see a cyanotic baby with gasping respirations and suprasternal retraction become relaxed and pink in such a short period of time. . . . In chronic respiratory conditions existing in adults this form of therapy can be most useful. . . . Secretions and sputum became less viscid, easier to expectorate, and less abundant; coughing is less exhausting, and ventilation improves."

Five respresentative case reports are included. In all of these patients, recovery was achieved with Alevaire aerosol.

The authors conclude that Alevaire is a most useful adjunct to other forms of therapy in respiratory diseases and that wetting agents properly administered are harmless to the patient, have no contraindications and can be used for prolonged periods of time.

USE OF COBALT AND IRON IN THE TREATMENT AND PREVENTION OF ANEMIA OF PREMATURITY. Coles, B. L., and James, U.: *Journal Lancet,* 75:79 (March) 1955.

The pathogenesis of the anemia of prematurity is still somewhat obscure, but the blood picture closely resembles that of anemia associated with infection. In each case the anemia is normocytic and normochromic with a low reticulocyte count, and this similarity between the anemia of prematurity and that of sepsis prompted an investigation of the effect of cobalt in premature infants.

The study included 126 infants who were divided into four groups. Of these eighty-three were followed for six months or longer. Group 1 acted as controls. Group 2 received 10 mg. of cobalt sulfate daily from one to twelve days. Group 3 received 20 mg. of cobalt sulfate daily from four to eight weeks. Group 4 received 20 mg. of cobalt sulfate and 4.5 gr. of ferrous sulfate daily from four to eight weeks.

Infants in Groups 3 and 4 combined had a significantly higher average hemoglobin content and red cell count

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at each examination from two months onward than Groups 1 and 2 combined. Infants in Group 4 had significantly higher hemoglobin contents from four to six months than Group 3, also receiving cobalt but no iron. At this stage iron deficiency becomes important in the development of anemia in premature infants, and these results were to be expected. No case receiving iron and cobalt from four to eight weeks required any additional therapy, but all cases that did were from the control group.

Cobalt appears to be of value in the prevention of early anemia in premature infants, and if iron is administered simultaneously, the risk of an iron deficiency anemia developing after the fourth month is considerably reduced. Cobalt has no toxic effects and no unfavorable influence on the weight gain in the dosage employed. The mode of action is uncertain, but two possibilities seem likely: (1) a direct action on the erythropoietic tissue in the marrow; (2) a possible catalytic action enabling available iron to be more readily utilized for hemoglobin synthesis.

What is to happen to the derelicts? Although tuberculosis occurs in any type, level or stratum, there are people especially prone—the dispossessed and the defeated, the disintegrated and the defenseless. The teacher who cannot get a job because of some x-ray shadow; the young man excluded from industry; the man who finds he cannot enter any superannuation scheme because of a past history of tuberculosis—what are we to do with them?—Harley Williams, M.D., *Nat. Tuberc. A. Tr.,* May, 1954.

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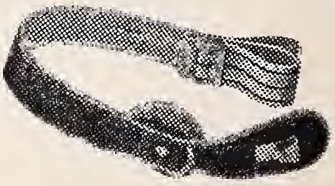

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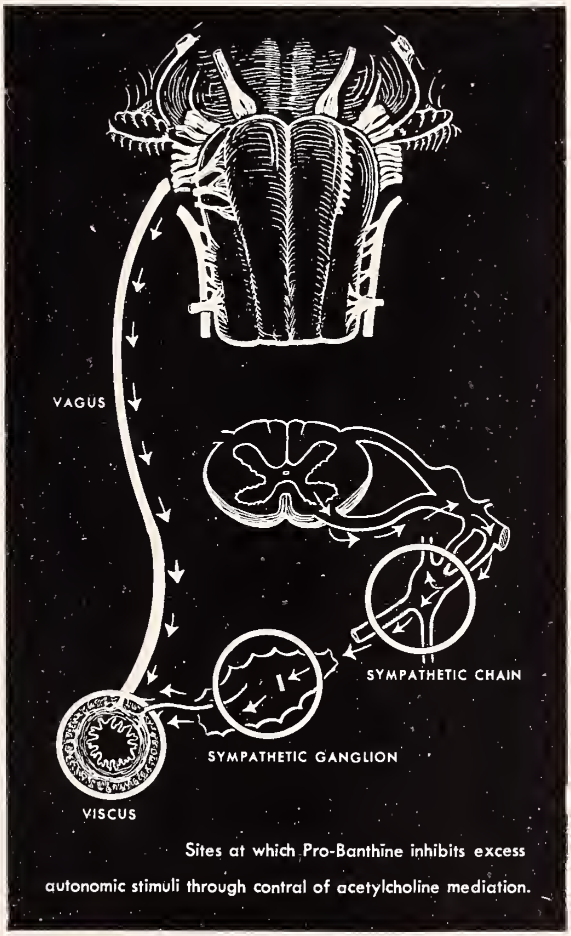
By controlling excess motility of the gastrointestinal tract, Pro-Banthine has found wide use[1] in the treatment of peptic ulcer, functional diarrheas, regional enteritis and ulcerative colitis. It is also valuable in the treatment of pylorospasm and spasm of the sphincter of Oddi.



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Roback and Beal[2] found that Pro-Banthine orally was an "inhibitor of spontaneous and histamine-stimulated gastric secretion" which "resulted in marked and prolonged inhibition of the motility of the stomach, jejunum, and colon. . . ."

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In Roback and Beal's[2] series "Side effects were almost entirely absent in single doses of 30 or 40 mg. . . ."

Pro-Banthine (β-diisopropylaminoethyl xanthene-9-carboxylate methobromide, brand of propantheline bromide) is available in three dosage forms: sugar-coated tablets of 15 mg.; sugar-coated tablets of 15 mg. of Pro-Banthine with 15 mg. of phenobarbital, for use when anxiety and tension are complicating factors; ampuls of 30 mg., for more rapid effects and in instances when oral medication is impractical or impossible.

For the average patient one tablet of Pro-Banthine (15 mg.) with each meal and two tablets (30 mg.) at bedtime will be adequate. G. D. Searle & Co., Research in the Service of Medicine.

---

1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25:*416 (Nov.) 1953.

2. Roback, R. A., and Beal, J. M.: Gastroenterology *25:*24 (Sept.) 1953.

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Miscellaneous

MEMBERSHIP INFORMATION

The following is information on membership in the American Medical Association and the Minnesota State Medical Association.

AMA Dues.—The American Medical Association House of Delegates set the dues for the coming year at $25. A subscription to the *Journal of the American Medical Association,* or another specialty journal, is included in the dues. Listed below are the specialty journals available. If members wish to receive a specialty journal in place of the *AMA Journal*, notice of this should be sent *directly* to the American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois. Available are:

AMA American Journal of Diseases of Children
AMA Archives of Neurology and Psychiatry
AMA Archives of Dermatology
AMA Archives of Surgery
AMA Archives of Ophthalmology
AMA Archives of Otolaryngology
AMA Archives of Pathology
AMA Archives of Internal Medicine
AMA Archives of Industrial Health

The AMA dues of a physician who joins his county society after July 1 will be $12.50; if he joins before July 1, his dues will be $25 for that year. Members may be excused, *on request,* from the payment of AMA dues, regardless of local dues exemption, on January 1, following their 70th birthday.

A physician who transfers from one state or county to another, will not be expected to pay the dues a second time; that is, he will not be expected to pay them in the state or county to which he has removed if he paid in the state or county from which he moved.

An active member is delinquent effective December 31, if he has not paid his dues for the past year. A notice will be forwarded to him by the AMA informing him of his delinquency, and he will forfeit his active membership in the AMA if he fails to pay such dues within thirty days after notice of his delinquency has been mailed by the AMA to his last known address.

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State Dues.—Dues for membership in the Minnesota State Medical Association for 1956 will be $40. All county society members are automatically members of the State Association, upon payment of dues.

State Membership.—Any doctor joining a county medical society should fill in an application blank and a biographical form, copies of which should be sent to the State Office for records, as this is one of the best ways in which necessary information can be secured concerning new members. County secretaries have been sent a supply of these blanks.

If a doctor who has not been a member of organized medicine previously, moves into a county and joins the society after the first of the year, he is entitled to prorated state dues. The year is divided on a quarterly basis and he should pay $30.00 if he joins after April 1; $20.00 if after July 1; and $10.00 if after October 1. A subscription to MINNESOTA MEDICINE is included in the dues.

Life Members.—A doctor who has been an active member of organized medicine for 40 years and has reached the age of 70 years is eligible for Life membership. He shall enjoy all the rights and privileges of active membership, and shall be subject to all the conditions of active membership. The privilege of receiving MINNESOTA MEDICINE shall be extended to Life members upon payment of the regular subscription price. Application for this type of membership must be approved by the Council.

Service Members.—Service members are those members of component county societies who have been temporarily called to active duty in the Armed Forces. Dues will be remitted from January 1 or July 1 following the date of the member's entrance into military service. All privileges of active membership are retained by the service member except he must pay the regular subscription price if he wishes to receive MINNESOTA MEDICINE. Application for this membership classification must be approved by the Council.

Honorary Members.—Honorary members are those who are distinguished for their services or attainments in the field of medicine, public health,

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research, or other scientific work contributing to medicine. Honorary members shall not pay dues and shall not have the right to vote or hold office.

Affiliate Members.—(Formerly associate members.) Affiliate members are those persons who are distinguished for their services in the allied sciences or in the field of public health. Affiliate members shall not pay dues and shall not have the right to vote or hold office.

Associate Members.—Associate members are divided into two classifications:

Regular Associate Members.—Associate members are those members of a county society who, because of disability, are unable to engage in the active practice of medicine or those members who have retired from the active practice of medicine, but had been members at the time they applied for such Associate membership. Provided that such Associate membership shall cease and active membership shall be reinstituted (1) upon termination of disability, or (2) upon resumption of active practice. Associate members who have had thirty years or more of membership in a state medical association shall have all the rights and privileges of active membership and will not be required to pay dues. Such Associate members will have the privilege of receiving Minnesota Medicine upon payment of the regular subscription price.

Junior Associate Members.—Associate members who have less than 30 years of membership in a state medical association and who shall be designated as Junior Associate members, shall not pay dues and shall not have the right of voting or holding office. Such Junior Associate members shall have the privilege of receiving Minnesota Medicine upon payment of the regular subscription price.

Applications for these classifications of membership shall be subject to the approval of the Council.

Residency Members.—Residency members shall be those members who are engaged in a residency program approved by the Council on Medical Education and Hospitals of the American Medical Association. Provided, however, that such member shall have been declared a residency member of such component society at its regular

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Less sodium retention, less potassium depletion

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meeting, such action having been approved by the Council. Such classification will include those members who are residents during the first five years following graduation, except that the time spent in the military service may be excluded in calculating the five-year limit. Residency members shall pay partial dues of $5.00 annually, which shall include subscription to MINNESOTA MEDICINE. Such residency membership shall cease upon termination of participation in graduate study or work.

INSURANCE PLAN TOPS GOAL

The Minnesota State Medical Association's group health and accident insurance plan, passed by the House of Delegates at the May meeting in Minneapolis, has topped the goal by a good margin, and is now in effect. In order for the entire plan to be effective, a goal of 50 per cent of the Association membership, excluding Olmsted and Hennepin county members, had to be reached by September 1. At that date, the goal was more than reached. The administrator extended the charter enrollment period to September 30 in order that more members could take advantage of the benefits offered in the plan. It is pleasing to report that as of September 30, 1955, approximately 1,200 Association members in active practice had enrolled in the plan. The plan is administered by the Charles O. Finley Company of Chicago, and is underwritten by the North American Accident Insurance Company. The Insurance Liaison committee of the Association, Dr. Corrin H. Hodgson, Rochester, chairman, is very pleased with the enrollment figures, and wishes to thank members in helping to make the plan the large success it has turned out to be.

AMEF CAMPAIGN TO START

This year the Minnesota State Medical Association is planning a concerted effort to increase contributions to the American Medical Education Foundation fund, by a stepped-up campaign among its members. The campaign, headed by Dr. H. E. Drill of Hopkins as chairman of the state committee, will be started as soon as possible, and

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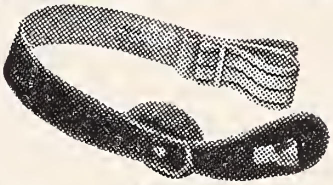

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STARTING DATES—FALL, 1955

SURGERY—Surgical Technique, two weeks, October 10, November 7
Surgical Technique, Surgical Anatomy and Clinical Surgery, four weeks, October 10
Surgical Anatomy and Clinical Surgery, two weeks, October 24
Surgery of Colon and Rectum, one week, October 17, November 28
General Surgery, one week, October 17
Gallbladder Surgery, ten hours, October 24
Fractures and Traumatic Surgery, two weeks, October 17

GYNECOLOGY—Office and Operative Gynecology, two weeks, November 28
Vaginal Approach to Pelvic Surgery, one week, November 7

OBSTETRICS—General and Surgical Obstetrics, two weeks, November 7

MEDICINE—Gastroenterology, two weeks, October 24
Electrocardiography and Heart Disease, two-week basic course, October 10
Gastroscopy, forty-hour basic course, November 7
Dermatology, two weeks, October 17

RADIOLOGY—Clinical Course, two weeks, by appointment
Clinical Uses of Radioisotopes, two weeks, October 10

PEDIATRICS—Clinical Course, two weeks, by appointment

UROLOGY—Two-week Course October 10

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members are urged to do all they can to help make it a success.

Last year in Minnesota only 138 physicians made any contribution to this fund, which is set up to collect funds for the seventy-nine medical schools of the nation. The amount collected was slightly over $5,000 for 1954.

This year's campaign will be organized a little more closely, with county society co-operation as its emphasis. Each councilor district of the State Association has a district chairman, and chairmen have been appointed for each of the county medical societies, to help arrange the campaign, and to work toward its successful completion.

Origin of the fund and the annual campaign is explained: The National Fund for Medical Education was set up a few years ago, with a federal charter from Congress, and sponsored by the American Association of Medical Colleges and leaders in industry. Its purpose is to raise $8,000,000 each year from industry for medical schools. The American Medical Association approved the program and organized a separate fund-raising unit for doctors. The American Medical Education Foundation's annual quota is $2,000,000.

If industry is to continue to pay $8 million a year in support of medical schools, it is necessary for physicians to take on a share of the burden, and it is easier to solicit successfully if doctors are also giving their share.

At present all institutions need funds. New equipment is needed and expenses are up. Other funds contributed are usually earmarked for special purposes. Minnesota's medical school has received approximately $102,000 from the Foundation, and it has often made the difference in carrying on an important project or dropping it. From 1952 to May, 1955, Minnesota doctors had contributed only $27,000.

It should be pointed out that all money that is contributed is given to medical schools; all promotion costs of the entire campaign are underwritten by the AMA. A donation can be earmarked for a specific medical school, but not a department, or to the AMEF general fund and at the end of the year it will be prorated among all medical schools.

Dr. Drill, his state committee, the district chairmen, and the county medical society chairmen, have pledged their continued efforts in this pro-

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gram, and they ask that all members consider the extreme importance of giving to this fund when they are solicited on a local basis in the near future.

RETROLENTAL FIBROPLASIA PRACTICES APPROVED

The Committees on Child Health and Ophthalmology of the Minnesota State Medical Association, as well as the Association's Council, have approved some recommended practices in the prevention of retrolental fibroplasia. These practices are:

1. It is recommended that the routine administration of oxygen to premature infants be discontinued. When the physician specifically orders oxygen administration to premature infants, the incubator oxygen concentration should be kept under 40 per cent.
2. To insure incubator oxygen concentration under 40 per cent, one of the following devices may be employed:

(a) Melco minus 40 per cent oxygen concentration device which is attached directly to the incubator with metal fittings and makes it impossible to administer oxygen in excess of 40 per cent concentration, regardless of oxygen flow. This is manufactured by the Melchior, Armstrong, Desseau Co., Ridgefield, New Jersey. Price, including humidifier which may or may not be used, for hospitals with piped-in oxygen—
Melco 758—$48.50 f.o.b., Ridgefield, New Jersey.
for hospitals without piped-in oxygen—
Melco 759—$78.00 f.o.b., Ridgefield, New Jersey.

(b) Analysis of oxygen concentration of incubator between one and two hours after turning on oxygen supply to incubator. There are two types of oxygen analyzers which are simple to operate and operate by means of contained batteries:

(1) The Mira oxygen analyzer OXA-100 is available from the Medical In-

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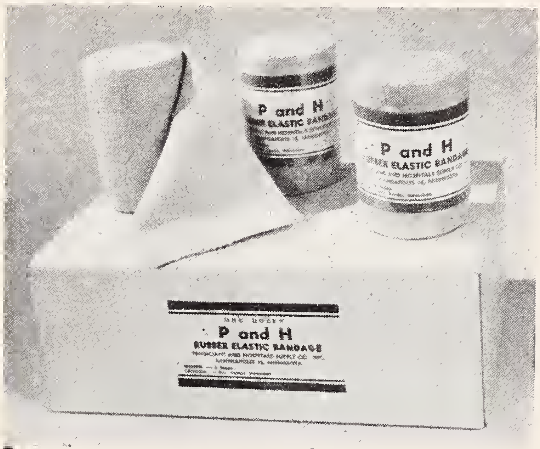

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A heavy duty extra fine quality elastic bandage. Because of color, flat woven edges and smooth finish they are inconspicuous in use. Stretch approximately double their length, roll evenly, fit comfortably and exert a constant firm pressure and support. Especially useful in cases requiring strong, continuous pressure. Live rubber prevents the loss of elasticity yet will not inhibit circulation. Washing will not deteriorate these bandages.
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strument Research Associates, 265 N. Pasadena Avenue, Los Angele 31, California. Price $147.00 f.o.b. Los Angeles, California.

(2) The Arnold O. Beckman Model D oxygen analyzer is available from Arnold O Beckman, Inc., 1020 Mission Street, South Pasadena, California. Price $190.00 f.o.b., South Pasadena, California.

(c) If the Melco minus 40 per cent concentration device is used, it is not necessary to use an oxygen analyzer. It is felt that the Melco device is more desirable and easier to use than the oxygen analyzers.

COUNCIL APPROVES AVIAN TB STUDY

Avian tuberculosis, hitherto regarded as unimportant in the control of tuberculosis, has emerged as a new problem in the spread of the disease in the human population, as well as in cattle and swine.

Dr. John F. Briggs, St. Paul, chairman of the Committee on Tuberculosis of the Minnesota State Medical Association, told the Council at its September meeting that one death and three cases of tuberculosis of the avian origin had occurred in Minnesota recently, and that veterinarians are encountering non-specific reactions to tuberculin among herds, many of which they suspect can be traced to contact with infected poultry in farm yards.

An area survey of tuberculosis in poultry in cooperation with the U. S. Public Health Service and veterinarian groups, is under consideration by the committee, Dr. Briggs reported. The Council approved participation by the committee in this survey.

HEART COMMITTEE QUESTIONNAIRE

The Heart Committee of the Minnesota State Medical Association, in cooperation with the Minnesota Heart Association, is conducting a survey of State Association members regarding the incidence and treatment of rheumatic fever in the state.

The committee's letter and questionnaire were mailed out in September, and returns on the questionnaire have been very good. Analysis of the information in the answers to the questions is now being done.

Physicians who have not as yet returned their questionnaire are urged to do so at once in order that figures arrived at will be as accurate and complete as possible.

THOSE DIFFICULT PATIENTS

There is a daily parade of people through almost every physician's office. Some of these individuals by their actions and attitudes slow down or bring to a complete halt this orderly procession. These are the patients doctors label as "difficult"—the shoppers, the talkers, the belligerents, and occasionally, those who are dissatisfied. These individuals complicate the task of running an efficient medical office and present definite public relations problems as well. Some are touchy and easily annoyed; others are inclined to become dissatisfied with the doctor or their medical care quickly. It has been found by medical societies that many of the complaints about medicine and the medical profession today are being voiced by such problem patients.

Consequently, even though such patients are trying to the physician, they must be dealt with thoughtfully and with special consideration. What makes certain patients "difficult?" Almost all of them reflect anxiety about their conditions. Some are distrustful of the doctor and suspicious of any medical procedure. Others are unable to cope with trying situations in life and so they take refuge behind physical symptoms. Many are reacting in a negative fashion to their illness and to the position of authority of the physician they have been forced to seek.

Dr. Howard P. Rome of Rochester, Minn., says: "Obviously, the static approach to clinical medicine which treats all patients as though they were simply the non-contributing carriers of diseases or injuries will yield a high percentage of difficult patients. Everything depends upon how the physician responds to the patient's recital of worries, whether with pointed inquiries, a shrugging dismissal of a symptom, or an enigmatic blandness that leaves the patient in an even worse quandary." Dr. Rome believes every patient requires a different method of approach according to the individual's psychology. Certain techniques are involved in these varying approaches.

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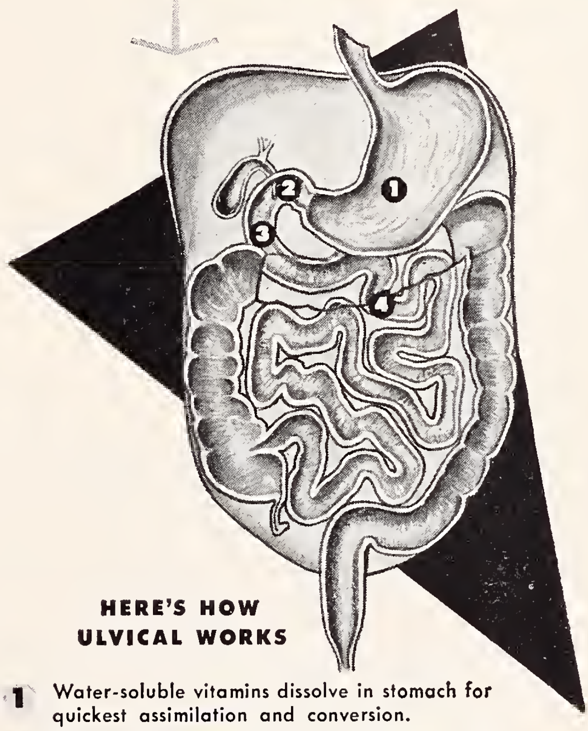

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Calcium Pyrophosphate *(Ca. 150 mg.)*	7.5 gr.
Ferrous Sulfate USP *(Fe. 38 mg.)*	3 gr.
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Here are some suggestions from practicing physicians for dealing with difficult patients:

Shoppers.—Every physician—especially a man just setting up practice—is confronted from time to time with "shoppers." These patients move from doctor to doctor, searching for the words they want to hear. Sometimes these individuals are seeking a contradictory diagnosis, sometimes the lowest-priced medical care; sometimes they are not sick at all but are searching for a doctor who will say there is a physical basis for their mental ailments. Should a doctor criticize a diagnosis that another physician made previously such patients are inclined to seek compensation for their earlier "mistake" or to complain loudly to the neighbors and friends, thus undermining public confidence in physicians. Shoppers should be firmly urged to return to their own doctors, unless they have actually terminated the earlier doctor-patient relationship. Physicians recommend in such cases complete histories be taken, and they warn against talking disparagingly against other physicians.

Talkers.—"Gabby" individuals run on forever if allowed to, taking up more than their share of the doctor's time and sabotaging the appointment schedule. Sometimes, in an early conversation with such persons, the doctor's receptionist can spot a "talker." In some offices, talkers' cards or case histories are marked with a symbol that alerts the physician. Other doctors have their assistants pointedly remind them when bringing in a talker that the next patient will arrive promptly on schedule. The physician must phrase his questions carefully when interviewing such patients, since the more general his questions the longer the answers will be. One doctor says that a firm interruption now and then to get the conversation back on the track can be used without the patient's taking offense (talkers are used to being interrupted by others). Some doctors use a concealed buzzer to signal their assistant when it seems impossible to conclude a visit with a talker in a reasonable length of time. The assistant then enters to say: "Doctor, I'm afraid you've run overtime. Your next patient has arrived." Another effective way to cut short lengthy, time-consuming conversations with such patients is to suggest they return for another appointment to discuss their condition. Few patients want to pay for another visit just to talk.

Belligerents.—Some patients actually seem to elight in disobeying the doctor's instructions. No matter what the psychological reasons for heir rebellion, the physician should try to conince such patients that his orders have merit and re to the patient's best interest. He may have o state firmly: "I will no longer serve as your hysician if you don't follow my orders. I won't e responsible for what happens if you do not o-operate." Often, the doctor should put such statement in the form of a letter to the patient o protect himself.

Dissatisfied Patients.—The physician whose lissatisfied patient brings the grievance directly o him for discussion is fortunate. Generally, uch persons complain loudly to friends and neighbors about their grievance, but the doctor ears about it only indirectly, if at all. When a atient does complain, the physician must listen atiently, even if the complaint seems ridiculous r unjustified. What are the reasons for the omplaint? Is misunderstanding the cause of the rouble? Or is the complaint justified? In many nstances the doctor can settle the complaint to the patient's satisfaction after a few minutes' discussion. When the patient cannot be satisfied, the doctor should suggest the patient put his complaint in writing and submit it to the local medical society's mediation committee for hearing. Such committees convince patients that physicians sincerely are trying to settle grievances justly.

In most cases, problem patients can be turned into amiable, co-operative patients. Each difficult patient presents a challenge to a physician's dexterity in human relations.—J.A.MA., Oct. 1, 1955.

PROBLEM CORNER

No physician enjoys crawling out of bed during the night to answer an emergency call. He knows from actual experience that the majority of such calls are false alarms. Yet—one out of ten such calls may be a real emergency. Since no doctor has a crystal ball to help him differentiate over the telephone the real from the fancied emergencies, he takes a great risk in refusing any such call. The one emergency case that goes

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Inflammatory skin conditions

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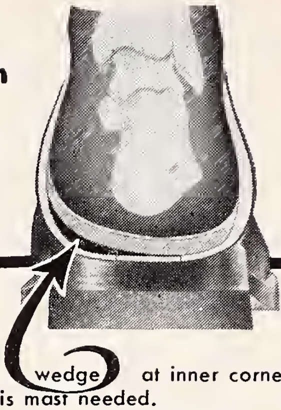
Foot-so-Port Shoe Construction and its Relation to Weight Distribution



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untended creates a tremendous public relation problem for the entire profession. Here's wha happened in one midwestern city recently:

At 5:45 a.m. one Sunday a frantic fathe told a telephone operator his five-year-old daug ter was in convulsions and that he could nc remember the telephone number or spellin of his doctor's name. The operator quickl traced the doctor and put through a call to hir His wife answered and berated the operator fo calling the doctor so early. When the father wa put on the line and explained that the inhalato squad had already been called to his home t revive the child, the wife finally allowed him t speak to the doctor. The physician agreed t accept the call. The doctor's wife then cam back on the line and told the operator not t ring them for any more emergency calls, and th doctor reiterated the request in somewhat abusiv terms.

Because of the telephone operator's persistence tragedy was averted. The doctor admitted to the father he was glad the parents insisted on his coming at once, as the little girl was more seriously ill than he had imagined. The grateful mother later wrote a letter of appreciation to the telephone company praising the efforts of the operator in securing a doctor. The telephone company got good public relations out of the incident, but how much ill will was engendered by the attitude of the physician and his wife in this instance? The telephone company official who reported the incident stated in a letter that there were "too many such occurrences."

The medical profession has accepted the responsibility for providing emergency medical care. What can the profession do to assure that individual physicians do not shirk this responsibility? The physician's wife must understand the nature of this responsibility, too, since she often can do irreparable harm to the public relations of the individual doctor. Public relations is more than just the responsibility of the physician—it is the responsibility of his wife, his family, and all those associated with him in his practice.—J.A.M.A., Oct. 1, 1955.

ENCOURAGING TRAFFIC SAFETY

Few persons are better qualified to point out to the public the terrible consequences of traffic accidents than physicians. By promoting traffic safety, physicians in a number of communities

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re rendering a real community service and in-idently netting better public relations. A War-enton, Va., physician reports one effective traf-ic safety promotion accomplished in his town. 'In our town when the highway department re-used a stop light for a dangerous intersection, ve sent a delegation of two doctors (one repre-enting the local hospital) and an undertaker to xplain how 'profitable' that intersection had been. The hospital and undertaker's bills were quite mpressive. We got *two* stop lights and at once."

In a similar move, physicians of the Schuyler County (N.Y.) Medical Society called attention of county officials to a dangerous curve near an ntersection. The letter pointed out that one erson had been killed and several injured at the pot. Action to abolish the curve was urged. The community newspaper used the society's let-er as the basis for an editorial and further em-hasized the need for action. Medical public re-ations need not be limited to medical affairs. The opinion of the medical society on any com-nunity situation often carries great weight. Voic-ng that opinion constructively often results in mproved public relations for the profession.—.A.M.A., Oct. 1, 1955.

MODERN CESAREAN SECTIONS

Obstetrical statistics indicate that the incidence of maternal deaths is being markedly lowered. Ten years ago, there was one death in about 450 deliveries; five years ago, one in about 950; but in 1953, there was less than six in 10,000 live births. The authors suggest that "factors resulting in the amazing reduction in maternal mortality in the era of modern obstetrics are reflected in the increased incidence and safety of cesarean section."

A series of 1000 consecutive cesarean sections from St. Elizabeth's Hospital in Boston, Massachusetts, were studied. Of the primary sections performed, "three times more . . . were done in the hope of obtaining a live and unharmed infant than for a predominantly maternal indication." However, it was felt that "it was no more dangerous to perform the operation in the interest of the mother than in that of the child." The morbidity rate and number of neonatal deaths were not increased by the number of previous sections. It was also determined that "sterilization after cesarean section is unnecessary."—Sullivan, C. L., and Campbell, E. M.: One thousand cesarean sections in the modern era of obstetrics, *Obstetrics & Gynecology,* 5:669 (May) 1955.

Classified Advertising

Replies to advertisements with key numbers should be mailed in care of Minnesota Medicine, *2642 University Avenue, Saint Paul 14, Minnesota.*

WANTED—General practitioner to assume large practice of M.D. in service. Ten-room, first-floor clinic, well equipped. Reasonable rent. Gross over $25,000. New hospital—unlimited privileges. Good housing. Partnership available after my return. Small town near Twin Cities. Address E-486, care Minnesota Medicine.

WANTED—Associate in general practice. Town of 500, 25 miles from Twin Cities. Salary depending on qualifications. Early partnership. Good housing, hospital facilities, and schools. Address E-493, Minnesota Medicine.

WANTED RESIDENTS IN PSYCHIATRY—Opportunity for beginning and advanced candidates in five-year Minnesota State program offered in collaboration with University of Minnesota and Mayo Foundation. Must be acceptable to above training centers. Stipends $5,000 to $7,000. Direct reply to Dale C. Cameron, M.D., Medical Director, Minnesota State Department Welfare, 117 University Avenue, Saint Paul 1, Minnesota, giving full information about training and experience.

MILLE LACS LAKE AREA—Excellent opportunity General Practitioner and Surgeon. New 27-bed hospital operated by Franciscan Sisters, 80 miles north of Minneapolis. Early decision necessary. Contact J. E. Murray, Onamia, Minnesota.

WANTED—General practitioner for State Hospital. Beginning salary $640.00 per month. Three bedroom house available, partially furnished, subsistence, garage. Government working conditions, paid holidays, vacation, sick leave, and retirement pension plan. Write Superintendent, Fergus Falls State Hospital, Fergus Falls, Minnesota.

QUALIFIED LABORATORY TECHNICIAN wants position in doctor's office or clinic. Minnesota preferred but not essential. Three years' laboratory experience in 41-bed hospital. Good references; single, 25 years old. Can do typing. Like meeting people. Can begin with two weeks' notice. Address E-496, care Minnesota Medicine.

FOR RENT—Medical Building, 28 x 50 feet, 7 rooms, 2 dressing rooms, 2 half-baths, darkroom and storage rooms. Located in center of Scottsdale. Roy Peterson, Scottsdale, Arizona.

PLACEMENT SERVICE

The Minnesota State Medical Association maintains a Medical Placement Service for the benefit of physicians who are looking for locations and positions; also for communities, medical groups and physicians who are looking for licensed medical assistance. For information, write to the Minnesota State Medical Association, 496 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota.

FOR RENT—New 4-room, first floor office in fast-growing Minneapolis suburb of about 4,000. Private front and rear entrances. Adequate parking space. Telephone: WE 8-9660.

SPACE FOR RENT—Highland Park, Saint Paul. Share full facilities with surgeon and dentist. Call DeSoto 3893, Saint Paul, or write 2069 Ford Parkway, Saint Paul 5.

ATTENTION DENTISTS AND DOCTORS—Modern space available for dentists' and doctors' offices. A location where several sets of doctors and dentists have been long established and then built their own clinics. A money maker. Above Kisch's Food Market. Plenty of auto parking space. Apply 637 Stryker Avenue, Saint Paul, Minnesota.

FOR SALE OR LEASE—Doctor's office with equipment. An old-established practice with completely modern equipment. Directly across the street from hospital. Brick building. Write Box 338, Starbuck, Minnesota.

DOCTOR OR DENTIST—Beautiful suite in South Minneapolis. Very modern. Six months free rent. Then option of lease at very reasonable rent. Wm. L. Cochrane, 4101 - 42nd Avenue South, Minneapolis, Minnesota. Telephone Ro 9-8758.

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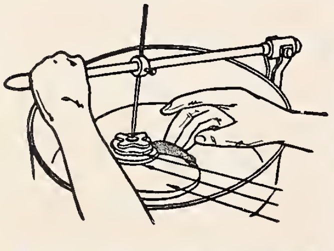

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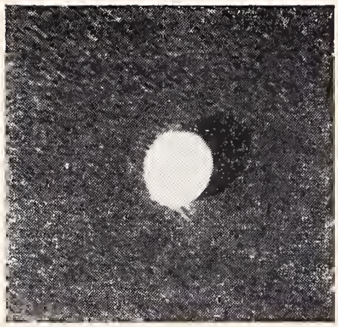

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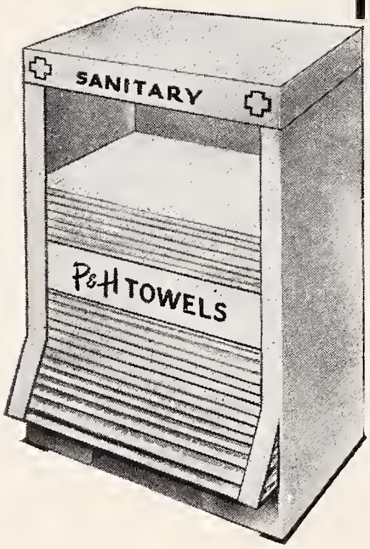

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Can be autoclaved and used for sterile drapes. 14 x 18¼ inch size . . . pure snowy white and packed 500 to the case.

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(Continued from Page 828)

Several hundred surgeons from throughout the world gathered at the symposium to pay tribute to Dr. Wangensteen, whose surgical contributions have been both numerous and important since he first became surgery head at the university at the age of thirty-two. A testimonial dinner was held in his honor at the Radisson Hotel, Minneapolis, on September 23, with John Russell, director of the John and Mary Markle Foundation, as the principal speaker.

* * *

Dr. Frederick A. Figi, head of the plastic surgery section at the Mayo Clinic, Rochester, was elected president of the American Society of Plastic and Reconstructive Surgery at its meeting in Atlantic City on September 29. Later Dr. Figi was made a member of the Carlos J. Finlay National Order of Merit of Cuba. Only three physicians outside of Cuba have been given the award, one of them being **Dr. Philip S. Hench,** also of the Mayo Clinic.

* * *

Dr. Robert A. Good, research professor of pediatrics at the University of Minnesota, was named the 1955 recipient of the E. Mead Johnson award for pediatric research. The award was made at the annual meeting of the American Academy of Pediatrics in Chicago on October 5.

* * *

Principal speaker at a meeting of the Parent-Teachers Association at Sacred Heart on October 12 was **Dr. F. T. Sorum,** member of the staff of the Willmar State Hospital. Dr. Sorum discussed various aspects of mental health.

* * *

Dr. Frederic E. B. Foley, chief of urology at Ancker Hospital, St. Paul, and clinical professor of surgery at the University of Minnesota, was given a Francis Amory award at the American Academy of Arts and Sciences meeting in Boston on October 5. The award was in recognition of Dr. Foley's work for the relief or cure of genito-urinary diseases.

* * *

"New Drugs and Treatments" was the title of a talk given by **Dr. Roy Good** of Glenwood at a meeting of the Pope County Nurses Club at the Glenwood Hospital on October 10.

* * *

New directors of the Minnesota Medical Foundation elected to office on September 27 include **Dr. N. Logan Leven,** St. Paul; **Dr. Lawrence R. Boies,** Minneapolis; **Dr. Bernard Halper,** Hibbing, and **Dr. Raymond D. Pruitt,** Rochester. President of the foundation is **Dr. Wesley W. Spink,** professor of medicine at the University of Minnesota. **Dr. Robert B. Howard,** director of continuation medical education, is secretary-treasurer. The foundation recently presented scholarships totaling $10,750 to twenty-one medical students.

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NEW LOCATIONS

Dr. Robert H. Nelson, formerly of International Falls, has become associated in practice with **Dr. Donald Holm** at Benson.

Dr. F. J. McCaffrey, who has been practicing obstetrics and gynecology in Minneapolis, has opened offices in Wayzata.

Dr. Edgar R. Gamm, recently of Pipestone, has opened offices for the practice of medicine at Triumph.

Dr. Sam P. McDaniel, formerly of Virginia and recently on the staff of the state hospital at Fergus Falls, has opened offices for the practice of medicine at Lakeville.

Dr. Carol L. Plott, formerly of Lancaster, Pennsylvania, has become associated in practice with **Dr. R. W. Nicholson** at Heron Lake.

Dr. C. B. Will, who practiced at Bertha for fifteen years, has moved to International Falls and is practicing there.

Dr. Henry B. Blumberg, after a year's absence from Fairmont, during which he served as chief resident physician in internal medicine at San Joaquin General Hospital near Stockton, California, has returned to Fairmont and reopened his offices.

Dr. Richard E. Payne, formerly practicing pediatrics with the Kansas Medical Center in Kansas City, has joined the staff of the East Range Clinic at Virginia.

Dr. Eugene V. Meyerding, son of **Dr. E. A. Meyerding,** executive secretary of the Minnesota Tuberculosis and Health Association, and a former resident of St. Paul, has opened offices for the practice of surgery at Medford, Oregon. For the past three years he was chief of the surgical service at the Air Force's Keesler Base Hospital near Biloxi, Mississippi.

MINNESOTA BLUE SHIELD-BLUE CROSS

Since 1933 to date, Minnesota Blue Cross has provided 11,160,345.6 days of hospital care for 1,840,947 subscribers totaling in excess of $124,000,000 according to figures just released from their statistical department.

During the first eight months of 1955, Blue Cross provided 681,339 days of hospital care for 110,953 Minnesota Blue Cross subscribers, as compared to 673,514.3 days of hospital care for 112,787 subscribers during the same period of 1954.

The average number of hospital days per case increased from 6.0 days to 6.1 during the same eight months period.

Although the number of Blue Cross subscribers hospitalized has shown a slight decrease, the number of days of care has increased, due to the increased number of days of care per case.

Decrease in the number of cases can be attributed to a noticeable decline in tonsillectomies. There were 7,243 such operations in the first eight months of 1954, compared to 4,690 during the same period in 1955. Although

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tonsillectomies average only 1.3 days per case, a decline in this category noticeably affects the overall length of stay.

During the first eight months of 1955, 15,471 Blue Cross babies were born compared to 14,974 during the same period of 1954. Maternity care totaled 18,871 cases during 1955, compared to 18,649 during 1954. Maternity benefits exceeded $2,000,000 during the first eight months of 1955, compared to $1,830,000 during the same period in 1954.

Accident cases showed only a slight increase during the first eight months of 1955 over the same period in 1954. During the first eight months of 1954 there were 17,025 cases compared to 17,103 during the same period in 1955; however, in this category, Blue Cross benefits paid during the first eight months period totaled $823,000 in 1955, as compared to $728,000 for the same period in 1954.

On November 1, 1955, Minnesota Blue Shield will be eight years old. Some of the highlights of its development and growth are worthy of consideration.

During November and December of 1947, the first two months of operation, 6,500 participant subscribers were enrolled. As anticipated, progress in 1948 was rapid. During this year, 100,666 participant subscribers were enrolled and Blue Shield paid $169,269 to doctors for services rendered the subscribers.

By the end of 1950, Blue Shield enrollment had increased to 411,733 participant subscribers, and during this year Blue Shield paid $2,249,032 to doctors for their services rendered participant subscribers, or over $2-million more than was paid in 1948. During March of 1950, Blue Shield established its own separate claims department.

Prior to 1952, a number of increases and improvements of Blue Shield benefits were made, and on January 1, 1952, the first and only increase in the subscribers' rates became effective. This made possible many increases of fees and additional benefits which were put in effect on October 1, 1952. Again on January 1, 1954, many Blue Shield allowances were increased, and on March 1 of that year a new, higher benefit, higher service income limit contract was issued.

Both enrollment increases and claims cost reflected these developments. At the end of the year 1954, enrollment had increased to 670,757 participant subscribers, and during 1954 Blue Shield paid almost $5-million, $4,970,231 to doctors for subscribers' medical, surgical and obstetrical care.

On July first, 1955, major increases in Blue Shield benefits again were provided. By the end of August, 1955, Blue Shield had 706,539 participant subscribers, and had paid $3,774,781 in claims during the first eight months of 1955. Should the average monthly cost of claims continue as it has been during 1955, by the end of this year Blue Shield will undoubtedly reach a total of $25-million paid to doctors since its organization for services rendered its participant subscribers.

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Indications:
Rheumatoid arthritis
Bronchial asthma
Inflammatory skin conditions

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Book Reviews

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

THE MECHANISMS OF HEALING IN HUMAN WOUNDS. Shattuck W. Hartwell, B.S., M.S., M.D., Ph.D. in Surgery, F.A.C.S., F.I.C.S. Diplomate of the American Board of Surgery; Attending Surgeon, Hackley Hospital and Chief of Surgery, Mercy Hospital, Muskegon, Michigan; Surgeon United States' Army's 237th Station Hospital (1943-1944) (Utica New York and New Guinea); Chief of Surgery, United States Army's 120th General Hospital, Manila, P. I., 1945; Member, New York Academy of Sciences. 166 pages. Illus. Price $4.75, cloth. Springfield, Illinois: Charles C Thomas, 1955.

PLANNING FLORIDA'S HEALTH LEADERSHIP. Medical Education in the University. Edited by Louis J. Maloof. 161 pages. Price $1.40, paper cover. Gainesville, Florida: University of Florida Press, 1955.

CHLOROPROMAZINE AND MENTAL HEALTH. Proceedings of the Symposium held under the auspices of Smith, Kline & French Laboratories, June 6, 1955, Warwick Hotel, Philadelphia. 200 pages. Illus. Price $3.00, cloth. Philadelphia: Lea & Febiger, 1955.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY. Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.R.S.E.; Emeritus Surgeon, Royal Northern Hospital, London; General Surgeon, Metropolitan Ear, Nose, and Throat Hospital; Consulting Surgeon, Italian Hospital, etc. 12th ed. 456 pages. Illus. Price, $8.00. Baltimore: Williams & Wilkins, 1955.

There are only a few medical texts which the average physician can read in an easy, comfortable fashion, but among these few "friendly" volumes must be included this latest revision by Hamilton Bailey. In this small volume, the author has combined a maximum of readability with a concise presentation of years of experience combined with astute observation. Every student of medicine, regardless of age or interest, will find the reading of this text a refreshing and stimulating experience, for the combination of clinical acumen and references to literature from the Greeks to Shakespeare make this book a classic in its field.

Dr. Bailey first discards all laboratory and special diagnostic aids; then, armed only with the five senses does he proceed to examine each patient from head to toe. The reader will find himself accompanying Dr. Bailey through the wards of the London hospitals, looking over the shoulder and through the mind of a careful, logical, and clever clinician. The liberal use of photographs and diagrams, some which bear notations

(Continued on Page xlv)

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Inflammatory skin conditions

(Continued from Page xliii)

in Dr. Bailey's somewhat illegible handwriting, allow the author to make the text pertinent and to avoid involved descriptive passages. Although the title of the book would appear to limit its usefulness to those in the surgical specialties, the actual subject matter concerns those diagnostic problems confronted by every physician in his daily practice.

This book has been carefully compiled, and its historical footnotes are of special interest. Admittedly elementary in its approach, this volume should serve as a whetstone to the skill of every clinician, and as such, be a valuable addition to his personal library.

JOHN V. THOMAS, M.D.

MANAGEMENT OF OBSTETRIC DIFFICULTIES. 5th Edition, Paul Titus. Revised by J. Robert Willson, Professor of Obstetrics and Gynecology, Temple University School of Medicine, Philadelphia, Pennsylvania. 737 pages. Illustrated. Price, $12.50. St. Louis: C. V. Mosby Company, 1955.

The aim of this fifth edition under the authorship of J. Robert Willson is that of the original author, Paul Titus, namely: ". . . to develop a practical aid to physicians who are meeting and contending with obstetric difficulties and emergencies." It certainly fulfills its purpose in being just that—a quick reference to the more practical aspects of obstetrics, to problems that arise in any obstetric practice.

This new edition has been considerably revised both as to organization, additions, and omissions. The presentation is easy reading, the type that encourages one to read on. The material presented avoids the theoretical and controversial and aims at everyday practice as experienced by the author and his associates. A brief recent biography is appended at the end of each chapter. Illustrations are good and plentiful, and the index is adequate.

To those of vast obstetrical experience, this book does not have much to offer, but to those of lesser experience, it is an excellent volume to have around at the time those difficulties arise.

R. T. SEASHORE, M.D.

SCIENCE EXHIBITS. Edited by Helen Miles Davis, Editor of *Chemistry* magazine. 100 pages. Cloth Bound, $2.00 Washington, D. C.: Science Service, 1955.

For young scientists and teachers, particularly, who are planning participation in science fairs and who are starting on their own projects, this new book is a valuable aid.

Science Exhibits tells how to select material, how to plan its presentation, how to display, to label and to light it, how to decribe the work done, how to tell about it so that those who think science is "too difficult"

(Continued on Page xlvii)

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(Continued from Page xlv)

will understand and share the enthusiasm of the exhibitor.

Science Exhibits contains samples of research project reports of the Science Talent Searches conducted annually by Science Service for the Westinghouse Science Scholarships. These reports have been selected by the Science Talent Search judging staff as outstanding for subject and method of presentation.

Science Exhibits describes the Science Youth Movement carried on by Science Clubs of America. This organization, comprising more than 15,000 Science Clubs in the high schools of the country, encourages young people's enjoyment of doing science projects, culminating in the local and National Science Fairs. This organization is doing something to ameliorate the shortage of young scientists, technicians and engineers.

CIBA FOUNDATION SYMPOSIUM ON HYPERTENSION. Humoral and Neurogenic Factors. G. W. Pickering, Symposium Chairman. 294 pages, 73 illustrations. Price, $6.75. Boston: Little, Brown and Company, 1954.

The intellectually earnest practitioner has his work laid out for him in this deceptively small volume. While the concepts of Page, Grollman and Perera of this country might be familiar from numerous other publications, the evening scientist now must contend with the conflicting theories of McMichael and Pickering of England and the unfamiliar notions of Etienne-Martin of Dijon and Genest of Montreal. It is no longer adequate to speak of renin and angiotonin as possible humoral factors in the cause of hypertension; to sound really informed, other vasoconstrictor systems such as cerebrotonin and serotonin must be injected into the hypertension conversation.

There are items of interest to the practical man. For example, after Goldenberg's paper on the usefulness of the chemical analysis of urine in the diagnosis of pheochromocytoma, the discussion brings out the great experience of several workers with false-positive and false-negative results in the pharmacological methods now commonly used in detecting these tumors. This discussion material is illuminating but scarcely intelligible without a full and fresh acquaintanceship with these common testing methods. It is very definitely at a postgraduate level.

That arteriolar retinitis and renal failure are not necessarily associated with abnormal sphygmomanometer measurements has led Doctor Perera to emphasize the probable existence of a diffuse arteriolar disease without the usual hypertensive readings. In pregnancy toxemia, this paradox certainly occurs. That it occurs also in common hypertensive disease is important both as a caution in prognosis and because it will further the reluctance to label as "hypertensive" all individuals showing higher than average blood pressure readings. The day may come when diagnostic thoroughness will

(Continued on Page l)

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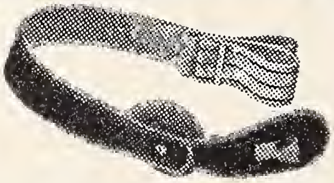

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(Continued from Page xlvii)

demand demonstrating diffuse arteriolar disease perhaps in the retina or elsewhere by means of a biopsy before the diagnosis of essential hypertension is made.

In therapy, too, the fact that the lowering of the blood pressure by hexamethonium or by other means does not always result in the subsidence of papilledema in malignant hypertension was generally agreed upon. In other words, blood pressure elevations are only one factor in the hypertensive process. The term "diffuse arteriolar disease" in preference to "essential arterial hypertension" seems increasingly justified by recent evidence. The fallacy of claiming "significant lowering of the blood pressure" as a result of this or that treatment can now be treated with well-reasoned skepticism based on the concepts alluded to above.

A new entity, "hexamethonium lung," a chronic organizing fibrinous edema recognizable radiologically, pathologically and perhaps clinically is briefly mentioned in this symposium.

William Bradley Martin, M.D.

HYGIENIC GUIDES AVAILABLE

The *American Industrial Hygiene Association Quarterly* announces the publication of a series of information bulletins, called Hygienic Guides, which will be made available to industry and other interested persons. This series is a development of AIHA through its Hygienic Guides Committee.

The purpose of the Hygienic Guides is to present authoritative and substantiated information on the toxicity of common industrial materials. It is also the purpose of these Guides to bring together all of the pertinent information required for hazard evaluation. It is planned that Guides will be prepared covering all of the common industrial chemicals and materials. Also, it is hoped that Guides can be prepared for the new chemicals introduced to the industrial markets.

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5. Engineering control procedures.
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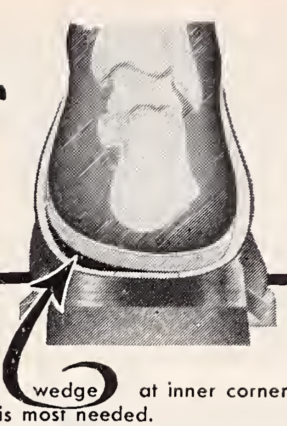
Guides are now available for acetaldehyde, cadmium, carbon tetrachloride, butyl alcohol (N-butanol) and amyl acetate.

Hygienic Guide sheets may be obtained from the American Industrial Hygiene Association, 14125 Prevost, Detroit 27, Michigan, at 25 cents each. All orders for less than $2.00 must be prepaid.

Members of the Hygienic Guides Committee are: William E. McCormick, Chairman; Francis F. Heyroth, M.D., Hervy B. Elkins, Ph.D., David W. Fassett, M.D., William B. Harris, Melvin W. First, Sc.D., H. E. Stokinger, Ph.D., H. E. Seifert, and Clyde M. Berry, Ph.D.



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SURGERY—Surgical Technic, two weeks, November 28, January 23
Surgical Anatomy and Clinical Surgery, two weeks, March 5
Surgery of Colon and Rectum, one week, November 28, February 27
General Surgery, one week, February 13; two weeks, April 23
Basic Principles in General Surgery, two weeks, April 9
Gallbladder Surgery, ten hours, April 9
Fractures and Traumatic Surgery, two weeks, March 12

GYNECOLOGY—Office and Operative Gynecology, two weeks, November 28, February 13
Vaginal Approach to Pelvic Surgery, one week, December 12, February 6

OBSTETRICS—General and Surgical Obstetrics, two weeks, February 27

MEDICINE—Internal Medicine, two weeks, May 7
Electrocardiography and Heart Disease, two-week basic course, March 12
Gastroscopy, forty-hour basic course, March 19
Dermatology, two weeks, May 7

RADIOLOGY—Diagnostic X-ray, two weeks, January 9
Clinical Use of Radioactive Iodine, one week, April 2
Clinical Uses of Radioisotopes, two weeks, May 7

PEDIATRICS—Intensive Review Course, two weeks, April 9

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OUTLOOK LESS GRIM FOR CHILDHOOD CANCER

"I do not believe that there is any doubt that the outlook for cancer in children is less grim than it was twenty years ago and that radiation therapy has contributed to this improvement," stated Ralph F. Phillips, M.D., of the Memorial Center, New York City, in an article published in the October, 1955 issue of *The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

However, Dr. Phillips lifted this warning finger in his report:

"It is clear that much more extended observations are required before the place of radiotherapy in pediatrics can be determined, not only as to its actual value in the treatment of childhood cancers, but even more as to its late effects on growth and development."

The study of late radiation effects, he continued, ". . . introduces another aspect of radiosensitivity, for the absence of any immediate reaction after irradiation does not preclude later manifestations of radiation damage. A big difference between pediatric and adult practice is the much longer life expectancy of successfully treated children, during which unexpected late sequelae of irradiation may occur. In the treatment of cancer such risks must be taken, but they can hardly be justified in nonmalignant conditions."

Some other conclusions advanced by Dr. Phillips:

. . . Malignant disease in children, although uncommon, ranks third among the causes of death.

. . . The radiosensitivity of normal and malignant tissues is not directly related to the age of the patient.

. . . Although the value of radiotherapy is uncertain, it has an essential part in the present management of childhood cancers, together with supportive measures, surgery and chemotherapy.

. . . The late effects of radiotherapy on growth and development are at present not thoroughly understood.

LUNG CANCER, A "GREAT SIMULATOR"

"While the detection of an abnormal process in the lungs can be accomplished with relative ease and accuracy, the identification of the nature of the disease presents many more difficulties. This is especially true of carcinoma of the lung, for it represents the great simulator of all other types of disease of the lungs," said Leo G. Rigler, M.D., of the University of Minnesota School of Medicine, in an article published in the September, 1955 issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

Dr. Rigler admitted that there are many methods whereby a definitive diagnosis of carcinoma of the lung can be established. Yet, he admitted, there are still a substantial number of patients with an abnormal process in the lung, in whom none of these methods is effective. In some instances, it is because of inability or reluctance to apply such methods, he continued.

For that reason, Dr. Rigler has investigated over a number of years the place of roentgen examination in the detection of this insidious lung disease.

Dr. Rigler admitted there are distinct limitations in the x-ray determination of the nature of the disease.

"But there are distinctive roentgenologic signs which seem so characteristic of malignant tumors that they carry with them almost the same weight as the more positive finding of actual cells, whether in the biopsy specimen or in the sputum," Dr. Rigler continued.

"Intensive roentgen study including comparison with previous films when available, examination in many positions and in different phases of respiration . . . will produce signs which are highly probable if not completely definitive for the diagnosis of tumor," the diagnostic specialist went on.

Some diagnostic signs suggested by Dr. Rigler for helping in the pinpointing of lung cancer included:

. . . The appearance of a solitary pulmonary shadow not present in previous films, especially in an individual past middle age, is of very great significance.

. . . The demonstration of a peripheral pulmonary mass or nodule in the lung which shows an increase in size when compared to a previous film is likewise of very great importance.

. . . In any solitary peripheral pulmonary nodule. the possibilities of diagnosis are always very difficult—or greater importance is the demonstration of the presence or absence of calcium in the mass.

. . . One sign which appears, so far at least, to be characteristic of malignancy consists of a notching or umbilication in the margin of the shadow. The deformity is not well seen in the

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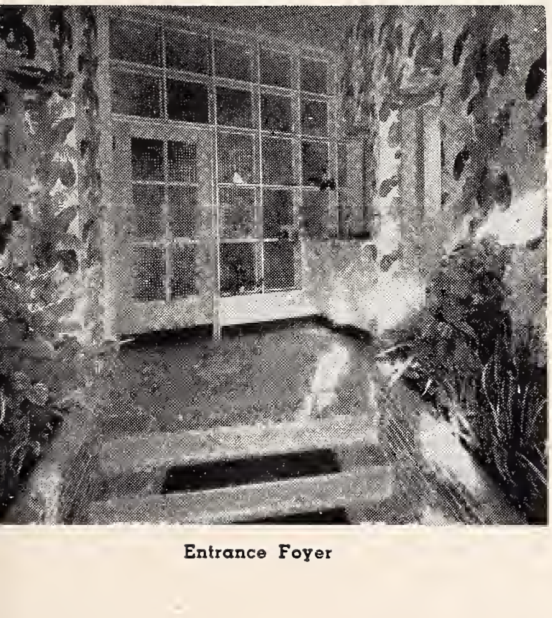

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ordinary roentgenograms, although occasionally we have seen it here as well, but it is quite clearly delineated in the body section roentgenograms when the film is made at the proper level. "In the absence of histopathologic evidence, we should utilize the roentgen examination to the fullest extent of its potential to make a definitive diagnosis before operation. This is absolutely necessary, not only to institute surgery at a time when it will be effective, but also to avoid indiscriminate surgery, especially in symptomless individuals," Dr. Rigler concluded.

CANCER OF LIP AND SKIN

Of 140 patients treated with radium for cancer of the lip and skin, 139 have remained free of the disease from one to five years, and the single re-occurrence was treated by surgery.

This report on radium therapy was made in the September, 1955, issue of *The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine* by B.L.Feurstein, M.D., Bayshore, N.Y.

"This very higher percentage of cures can be explained . . . Patients are appearing with lesions at a much earlier stage, due to the public awareness of cancer . . . and are prone to seek medical care early," Dr. Feuerstein pointed out.

"There are types of cases, in which one of two methods (x-ray or radium) is preferable and can be expected to produce a superior end result. Such cases may be therapeutic problems if only roentgen radiation, or radium, is available; if both modalities are available, the problem case may become a simple one," he added.

Some of his other conclusions:

. . . Most cases of cancer of the skin can be cured by roentgen rays or radium. However, treatment can often be simplified and better cosmetic results obtained in certain types of lesions if one is adept at both methods.

. . . Radium therapy usually gives a better cosmetic result and is preferred where high depth dose is to be avoided and in small early lesions.

. . . Roentgen therapy is preferred in less accessible areas, such as near the eye and in large bulky lesions.

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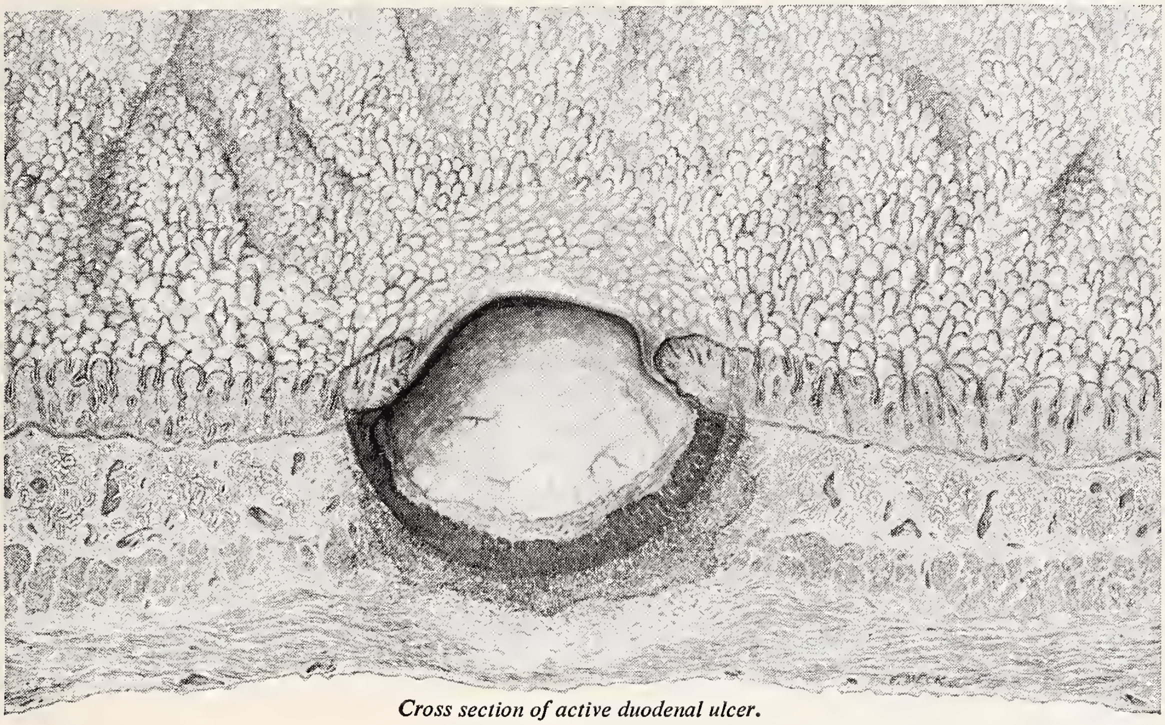

Cross section of active duodenal ulcer.

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Pro-Banthine is proving equally effective in the relief of hypermotility of the large and small bowel, certain forms of pylorospasm, pancreatitis and ureteral and bladder spasm. G. D. Searle & Co., Research in the Service of Medicine.

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1. Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.

2. Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthīne, Gastroenterology *25*:416 (Nov.) 1953.

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GENERAL INTEREST

(Continued from Page 954)

Pueblo, Colorado, first vice president, and **Dr. Jam F. Weir**, Rochester, second vice president. **Dr. Do ald C. Balfour** and **Dr. George B. Logan,** both Rochester, were re-named honorary president an secretary-treasurer, respectively.

NEW LOCATIONS

Dr. A. M. Scheidel, who recently completed three-year fellowship in internal medicine at th Mayo Clinic, has become associated in practice wit Dr. B. R. Geurs and Dr. R. H. Conley in Mankat

Dr. Robert L. Radke, formerly with the Yankto Indian Hospital, Wagner, South Dakota, is no affiliated with the Cass Lake Indian Hospital, Cas Lake.

Dr. E. E. Ulrich, formerly of Carey, Ohio, ha become associated in practice with Dr. C. G. Wingquist and Dr. D. R. Nichols at Crosby.

HOSPITAL NEWS

Election of officers highlighted the annual meeting of the medical staff of the Waconia Community Hospital, Waconia, October 18. Named a chief-of-staff was **Dr. K. B. Romness,** Mound. Othe officers elected include **Dr. R. S. Johnson,** Excelsio vice chief-of-staff, and **Dr. Iwan Sawaryniuk,** Waconia, secretary.

MINNESOTA BLUE SHIELD-BLUE CROSS

On March 1, 1950, Blue Shield had 292,455 participar subscribers, while on September 30, 1955, 713,515 persons were enrolled in Blue Shield. During the mont of February, 1950, 3,322 Blue Shield claims were pai whereas, during September, 1955, 12,231 claims wer paid. These figures show that there has been mor than a two-fold increase in the number of subscriber and more than a three-fold increase in the number o claims paid which constitutes the work-load.

During this same period, the number of Blue Shiel employes has increased from 21 to 34. Most importan of these increases is that involving claims auditors o examiners who have increased in number from thre to eight. This increase in the number of claims examiners has been disproportionately small as compare with the increased number of claims. Two of the additional claims examiners are registered nurses increasing to three the number of nurses now processin claims.

In 1953 a professional relations program was initiated Currently three members of this staff are engaged i calling upon doctors and their office personnel throughout the state. Besides keeping the medical professio informed regarding Blue Shield, the professional relations staff assists doctors' secretaries, nurses and receptionists in handling Blue Shield claims.

Along with the increases in activities, work, and per-nnel have come improvements in efficiency directed ward prompt payment of claims, more rapid handling correspondence, more accurate telephone information d wider dissemination of information about Blue ield in the medical profession.

According to figures just released from the statistical partment of the Minnesota Hospital Service Associa-n (Blue Cross), patients are staying in the hospital r slightly longer periods in 1954 than during 1955.

During the first nine months of 1955, 766,037.8 days hospital care were provided for 124,949 Blue Cross bscribers compared to 751,778.2 days for 125,602 bscribers during the same period of the previous year.

Though there was a decrease of 653 cases in the st nine months of 1955 over the same period in 1954, ys of patient care increased to 14,259.6.

The average length of hospital stay increased from) days in the first nine months of 1954, to 6.1 days ıring the same period of 1955.

Pregnancy care represented 17.1 per cent or 21,338 ses of all hospital care during the first nine months 1955, compared to 21,166 cases or 16.9 per cent of re during the same period of 1954.

Accident cases accounted for the second largest seg-ent of hospital cases. There were 19,574 accident ses representing 15.7 per cent of total cases paid during '55 compared to 19,356 cases or 15.4 per cent of total ses paid during the same period of 1954.

Ranked as the fourth largest segment in 1954, diges-ve illnesses rose to third in 1955. There were 17,277 ıch cases during the first nine months of 1955, account-g for 13.8 per cent of all cases as compared to 16,819 ses or 13.4 per cent of the total for the same period 1954.

Respiratory illnesses dropped from third place in 1954 fourth place in 1955. There were 13,977 cases or 11.2 r cent of the total for the first nine months of 1955, compared to 17,256 cases or 13.7 per cent in 1954.

Surgical, tonsillectomies and adenoidectomies seem to ıve accounted for the decrease. During the first nine onths of 1955, there were 4,774 tonsillectomies, as mpared to 7,344 such operations during the same riod of 1954.

In comparing benefits paid, digestive illnesses seem be the most costly. Hospital benefits for such care ıring the first nine months of 1955 exceeded $2,600,000. regnancy benefits amounted to $2,300,000. Diseases of e circulatory system ranked third in benefits paid, nounting to approximately $1,800,000 for the same ne-month period.

During the first nine months of 1955, Blue Cross ses and days in relation to number of effective con-acts show a decrease of eight cases per year per 000 contracts, and an increase of 15 days per year er contract. This fact is reflected by the 0.1 day crease in length of stay compared to the same nine-onth period of 1954.

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Bumbalo, T. S., Gustina, F. J., and Oleksiak, R. E.: J. Pediat. 44:386, 1954.

White, R. H. R., and Standen, O. D.: Brit. M. J. 2:755, 1953.

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Brown, H. W.: J. Pediat. 45:419, 1954.

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Book Reviews

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SYNOPSIS OF PATHOLOGY. By W. A. D. An son. 3rd ed. 788 pages Illus. Price. $8.00. St. Lo C. V. Mosby Co., 1952.

This book is truly a condensed pathology book. fills a need for a rapid review of the important po of pathology. This form would be suitable for tl physicians who have set up a comprehensive reac program to review the fields of basic medical scien

Donald W. Koza,

SEVENTY-FIVE YEARS OF MEDICAL PR RESS, 1878-1953. Edited and with a foreword Louis H. Bauer, M.D., F.A.C.P., Secretary-Genera the World Medical Association, Past President the American Medical Association. 286 pages. I Price, $4.00. Philadelphia: Lea & Febiger, 1954.

The First Western Hemisphere Conference of World Medical Association was held in Richmond, in April, 1953. "Seventy-five Years of Medical Progr was the theme of the conference. This book is a c pilation of the papers presented by men who are am the outstanding in their specialties.

A period of seventy-five years was chosen becaus was felt that during these years more has been lea about disease and its treatment than during the prev three thousand years. That is a startling claim, bu you read these papers you can begin to see the ju cation for such a claim.

The nineteen specialties chosen were the specia as defined by the Advisory Board for Medical Spe ties. In addition, general practice was also represe Among the contributors, to name but about a fourt them, are M. B. Sulzberger, N. J. Eastman, U. R. ner, J. S. Simmons, A. Blalock, and M. E. DeBake

It is exciting to read of the rapid advances mad each field. Besides the great achievements of this being listed, broad trends are sometimes set forth; standing pioneers are given recognition; specific pa or books, which have opened new vistas are occasio cited. Each contributor, or team of contributors, has sented his specialty in the way he feels best depict progress. The interdependence of the various specia is made clear to the reader as he sees how a discover one field opens new avenues of progress in another

The book is a good summary of medical prog since 1878.

M.

MEDICAL SUPPORT OF THE ARMY AIR FO IN WORLD WAR II. By Mae Mills Link, P and Hubert A. Coleman, Ph.D., with a forewor Maj. Gen. Dan C. Ogle, Surgeon General, U States Air Force. Price, $7.00. Washington 25, D. Government Printing Office, 1955.

This book, which required over ten years to com write, edit and print, tells how military medicine elled into a new dimension as it became airborne. ontains valuable reading for military and civilian ners concerned with manpower, and every doctor gain new insight into how the talents of the medical ession are forged into the iron framework of comd to meet a national emergency. At the same time, volume is interesting general reading because the ian element is never lost.

ach chapter is thoroughly documented, with footnotes he end of every chapter for easy reference. The me is well illustrated with charts, diagrams, and ographs and includes extensive tabular data. There detailed index.

NUSCRIPTS COLLECTIONS OF THE MINESOTA HISTORICAL SOCIETY. Compiled by ucile M. Kane and Kathryn A. Johnson. 212 pages. rice, $3.60. Saint Paul: Minnesota Historical ociety, 1955.

second guide to the Minnesota Historical Society's ection of four million manuscripts has just been ed. *Manuscripts Collections of the Minnesota Hiscal Society,* compiled by Lucile M. Kane and hryn A. Johnson, is a supplement to *Guide to the sonal Papers of the Minnesota Historical Society,* npiled by Grace Lee Nute and Gertrude Ackermann issued in 1935. Together, these guides describe 5 groups of papers collected by the Society since establishment in 1849.

Iost of the manuscripts deal with Minnesota and h its many relationships with the other states in Union. Several groups of papers that document the tward movement of a family from the east coast he west are good examples of the national character this "regional collection."

thirty-seven-page index is the key that unlocks the lth of material found in the volume.

VISED SOUND FILM AVAILABLE

completely revised, sound version of the film, "Depment of the Gastro-Intestinal Tract," has just been ased by The Center for Mass Communication of umbia University Press. The 35-minute film, in or, was produced by Joseph J. McDonald, M.D., Prosor of Clinical Surgery at the College of Physicians, umbia University, and at present Dean of the Medical ulty at the American University of Beirut, Lebanon.

he film depicts not only the embryology of the tract also associated features such as the implantation of ovum, the development of the placenta and early ulatory system as well as other structures closely ated to the gastro-intestinal tract such as the body ities, the main vessels and the diaphragm. It will be ful as an adjunct to the teaching of gross anatomy well as to abdominal surgery and is most appropriate showing to students of embryology and anatomy on h the graduate and undergraduate level.

rints in 16mm for use by medical schools are availe either for rental or purchase exclusively from The nter for Mass Communication, of Columbia Univery Press, New York 25, New York.

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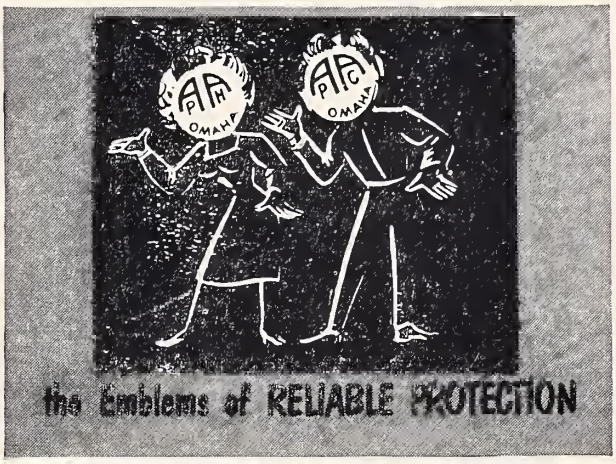
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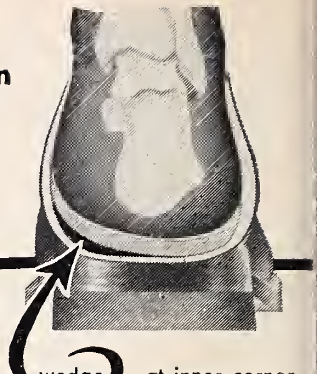

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CORPS EXAMINATIONS FOR MEDICAL OFFICERS USPHS

A competitive examination for appointment of Medical Officers to the Regular Corps of the United States Public Health Service will be held in various places throughout the country on March 20, 21, and 22, 1956.

Appointments provide opportunities for career service in clinical medicine, research, and public health. They will be made in the ranks of Assistant and Senior Assistant, equivalent to Navy ranks of Lieutenant (j.g.) and Lieutenant.

Entrance pay for an Assistant Surgeon with dependents is $6,017 per year; for Senior Assistant Surgeon with dependents, $6,918. Qualified officers are promoted at regular intervals.

Benefits other than promotions include periodic pay increases, 30 days' annual leave, sick leave, medical care, disability retirement pay, regular retirement pay which is three-fourths of annual basic pay at time of retirement, and other privileges.

Active duty as a Public Health Service officer fulfills the obligations of Selective Service.

Requirements for both ranks are U. S. citizenship, age of at least twenty-one years, and graduation from a recognized school of medicine. For the rank of Assistant Surgeon, at least seven years of collegiate and professional training and appropriate experience are needed. For Senior Assistant Surgeon, an additional three years, for a total of at least ten years of collegiate and professional training and appropriate experience, are required.

Entrance examinations will include an oral interview, physical examination, and comprehensive objective examinations in the professional field.

Application forms may be obtained from the Chief, Division of Personnel, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D. C. Completed application forms must be received in the Division of Personnel *no later than February 10, 1956.*

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NORTHWESTERN UNIVERSITY REPORTS ON NEW GERMICIDE

Inflammation of the bladder apparently can be treate successfully with a new germicide, it is reported in th *Quarterly Bulletin of Northwestern University Medic School.*

The germicide, chlorpactin WCS-90, a derivative chlorine, gave "dramatic improvement" to patients wi painful cystitis, said Dr. Vincent J. O'Conor, chairma and professor of urology at the school.

The recently developed germicide solution was use in forty-eight patients who for many years had inflam mation of the bladder and painful, frequent urinatio which could not be eradicated by drugs. With chlor pactin, "spectacular improvement was achieved" nearly all the patients, said Dr. O'Conor.

The exact cause of the bladder inflammation, calle chronic interstitial cystitis, is not known. The capaci of the bladder gets smaller and smaller, so urinatio is frequent and painful. The condition is 30 time more prevalent in women than in men.

Dr. O'Conor wrote that "one hesitates to recommen a new treatment for interstitial cystitis. This intractabl condition has been a bugbear to urologists ever sinc Dr. Hunner of Baltimore described it in 1915. Abou every sort of treatment, both local and general, fo increasing the capacity of the bladder in cases of elusiv ulcer with interstitial cystitis has been used."

But he continued that "this treatment was applied individuals who had been under our care for man years and who had never had a satisfactory improvemen of anything but a very temporary nature. The result in the limited series of these very chronic and intrac table cases have been so spectacular that it seems further trial is warranted by the profession at large."

The new germicide also has been reported to b successful in preliminary trials for treating tuberculos of the bladder and diseases of the mouth, for sterilizin instruments, and as an antiseptic for surgery. Whe the powder form is added to cool or lukewarm wate it results in liberation of hydrochlorous acid.

"It is said," wrote Dr. O'Conor, "that even the con centrated powder is not harmful to normal tissues."

In the preliminary trials with chlorpactin, a wea solution of the germicide was used to stretch th bladder. Usually after the first treatment, the cond tion became worse, he said, but the temporary distre soon disappeared with subsequent "great and rapid im provement." The treatments were continued one or tw times weekly for five treatments, then decreased once a month or longer.

The trials with chlorpactin have only lasted seve months, but, wrote Dr. O'Conor, "the improvemen obtained in these patients has been so dramatic tha it prompts us to make this preliminary report in th hope of encouraging others to try a similar method o treatment."

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Symposium on Parenteral Fluids, Nutrition and Electrolytes

March 5-6, 1954

Radisson Hotel

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Section II, Supplement to Minnesota Medicine,
March, 1955, Volume 38, Number 3

Symposium on Parenteral Fluids, Nutrition, and Electrolytes

March 5-6, 1954
Radisson Hotel
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This symposium, sponsored by the Hennepin County Medical Society and the Academy of General Practitioners, was suggested by Dr. Robert Herwick, medical director of the Baxter Laboratories, to Dr. Carl O. Rice who was responsible for its organization.

The symposium was made possible through the generosity of the Baxter Laboratories, Cutter Laboratories, Lilly Laboratories, Wyeth Laboratories, and the Ulmer Laboratories.

The Milliequivalent as a Unit of Measure in the Interpretation and Correction of Electrolyte Disturbances

O. M. HELMER, PH.D.
Indianapolis, Indiana

A FEW YEARS ago after almost every medical lecture, there was at least one person in the audience who would rise and ask the speaker what the pH of the solution was. As he settled himself comfortably in his chair, one could observe a satisfied expression on his face which indicated that he felt he had impressed both the speaker and the audience with his erudition. Now pH is commonly used and has been accepted as the most convenient and simplest way to express hydrogen ion concentration in biological systems.

Although the term "milliequivalent" probably could also be used nowadays to impress the uninitiated, its chief value lies in the fact that the application of chemical equivalence to the measurement of electrolytes simplifies the diagnosis and treatment of disturbance in electrolyte and fluid balance.

Before defining the term "milliequivalent," it would probably be best first to explain the meaning of chemical equivalence. If the weight in grams of NaOH, KOH, and HCI corresponding to the molecular weight of these substances has been dissolved in enough water to make one liter, each solution will contain, respectively, 40 gm. of NaOH, 56 gm. of KOH, and 37 gm. of HCl (*i.e.*, molecular weight of hydrogen = 1, chloride = 36, and HCl = 37). These are called molar solutions, and they contain one mole of the substance per liter. "When equal volumes are mixed (for example, 1 cc. of the alkaline NaOH solution and 1 cc. of the acid HCl solution), they neutralize each other. The same result is obtained when 1 cc. of alkaline KOH solution is mixed with 1 cc. of the HCl solution. The two alkaline solutions show the same combining power or the same activity for equal volumes. Each contains the same number of active particles per unit volume, but the weights of the substances per unit volume are not the same. Since one mole of NaOH exhibits the same combining power as one mole of KOH, they are equivalent, mole for mole. Hence, one equivalent of any of the three substances is one mole of the substance. It is evident that the comparison of two alkalies in terms of their equivalence brings us much closer to what we want to know about their presence in living systems than does the comparison in terms of weight."[1]

Substances react also on the basis of their valence. The chemicals mentioned thus far are all univalent. But calcium (atomic weight = 40) is bivalent; that is, one mole of Ca, which is 40 gm., possesses twice the combining power of one mole of Na. Hence, one mole of Ca is two equivalents of Ca, and one equivalent of Ca weighs 20 gm.

Since the concentration of electrolytes in the blood is relatively low, it is easier to express the values in whole numbers by using as a unit of measure 1/1,000 of an equivalent, or milliequivalent.

The average concentration of each electrolyte expressed in terms of milliequivalents is shown on left side of Table I.

Under normal conditions the extracellular fluid (the plasma plus the fluid that surrounds the cells — the interstitial fluid) contains approximately 155 milliequivalents per liter of cations (base ions which have a positive electrical charge) and an equal number of anions (acid ions which bear a negative charge).

All of the components except protein are readily diffusible into the interstitial fluid, so that, with the exception of protein, the concentration of these

---

Dr. Helmer is head of the Department of Physiological Chemistry Lilly Laboratory for Clinical Research, Indianapolis General Hospital and associate professor of experimental medicine at Indiana University School of Medicine, Indianapolis, Indiana.

TABLE I.

ELECTROLYTE COMPOSITION OF BLOOD PLASMA

(Cations) + mEq./liter		(Anions) − mEq./liter		(Cations) + mg./100 cc.		(Anions) −	
		HCO_3	27			HCO_3	60 vols. %
		Cl	103			Cl	365 mg. %
Na	142	HPO_4	2	Na	327	HPO_4	3.5 mg. %
K	5	SO_4	1	K	20	SO_4	1.5 mg. %
Ca	5	Org. Acids	6	Ca	10	Org. Acids	
Mg	3	Protein	16	Mg	3.6	Protein	7 gm./100 cc.
	155		155				

$HCO_3 + Cl = 27 + 103 = 130$
Na = 142 therefore 142 − 130 = 12
Therefore $HCO_3 + Cl + 12 =$ Na concentration

ions in the fluid surrounding the cells is essentially the same as that in the plasma. This table also illustrates the advantages of expressing the concentration of all components in the same unit, so that the total concentration of cations and anions can be obtained. This is the main advantage of the system based on chemical equivalence. The total concentration of cations always equals that of the anions in all fluids.

On the right side of Table I is shown the values of electrolytes expressed in the terms used before the introduction of milliequivalents. Note that 3 different units are used: mg. per cent, volumes per cent, and grams per 100 ml. Chloride is sometimes even expressed as mg. of NaCl per ml. of plasma. It is difficult to compare the biological relationships of the different components of plasma under these conditions.

At the bottom of Table I is shown the relationship of the carbon dioxide content and chloride concentration to the sodium concentration of the plasma when expressed in milliequivalents per liter. The sum of values for bicarbonate and chloride ions normally is about 130 mEq. The difference between this value and the concentration of sodium is 12 mEq./liter. Consequently in the absence of an abnormal accumulation of other anions, the sum of the bicarbonate and chloride ion plus 12 is an approximation of the sodium concentration.

When laboratory facilities are not available for sodium and potassium analysis, this calculation is often a valuable means of estimation of the sodium concentration of plasma.

The application of this calculation in estimating the sodium concentration in the plasma of surgical patients in electrolyte imbalance is shown in following examples. On the left is shown the actual analysis. On the right the estimated sodium concentration.

Example 1

Determined Values	Calculation
Na^+ = 121 mEq./L.	20.8 + 84.3 = 105
K^+ = 4.5 mEq./L.	105 + 12 = 117 mEq. of Na.
Cl^- = 84.3 mEq./L.	
HCO_3^- = 20.8 mEq./L.	

Example 2

Determined Values	Calculation
Na^+ = 123 mEq./L.	27.2 + 84.1 = 111.3
K^+ = 2.8 mEq./L.	111 + 12 = 123 mEq. of Na.
HCO_3^- = 27.2 mEq./L.	
Cl^- = 84.1 mEq./L.	

In the second example, the normal CO_2 content with the low chloride value yields additional information. As Darrow pointed out, a high CO_2 content compared to the chloride content is often a sign of intracellular potassium deficiency. In this plasma, a low K value was found by analysis with a flame photometer.

Conversion of Milligrams Per Cent to Milliequivalents Per Liter

Values expressed as milligrams per 100 ml. can be converted to milliequivalents per liter by using the following formula:

$$\frac{\text{mg. per 100 ml.} \times 10}{\text{atomic weight}} \times \text{valence} = \text{mEq./L.}$$

$$\text{Example: 360 mg./100 ml. chloride} = \frac{360 \times 10}{35} \times 1$$

$$= 103 \text{ mEq./L.}$$

In the case of gases (e.g., carbon dioxide), advantage is taken of the fact that a quantity of gas equal in weight to its molecular weight in grams occupies 22.4 liters under standard conditions. The formula used is:

$$\text{mEq./L.} = \frac{\text{vol. \% } \times 10}{22.4}$$

$$\text{Example: } CO_2 \text{ of 60.5 vol. \%} = \frac{60.5 \times 10}{22.4} = 27 \text{ mEq./L.}$$

Factors which simplify these conversions are given in Table II.

The usefulness of milliequivalents in planning the treatment of electrolyte and fluid imbalance is illustrated by the following example:

Calculation of Theoretical Electrolyte Deficit

Example: A patient weighing 132 pounds is found to have a concentration of 128 mEq./L. of sodium in the plasma. How much sodium chloride

TABLE II. DATA FOR CONVERSION OF SERUM (OR PLASMA) ELECTROLYTES TO mEq./LITER

Electrolyte	Calculated As:	Valence	Equiv. Weight	Conversion Factor	
				Multiply By	Divide By
Sodium	Sodium	1	23	0.434	2.3
Potassium	Potassium	1	39	0.256	3.9
Calcium	Total Ca	2	20	0.500	2
Magnesium	Magnesium	2	12	0.833	1.2
Bicarbonate	CO_2 capacity	—	22.4*	0.446	2.2
Chloride	Chloride	1	35.5	0.281	3.5
	NaCl	1	58.5	0.170	5.8
Phosphate inorganic	Phosphorus	1.8	17.2	0.580	1.7
Sulfate inorganic	Sulfur	2	16	0.625	1.6
Protein	Protein	—	—	2.43	

*22.4 liters per mole.

TABLE III. ELECTROLYTE CONCENTRATIONS OF SEVERAL COMMONLY USED PARENTERAL FLUIDS

Solution	Gm./L.	mEq./L. Cations	mEq./L. Anions
Isotonic saline (0.9% NaCl)	9.0	154 Na	154 Cl
Hypotonic saline (0.45% NaCl)	4.5	77 Na	77 Cl
Hypertonic saline (5.0% NaCl)	50.0	850 Na	850 Cl
Ringer's Solution (Isotonic Solution of Three Chlorides)	8.6 NaCl 0.3 KCl 0.33 $CaCl_2$	147 Na 4 K 6 Ca	157 Cl
One-Sixth Molar Lactate (molar solution diluted to 1/6)	18.66 Na Lact.	166 Na	166 mM. lactate
Sodium bicarbonate (1.5%)	15.0 $NaHCO_3$	178 Na	178 mM. bicarbonate
Ringer's Lactate	6.0 NaCl 0.3 KCl 0.2 $CaCl_2$ 3.1 Na Lact.	130 Na 4 K 4 Ca	111 Cl 27 mM. lactate

is needed to replace the deficit? The amount of sodium chloride needed to replenish the extracellular deficit of sodium can be estimated as follows: To convert pounds to kilograms, divide the patient's weight by 2.2. Therefore $\frac{132}{2.2} =$ 60 Kg.

The average normal concentration of sodium (Table I) is 142 mEq./L. The patient's plasma level for this ion is 128 mEq./L. Substituting in the following formula:

Amount of ion needed (in mEq.) = volume of extracellular fluid (1/5 body weight) X (normal value of ion mEq./L. - patient's value mEq./L.).

Example: $\frac{60}{5}$ X (142 - 128) = 168 mEq. of sodium needed.

It is seen from Table III that this calculated deficit of sodium is supplied by 1,090 milliliters of isotonic saline, 190 milliliters of 5 per cent sodium chloride, etc.

As many authors have pointed out, calculation of sodium deficit on the basis of 20 per cent of the body weight results in a quantity much lower than the actual needs for replacement, apparently because of replenishment of intracellular deficit. Some authors prefer to make such calculations on total body water, using 60 per cent of ideal body weight (in kilograms).

In addition to expressing plasma electrolyte concentrations in milliequivalents, it is also advantageous to express the concentration of repair solutions in the same unit. In chart III, for example, if one compares the concentration of the salts in Ringer's solution in terms of grams with milliequivalents per liter, it is obvious how much easier it is to relate the amount of individual ions in the repair solutions to the deficit in tissue electrolytes to be corrected.

It can be seen, then, that the milliequivalent as a unit of measure simplifies and facilitates the interpretation and correction of electrolyte disturbances.

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Relation of Alkalosis to Potassium Deficiency

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FOR THE past ten years, I have been studying the relation of alkalosis to loss of body potassium. The recent work which seems to clarify the pathogenesis of alkalosis has been carried out together with Robert E. Cooke and a group of students—Martin Vita, Saul Brusilow, Claude Reed and Donnel Etzwiler—who did various parts of the study as their theses. I shall not apologize for discussing more of the physiology of body fluids than the therapeutic practice. As you all know, therapy cannot be designed to meet individual problems without understanding the physiologic disturbance which one is trying to correct.

As long as it was thought that alkalosis did not involve changes in the composition of the cells and changes in renal function, it was rational to treat alkalosis with physiologic saline alone. We now know that alkalosis is frequently, if not invariably, accompanied by low K and high intracellular Na in the muscles. The work which I shall discuss shows certain changes in renal function which accompany K deficiency. These studies demonstrate that K as well as NaCl is necessary in the treatment of most cases of alkalosis.

Table I shows the data obtained on rats receiving a diet practically devoid of Na, K and Cl, together with drinking water having the contents of Na, Cl and K shown in the table. The rats received these solutions and the diet for fourteen to twenty-one days and were then killed. This procedure gave a large load of $NaHCO_3$ and variable amounts of K and Cl. Your attention is first directed to the serum concentrations and the muscle contents per 100 gm of fat free solids. It is notable that alkalosis did not develop except when muscle K became low and intracellular Na high. The first two groups show that alkalosis did not develop in response to the large load of $NaHCO_3$ as long as 5 mm. or more of KCL per liter was present in the drinking solution. The next two groups show that alkalosis developed when the solution contained 3 mm. or less of KCL per liter. However, alkalosis developed in Group fivewhen Cl was 1.5 mEq, and K, 5 mEq per L. Thus, 5 mEq of K did not prevent alkalosis when Cl intake was very low. In Group seven the characteristic changes of alkalosis developed when the load of $NaHCO_3$ and K and Cl were reduced in proportion. For this reason, one may conclude that minimum intakes of K and Cl are necessary to protect from alkalosis even with a reduced load of $NaHCO_3$. Group eight shows that a higher intake of K prevents alkalosis when the Cl intake is the same as in Group five. As a whole the experiments demonstrate that K and Cl are necessary to prevent alkalosis when a large load of $NaHCO_3$ is given.

TABLE I.

15 - 20 Day $NaHCO_3$ Loads

	Drinking Water			Serum		Muscle	
	HCO_3	K	Cl	HCO_3	Cl	K	Na_i
1)	150	10	10	24	101	-	-
2)	150	5	5	25	100	43	4.6
3)	150	3	5	34	87	35	10.5
4)	150	1.5	5	34	88	-	-
5)	150	5.0	1.5	30	88	41	9.7
6)	150	1.5	15	31	88	39	10.3
7)	30	1	1	30	95	39	7.2
8)	150	15	1.5	25	98	43	4.9

Concentrations in mEq per L for serum and drinking water, and per 100 gm fat-free solids for muscle. HCO_3 is presented as Na HCO_3. Na is calculated intracellular Na.

When the intake of K per Kg per day is related to serum bicarbonate concentration, it was found that alkalosis did not develop as long as 0.4 mEq of K per Kg per day was ingested. Chart I shows that when the intake of K was 0.4 mEq, alkalosis

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developed if the intake of Cl was less than 0.4 mEq per Kg per day.

The next group of experiments shows the change in serum and muscle composition of rats receiving a single intraperitoneal injection of 15 mEq of $NaHCO_3$ per Kg. The analyses were made twelve hours later. The results in normal rats and rats with K deficiency produced by injection of desoxycorticosterone acetate while fed a diet low in K are shown in Table II. The normal rats are shown in the top two rows, and the K deficient rats in the last two rows.

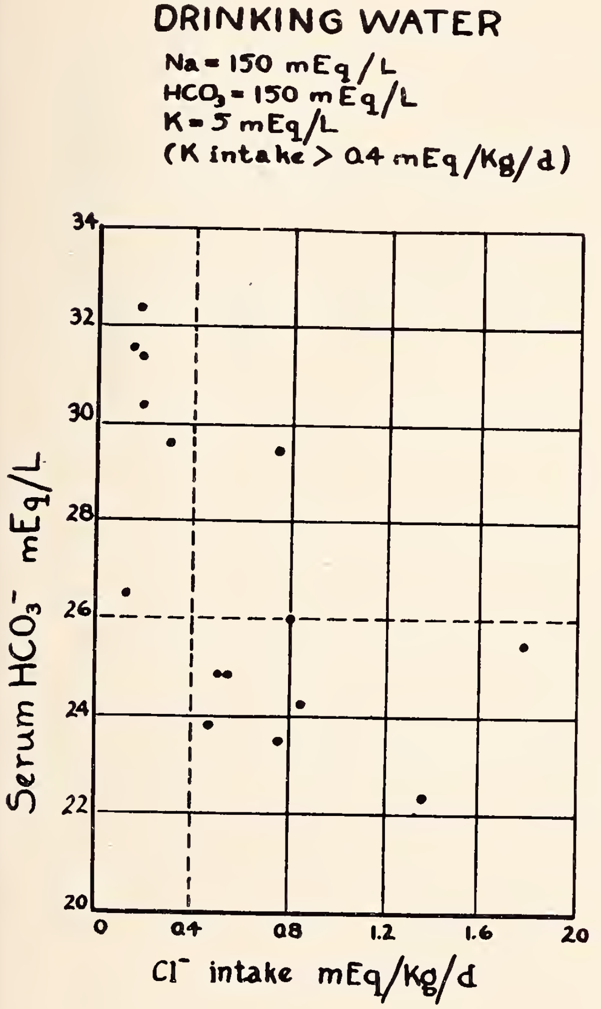

Chart I

Despite the large dose of $NaHCO_3$, the normal rats did not develop alkalosis of the serum but about 6 mEq of K were lost from the muscle and intracellular Na increased about 4 mEq. These values represent approximately the muscle changes in a Kg of rat. The normal rats excreted about 60 per cent of the $NaHCO_3$ and 90 per cent of the water. Cation excretion was about equivalent to dose of 15 mEq of $NaHCO_3$, but 9 mEq was Na and 6, K. The excretion of Na and the exchange of intracellular K for Na explains the failure to develop extracellular alkalosis. Practically no Cl was excreted by these rats.

TABLE II. SERUM CONCENTRATIONS IN MEQ PER L. MUSCLE PER 100 *gm.* FAT-FREE SOLIDS.

Serum and Muscle Composition
After 15 mM/Kg of $NaHCO_3$ I.P.

Group	Serum			Muscle		
	Cl	HCO_3	K	Cl	K	Na_i
Normal						
0 h.	100	25	4.5	5.5	46	3
12 h.	99	25	3.6	6.4	40	7.4
K Deficient						
0 h.	90	28	2.3	5.8	33	12.2
12 h.	78	42	2.4	5.2	33	12.4

TABLE III. SERUM CONCENTRATIONS MEQ PER L.

Average Serum Concentrations
Treated Animals Received 3 mM/Kg of $KHCO_3$ b.i.d.

Group	No.	Days	Na	K	Cl	HCO_3
Normal			143	5.0	100	24
K deficient	4	0	142	2.2	84	33
$KHCO_3$ 6 mM	4	1	145	3.1	91	34
$KHCO_3$ 12 mM	4	2	143	3.7	93	28
$KHCO_3$ 18 mM	4	3	143	4.0	99	27
$KHCO_3$ 24 mM	8	4	142	4.2	97	26
Completely Corrected Animals						
$KHCO_3$ 24 mM	5	4	142	4.5	101	23.5

The rats deficient in K excreted only about 40 per cent of the $NaHCO_3$; the serums showed marked alkalosis but the muscles did not undergo further changes. Appreciable amounts of Cl were excreted but practically no K. These experiments illustrate the two mechanisms of defense against alkalosis: first, the ability of the rats to excrete Na in excess of Cl which is impaired when K is not available and second, the exchange of intracellular K for extracellular Na which requires normal cellular composition to be effective.

In previous experiments, alkalosis with K deficiency was corrected by administering 6 mM of KCl per Kg per day. It was shown that the exchange of the administered KCl between extracellular and intracellular fluids of muscle corrected the alkalosis without intervention of the kidneys. In effect, 3 K exchanged for 2 Na in the muscles. This left extracellular fluids with 2 NaCl and one Cl and one H. The H and Cl reacted with $NaHCO_2$ to form one NaCl. Table III shows similar experiments in which 6 mM of $KHCO_3$ were injected each day into rats with alkalosis and K deficiency. The exchange in the muscle is essentially the same; 3 K exchanges for 2 Na. Extracellular fluids are left with 2 $NaHCO_3$ and one H and one HCO_3. Treatment with $KHCO_3$ cannot correct alkalosis without excretion of Na in excess of Cl.

TABLE IV. MUSCLE CONTENTS PER 100 GM. FAT-FREE SOLIDS.

Average Muscle Composition Per 100 Gms. Fat-Free Solids
Treated Animals Received 3 mM/Kg of $KHCO_3$ b. i. d.

Group	No.	H_2O	Cl	Na	K	Na_i
Normal		340	5.5	10.0	46	3
K deficient	4	323	5.0	19.5	28.8	15
$KHCO_3$ 6 mM/Kg	4	326	5.0	16.0	35.8	9.5
$KHCO_3$ 12 mM/Kg	4	326	5.0	13.5	38.0	7.8
$KHCO_3$ 18 mM/Kg	4	325	5.1	13.0	40.0	7.5
$KHCO_3$ 24 mM/Kg	8	317	5.0	11.8	41.3	6.5
Completely Corrected Animals						
$KHCO_3$ 24 mM/Kg	5	320	5.0	10	43.5	4.5

Tables III and IV show the gradual correction of extracellular alkalosis and muscle composition. In four days, five of eight rats showed essentially normal composition of both serums and muscle. A decrease of about 10 per cent in body weight indicated a loss of body water.

Chart II shows the balances based on urinary excretion and the injected $KHCO_3$. Almost all the administered K was retained during the first two days but practically all the injected K was excreted during the fourth day. Accompanying the retention of K, Na was excreted but the urines were practically free of Cl. Other data which will not be presented show that the excretion of an excess of cations over fixed anions was accomplished with little or no change in urinary pH since urinary organic acids were increased. In rats, at least, an excess of cations may be excreted by substitution of organic acids for Cl just as an excess of anions are excreted in acidosis without a great change in pH by the substitution of NH_4 for Na and K.

The balance data predict the changes in serum and muscle composition within the accuracy of the methods. Wallace and associates showed that total body Cl was unchanged in alkalosis with K deficiency despite low chloride in the serum. For this reason, extracellular fluids are apparently expanded in this type of alkalosis. Thus Cl retention is not necessary for recovery of Cl concentration in rats having alkalosis with K deficiency. The rise in serum concentration of Cl during administration of $KHCO_3$ is explained by the decrease in body water leading to a decrease in extracellular volume. The correction of this type of alkalosis, therefore, involves not only a control of extracellular concentrations but a regulation of extracellular volume.

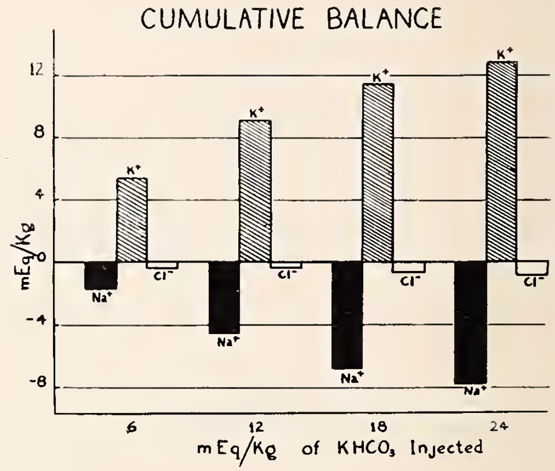

Chart II.

Renal function is usually described in terms of excretion. However, since about 180 liters of glomerular filtrate is formed each day in the average adult, about fourteen times the extracellular contents of water, Na and Cl pass through the glomeruli each day. Since only one to two liters of water and a very small fraction of the filtered Na and Cl are excreted in the urine each day, most of the glomerular filtrate is returned to the body. Consideration of the quantities involved shows that extracellular composition is preserved by reabsorbing Na and Cl from the glomerular filtrate at the concentrations that are maintained. This is the mechanism which preserves body composition. For this reason renal function is best described in terms of the overall composition of the fluid reabsorbed from the glomerular filtrate. If normal extracellular composition is maintained the concentration of the fluid reabsorbed must be approximately the same as that of a glomerular filtrate, namely, Na 140 and Cl 114 mEq per liter. If alkalosis is maintained the fluid reabsorbed must be low in Cl relative to Na and have the composition that the body tends to maintain.

TABLE V.

Glomerular Filtrate and Reabsorbate
Normal

	Concentrations		Amounts	
	Filtr.	Reabsorb.	Reabsorbed	Excreted
Cl mM	110	110	110	0
Na mM	140	140	140	0
H_2O gm	1000	1000	1000	0

The implications of these concepts are illustrated in Tables V, VI and VII. On the left, the concentration of the glomerular filtrate and of the fluid reabsorbed are shown. To the right the maximum possible reabsorption from a liter of glomerular filtrate is shown together with the consequent balance of water, Na and Cl.

Table V demonstrates that all the water, Na and Cl can theoretically be absorbed when normal composition is maintained by absorption at the same concentration as the filtrate. Table VI shows the change in the fluid reabsorbed when alkalosis develops and is maintained as a result of K deficiency. All the water and Na may theoretically be reabsorbed from a glomerular filtrate of normal composition if the fluid reabsorbed has the composition which will be maintained in alkalosis. However, some Cl must be excreted. In Table VII the recovery from alkalosis is shown. In this case all the Cl may be reabsorbed, but part of the water and Na must be excreted. The change in the fluid reabsorbed is probably gradual but the direction of the changes are correctly indicated by the tables. It will be seen that if a load of NaCl is given during K deficiency, the tendency to reabsorb Na and water in excess of Cl will tend to expand extracellular fluids. During recovery from alkalosis, the tendency to reabsorb only part of the water and Na will tend to contract extracellular fluids unless a high intake of NaCl overcomes this tendency. Thus, consideration of the control of extracellular concentrations in terms of the composition of the fluid reabsorbed shows that this process also explains the expansion of extracellular fluids during development of alkalosis and the contraction during recovery.

TABLE VI.

Glomerular Filtrate and Reabsorbate
Development of K Deficiency Alkalosis*

	Concentrations		Amounts	
	Filtr.	Reabsorb.	Reabsorbed	Excreted
Cl mM	110	100	100	10
Na mM	140	140	140	0
H_2O gm	1000	1000	1000	0

*The table applies equally well to the compensated respiratory acidosis.

TABLE VII.

Glomerular Filtrate and Reabsorbate
Recovery from K Deficiency Alkalosis

	Concentrations		Amounts	
	Filtr.	Reabsorb.	Reabsorbed	Excreted
Cl mM	100	110	100	0
Na mM	140	140	128	12
H_2O gm	1000	1000	910	90

What, then, are the practical therapeutic implications of these studies? I am sure that alkalosis can develop without K deficiency. We have shown, however, that otherwise normal experimental rats do not develop alkalosis except when renal function is impaired by deficit of water. As long as renal function is relatively normal and body water has been maintained, alkalosis is apparently accompanied by some degree of K deficiency. Furthermore, as long as K deficiency persists, the kidneys maintain alkalosis owing to the change in renal function shown in the experiments which have been discussed. For this reason the treatment of alkalosis should practically always include the administration of KCL as well as NaCl. KCl is essential as long as food cannot be taken. The cure of alkalosis with K deficiency by NaCl alone is only apparent since food contains abundant K. The use of NH_4Cl is contraindicated since the changes in body composition show that the deficit of base in the cells is greater than the deficiency of Cl in extracellular fluids. The body suffers from acidosis of the cells which is greater than the alkalosis of extracellular fluids. NH_4Cl can only aggravate the cellular acidosis while temporarily restoring extracellular bicarbonate.

The amount of NaCl required is variable. If Na concentration is low, about 60 ml per Kg of physiologic saline in adults and perhaps 80 ml in infants is indicated. However, if serum Na concentration is normal, 20 to 40 ml per Kg should suffice.

The deficit of K is likely to be at least 6 mEq per Kg and may be as great as 17 mEq per Kg. Owing to the dangers of too rapid administration of K, the deficit of K cannot be rapidly replaced and usually requires four to six days of K therapy for complete correction.

Administration of K requires two considerations: (1) the normal rate of expenditure when all fluids are given parenterally. This is 1 to 2 mEq per 100 calories metabolized or 1 to 2 mEq of each 125 to 150 ml of fluid administered to maintain body water; (2) sufficient additional K must be given to assure K retention. It is safe to administer 3 mEq of KCl per Kg of body weight per day if renal function is normal and a cellular deficit of K is present. More than this amount of K may be given if it is known that serum K remains low or if electrocardiogram shows the changes characteristic of low serum K concentrations. The rule is to inject the estimated dose of K for twenty-four hours over a period permitting equal distribution in body fluids. If the total dose is given over a period of four hours, equal distribution should be attained. However, it is recommended that the daily dose of K be given over a period of at least six hours.

The KCl is added from a concentrated solution to the fluids planned for a certain period of time —usually twelve to twenty-four hours. The solution which we use contains 2 mM KCl per ml, but the concentration is greatly reduced in the fluids injected. It is possible to give adequate amounts of K to infants at a concentration of 25 mEq or less per liter since the water requirement is high. In adults it is difficult to give the indicated amounts of K at a concentration less than 40 mEq per liter. Many physicians use higher concentrations, however very careful control is necessary if higher concentrations are used. Furthermore, concentrations greater than 40 are often painful when injected into veins.

The above rules make K therapy relatively safe if the disturbances in body composition and renal function are understood. Confusion will be avoided if the requirement of fluids for twenty-four hours is estimated. The deficit of water is likely to be 50 ml per Kg and is unlikely to be greater than 100 ml per Kg. The deficit of NaCl is likely to be equivalent to 20 to 40 ml of physiologic saline per Kg and only when Na concentrations are low will NaCl deficits be as great on the contents of 60 ml of physiologic saline. The water and NaCl may be given rapidly and thereby restore circularly and renal excretion. Although K deficits cannot be corrected rapidly, K should be started as soon as feasible, usually within two hours. The requirement for maintenance is added to the replacement of fluids. The former is computed on the basis of 125 to 150 ml per 100 calories of estimated heat production; about 3 mM of NaCl and 1 to 2 mM of KCl should be contained in each 125 to 150 ml of maintenance fluid. Except for about 20 ml of physiologic saline in 125 to 150 ml, the bulk of the maintenance fluid is five or ten per cent glucose in water. Additional KCl is added to provide 1 to 3 mM of KCl for replacement. The second day of therapy is the same except that only maintenance water and NaCl need be given, but KCl should be given at the rate of 1 to 3 mM per Kg per day. This form of therapy considers the magnitude of the deficits of water, Na, Cl and K, and the role of K in renal function. It has been safe and effective in our hands. The present studies show that K is necessary to prevent alkalosis when all fluids are given parenterally and food is not ingested. Addition of K is particularly necessary to prevent alkalosis after operation and is essential when gastric suction is used.

Pediatric Electrolyte Problems

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THE REALLY difficult assignment this morning, laying the groundwork for the presentation of what I hope will be some practical points in pediatric fluid therapy, has been ably handled by Doctor Darrow. I have chosen to talk largely about the management of pediatric surgical patients, for it is my opinion that other diseases requiring parenteral alimentation are of relatively less importance today than they used to be. It appears to me as a pediatrician in a children's hospital that the challenge of the postoperative management of the infant is a real one. Therefore, it is to the discussion of this problem that I would particularly like to call your attention.

First let us consider some basic assumptions.

1. It is obvious to all that when an individual stops eating he develops a deficit of sodium, chloride, potassium, probably of phosphorus, magnesium, nitrogen, and calories. In the absence of water intake, a patient will also become deficient in water. Therefore, logically, any parenteral fluid regimen must provide not only water and calories but also a variety of electrolytes.

2. It is probably easier to calculate the fluid requirements for an individual on the basis of surface area than it is upon the basis of body weight. It is my own feeling that it is also safer to use surface area in calculating fluid requirements since the major, possibly the only, safety valve provided in the body for the excretion of excess electrolytes or water is the kidney, and the capacity of the kidney to handle water and electrolyte is a function of surface area and not of body weight.

3. We assume that the body is better able to cope with parenterally supplied materials when these are given throughout a twenty-four-hour period rather than sporadically, for one, two or three hours several times during the twenty-four-hour period. Therefore, the regimen outlined will be a twenty-four hour regimen.

4. It is dangerous to administer potassium to a patient who has severe hemoconcentration secondary to dehydration. There are two reasons for this. One danger is that the serum potassium concentration may already be elevated; the second is that the kidney which normally excretes potassium is not functioning adequately because of a decrease in the glomerular filtration rate. Hence injection of potassium containing solutions under these circumstances may lead to toxic levels in the plasma.

5. There is a final, very important, assumption which is made and upon which the fluid program to be presented is predicated: any individual who has suffered trauma or stress, be it the stress of surgery, the stress of infection, or the stress of diarrheal disease, has lost potassium from his cells. The logical conclusion is, therefore, that any parenteral regimen designed to treat ill individuals should contain potassium. It is obvious that this does not apply to a patient with Addison's disease, it probably does not apply to a patient with chronic renal failure and it does not apply to certain other specific illnesses. But in the ordinary course of medical and pediatric practice, it seems to be a valid assumption.

Table 1 indicates the composition of the two main body fluid compartments; the extracellular and the intracellular. It also indicates the composition of two different fluids designed for parenteral administration. Both of these have 10 gm. of carbohydrate per 100 ml. of solution. Your attention is called to the fact that in extracellular fluid there is a difference between the concentration of sodium and chloride. This is a significant difference and probably should be maintained in fluids which are used for parenteral alimentation. We call the two fluids we use for parenteral alimentation polyionic solutions No. 1 and No. 2. Each is made up in 10 per cent dextrose and water and they are rather similar in composition to those

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originally described by Butler. You will notice that both of these solutions are quite dilute with respect to sodium, when compared to extracellular water. The sodium in extracellular water is about 155 mEq/L while these solutions have 30 and 57 mEq/L, respectively; in other words, one is one-fifth isotonic and the other is approximately one-third isotonic. You will also notice that there is more sodium than chloride in both of these solutions, and finally, that both of them contain potassium, some phosphate and that P.I.S. #2 contains magnesium.

Table I.

COMPOSITION OF PARENTERAL FLUIDS
AND BODY WATER

	meq/L				
	Na	Cl	K	PO_4	Mg
H_2O_E	155	110	5	3	2
H_2O_I	5	?	155	100	44
P.I.S. #1	30	22	15	3	-
P.I.S. #2	57	50	25	12.5	6

Table II.

COMPOSITION OF MILK

	meq/L		
	Na	Cl	K
Colostrum	18	32	22
Mature Human Milk	6	23	10
Cows Milk	30	42	35
Formula, 1/3 water	20	28	24
P.I.S. #1	30	22	15

Since there is a difference between the concentration of the sodium and chloride in these solutions, it is logical to assume that there must be some anion present to produce electrical neutrality. The anion present is lactate, but this does not mean that these are alkalinizing solutions. It simply means that lactate happens to be the convenient and biologically acceptable anion for the purpose. The pH of both solutions is about 7.4.

Table II indicates certain facts about the composition of milk. Since milk is the major source of food of most infants, I think it is important to examine its electrolyte content. Let us first look at mature human milk. You will notice that it is rather dilute with respect to sodium, rather concentrated with respect to chloride and that potassium concentration is greater than the sodium concentration. Cow's milk is used for the formula of most children and you can see that this milk is considerably more concentrated than human milk with respect to electrolyte. It is particularly generous in its sodium and chloride content. We conveniently dilute cow's milk with water in the usual infant formula but this nevertheless still provides a food considerably more concentrated with respect to sodium, chloride and potassium than mature human milk.

Table III.

COMPOSITION OF INTESTINAL CONTENTS

	Na	meq/L Cl	K
Nasogastric decompression (18)	46	72	7
Ileostomy, recent (adults)	130	115	11
Ileostomy, recent (child)	134	81	14
	116	90	12
	96	70	11
Ileostomy, adapted (adults)	45	20	3

Table IV.

FLUID REQUIREMENTS FOR A 1 M^2 INDIVIDUAL (30 kg.)

1% Glomerular Filtration Rate for urine:	
100 L/M^2/24 hours:	1000 cc
Insensible water Loss:	
500 cc/M^2/24 hours:	500 cc
Loss: 10% of body weight:	
3000 cc	
Try to replace 50% water:	
Loss in 1st 24 hours:	1500 cc
Total fluid required:	3000 cc
In 2nd and 3rd 24 hour periods can replace remaining 50% of fluid loss:	2200 cc/24 hours

One of the important periods of life, when surgery is difficult but not infrequently necessary, is during the newborn period. Physicians then are faced with the problem of providing parenteral nutrition to newborn patients with illnesses such as tracheo-esophageal fistula, or other congenital intestinal abnormalities. I would like to remind you that a breast-fed infant of one, two, three or even five days of age is probably not going to get mature human milk but rather colostrum. Colostrum is considerably more concentrated in sodium, chloride and potassium than is mature human milk and is more like a cow's milk formula than human milk. Therefore, we feel, in this group of patients, it is sound to provide a solution for parenteral administration during the early neonatal period similar in composition to colostrum. Newborn infants with intestinal anomalies, although in need of surgery, are not deficient in electrolyte and water as is a child who has had diarrheal disease, or an infant who has had pyloric stenosis and has vomited for a week. These are patients who simply need maintenance amounts of fluid and we feel it is safe to administer a fluid whose composition is similar to the one we call P.I.S. #1. The amount administered should be similar to that which an infant would be receiving at breast or from a formula that might be offered to a similar infant who is without disease.

Most patients who have gastrointestinal surgery also have a tube put into their stomach for the purpose of relieving distention. In the process of relieving distention, suction is applied and intestinal contents are removed. Table III records the average of eighteen determinations of electrolyte composition of material removed by stomach tube from six different children. You will note that there is more chloride than sodium in this fluid; this you might have expected, but the chloride concentration is considerably below that of plasma. I would like to call your attention also to the extraordinary amount of electrolyte which is lost in new ileostomies. You can see the dramatic change in salt content which occurs with adaptation and also the fact that the composition of ileostomy material of children and adults seems rather similar.

On Table IV is outlined the parenteral fluid program to which we subscribe at the present time. The data are indicated for a 1 M^2 individual; you may remember I mentioned that we were going to use surface area rather than weight as a basis for our calculations. We assume that an ideal urine volume is approximately one per cent of the glomerular filtrate which in a one square meter individual is roughly 1000 ml. per day. We assume that the insensible water loss is 500 ml/M^2/24 hours. It might be well to remember that a 30-kilogram individual is approximately one square meter. Any patient who is severely dehydrated has lost between 7 and 10 per cent of his body weight as water. In adults this figure can go considerably higher, but this is a useful one in pediatrics. Ten per cent of a 30-kilogram individual would be 3000 ml. We can only replace a limited amount of the initial loss in the first twenty-four hours, and a reasonable figure would be 50 per cent of that loss. That would mean that such a patient required 1000 ml. for urine,* 500 ml. for insensible water loss and 1500 ml. for replacement of his initial water deficit. Therefore, during the first twenty-four-hour period we would provide 3000 ml. of fluid for a dehydrated patient. Now the solution described on Table I, P.I.S. #2, was constructed so that if sufficient water be provided to a patient one would automatically provide sufficient electrolyte.

During the second and third twenty-four-hour periods, one tries to replace the rest of the 50 per cent deficit; this will be half of 1500 ml.; that actually makes a total of about 2200 ml. for the second and third twenty-four-hour periods. We have modified this to make it 2400 ml/M^2/24 hours only because it is an easy figure to remem-

Table V.

THEORETICAL FLUID AND ELECTROLYTE REQUIREMENTS
OF 1 M^2 INDIVIDUAL REQUIRING ACUTE SURGERY

	ml	meq		
		Na	Cl	K
I.W.L	500	11	11	2
Continued Urine Loss	-	29	25	48
Usual Urine Loss	1000	17	13	13
50% Replacement	1500	112	82	27*
Ng. Suction	500	23	36	4
Total	3500	192	167	94
Amount Provided	3500	200	175	88

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*The volume suggested for urine presupposes minimal renal work and a specific gravity of 1.012.

ber. Because the losses of water and electrolyte in urine may be quite high if disease continues beyond three days (the period after which we assume deficits have been met) we administer 2400 ml/M^2 thereafter and as long as parenteral fluids are necessary. When some fluid is taken by mouth, this can be subtracted from the 2400 ml to be given by vein. Not included in Table IV is the program for a patient who requires parenteral alimentation but has no deficits of salt or water. This patient will require no more than maintenance amounts of fluid, about 1500 ml/M^2/24 hours. If gastric suction is in use, the salt loss will be greater than otherwise hence P.I.S. #2 should be used not only for the basic fluid requirement, but also for replacement of losses from the gastro-intestinal tract. When there is no gastric intestinal suction, basic fluid needs can be met by P.I.S. #1.

To recapitulate, we provide 3000 ml. of fluid per square meter in the first twenty-four hours, 2400 ml per square meter thereafter. For the non-dehydrated patient, 1500 ml/M^2/24 hours is sufficient. These are not absolute figures, but they are convenient to remember, and come close to the needs of most patients.

Because of the danger involved in giving potassium containing solutions to anuric patients, in dehydrated patients we use an initial fluid of one-third isotonic saline (50 mEq/L) in 10 per cent glucose as a hydrating solution. This is administered at the rate of 360 ml/M^2 in forty-five minutes. Such a solution given at this rapid rate initially expands the plasma volume and almost always results in urine formation. If, after the fluid is administered, a patient still has not voided (this is rare in our experience), the rate of fluid administration is reduced to 2400 ml/M^2/24 hours and continued until the patient does void. After the hydrating solution has been given and the patient has voided, it is safe to start the potassium containing solution.

TABLE VI.

MAINTENANCE REQUIREMENTS OF FLUID AND ELECTROLYTE, 1 M^2 INDIVIDUAL

	ml	meq		
		Na	Cl	K
I.W.L.	500	11	11	2
Continued Urine Loss	-	29	35	48
Usual Urine Loss	1000	17	13	13
25% Replacement	750	66	41	27
Ng. Suction	500	23	36	4
Total	2750	146	136	94
Amount Provided	2900	165	145	73

When there are losses by nasogastric suction, these must be measured and replaced ml for ml by an amount of P.I.S. in addition to the calculated twenty-four-hour requirement. In practice it has proven practical to add the extra fluid on the subsequent day, so that one can plan the fluid program for each day on the basis of the losses during the previous twenty-four hours. (P.I.S. # 2 Table VIII.)

Table V indicates the derivations of the electrolyte composition of P.I.S. #2. The volumes used in preparing this chart are similar to those used in Table IV; the electrolyte figures used have been derived from a number of different sources, but because of time limitations I shall not discuss them in detail at the present time. Since today we are particularly interested in surgical problems, I have included a theoretical fluid and salt loss by nasogastric tube. You can see that in supplying to this patient 3000 ml/M^2 plus the nasogastric loss, in theory, we have replaced

50 per cent of his previous water, and presumably, electrolyte loss and furthermore made available water, calories and salt for maintenance.* The asterisk after potassium in the "50 per cent replacement" column indicates that 24 mEq. represents only 25 per cent of the potassium loss, while the figures for water, sodium and chloride represent 50 per cent of the loss. There is a good reason for this: By use of K^{22} it has been demonstrated that only 25 per cent of a potassium deficiency can be replaced per twenty-four hours. Hence it seems unnecessary to provide more than 25 per cent of the deficit per twenty-four hours.**

Table VII.

PLASMA VALUES OF CHLORIDE AND BICARBONATE:

Effect of Potassium Therapy in Pyloric Stenosis

Day of Rx:	1	2	3	4	5	6	7	8	9	10	11	12	16
POTASSIUM													
CO_2 meq/L	38		38		32		31		-				25
Cl meq/L	100		-		70		84		94				103
NO POTASSIUM													
CO_2 meq/L						-		39	39	39			
Cl meq/L						75		71	94	80			

In Table VI similar data indicating that the requirement for fluids and salt on the second day is less than on the first. Instead of 1500 ml you need 750 ml for replacement. Otherwise, the figures are similar.

Now, let's look at something practical. Does this system work? You might ask, "Can you use one type of solution for treatment of patients with either acidosis or alkalosis?" Before answering this question, I would like you to recall something that I said initially: we can assume that all patients who are under stress unless given exogenous potassium will become deficient. Given time they will manifest symptoms of potassium deficiency and may even exhibit characteristic serum values, an increase in carbon dioxide and a decrease in chloride concentration, provided they do not die of other causes. In the normal course of events we do not wait for the exhibition of these changes, and furthermore, we know that the initial concentration of CO_2 in the plasma does not indicate the degree of potassium deficiency. Finally, we know that the serum K concentration neither necessarily reflects the concentration of this ion in the cells nor does it bear any predictable relation to the degree of overall K deficiency. Only in rather severe K deficiency does the serum level fall. In Tables VII and VIII are data from three patients, one with acidosis and two with alkalosis.

The first patient in Table VII had pyloric stenosis and was treated with sodium and chloride-containing solutions (as well as glucose) until the sixth day. You will note that he exhibited the conventional values for potassium deficiency; the bicarbonate was increased and the chloride decreased. Potassium administration was started on the sixth day; the chemical values of his blood improved and after operation on the twelfth day, the chloride and carbon dioxide were normal. Obviously this chart was not prepared to illustrate ideal management of pyloric stenosis. Rather we have chosen a patient who illustrates the failure of sodium chloride therapy and the success of potassium therapy in the presence of hypochloremic alkalosis. Tabulated on the same chart are data from a patient with pyloric stenosis who did not receive potassium, but rather normal saline. On the eighth day he received ammonium chloride intravenously in an attempt to correct the hypochloremia and you will see that on the next day his serum chloride was 94, but by the following day it was back to 80 mEq/L and the alkalosis was apparently unchanged. On the basis of what is known about blood pH in potassium deficiency, I think it would be safe to assume that both of these patients were initially alkalotic, even though we do not have pH values to prove it.

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*If one converts the total electrolyte loss to mEq. per liter of fluid lost, one obtains a solution about one-third as concentrated in respect to sodium as is extracellular water.

**This also means that at the end of three days, when the deficits of Na and Cl will have been completely relieved, only 50 per cent of the K deficit will have been made up. This is not a serious problem, however, for some of the K loss represents loss of protoplasm with its associated K, and so the cell need for replenishment of this ion is not as great as the total loss. Only when new protoplasm is made and deposited in the cell will more K be needed.

Table VIII.

D.D. 9 months Day	1	2	3	4	5	6
Wt. kg. 8.02:	.	.	.	.	.	.
S.A. M^2 0.4:	.	.	.	.	.	.
Loss:	360	550	255	450	0	0
Intake: actual: cc	635	1200	1375	1350	1350	1495
Intake: theoretical:	.	.	.	.	.	.
Requirement cc	600	960	960	960	960	960
Loss cc	.	360	550	255	450	0
Total cc	600	1320	1510	1215	1410	960
Plasma Values:						
Cl meq/L	112	101	92	97	.	.
CO_2 meq/L	6	19	30	29	.	.
Total intake/ 24 hours cc:					1345	
Total theoretical intake/24 hours cc:					1275	

Table VIII contains data on both treatment and chemical values in the blood of a patient with an intussusception who was acidotic. I call your attention only to the figures for carbon dioxide and chloride. He entered the hospital with a CO_2 of 6 mEq/L and a chloride of 112 mEq/L and received the amounts and type of fluid predicted by our theoretical considerations. You will note that his acidosis was rapidly relieved (operation was performed on the first day), and he quickly returned to normal electrolyte equilibrium.

We have here what, from the chemical point of view, seems to be two entirely different situations. From the therapeutic point of view, however, it seems clear that both can be handled in the same way.

Table IX.

INADEQUATE POST-OPERATIVE MANAGEMENT

Pt: S.C. BCH #110 872

Age: 9 yrs.

Wt: 32 Kg.

S.A: 1 M^2

D_x: Congenital Dislocation of Hip

Parenteral fluids administered: 13 days

Total, L.

Intake:			39.6
Output:	Urine	14.2	
	I.W.L. (est)	6.5	
	Ng. Tube	16.4	
			37.1

You might at this point raise an important question. Is it necessary to give parenteral fluids to all surgical patients? Probably not; however, we take the general position that almost all patients who have undergone abdominal surgery and re-

TABLE X.

INADEQUATE POST-OPERATIVE MANAGEMENT

Pt. S.C. BCH #110 872

Day	1 - 4	5	6	7	8	9	10	11	12
Theot. Deficit, ml	-9160	0	-1400	+100	-700	+100	-1400	-2100	-400
Na meq		68	196	180	110	35	134	131	25
Cl meq		40	170	70	88	22	118	110	23
K meq		20	91	35	82	22	46	57	11
Plasma, meq/L									
Na						133	138	138	142
K					3.5	3.9	4.9		4.8
Cl		96			75	75	78	84	87
CO_2		21			42	40	45	36	37
pH						7.59	7.45	7.45	

quire either the withholding of oral alimentation, or gastro-intestinal suction, need parenteral fluid therapy. A second question might also be posed at this time: Is the rule of thumb 2400 ml/M^2/24 hours necessary or valid for all patients? Again the answer is "probably not." However, on the basis of theoretical data and clinical experience this seems to be a very safe amount of fluid and I prefer to supply fluid to an occasional patient who could get along without it than deny it to a patient who needs it.

The data on Tables IX and X illustrate what happened to a child whose postoperative course was poorly managed. They illustrate what was a catastrophe, for a patient's life was lost because the assumption was made that only minimal amounts of parenteral fluid were necessary. This was a nine-year-old child who had a congenital dislocation of the hip, and came into the hospital for surgical correction of this defect. During a period of thirteen days, her intake of parenteral fluid was almost 40 liters. Her output was just a little bit less, so one might conclude from superficial examination that she got plenty of parenteral fluid. But three important errors were made in management. In the first place, she received only glucose and water for the first two days after surgery. Then, when she developed intestinal distention and hypochloremia, she received normal saline in large amounts. That would be a second error. A third error was made when her nasogastric tube was irrigated with tap water so that in essence she received tap water by mouth and the nasal gastric suction naturally withdrew fluid of the composition of gastric contents. She never did rally, and in spite of the fact that potassium administration was begun in modest amounts on the ninth day, she expired, apparently of severe electrolyte imbalance. The balance data in Table X are theoretical, since the figures were derived by subtracting actual intake from theoretical requirements in this case, 1500 ml/M^2/24 hours plus loss by gastric suction.

Let me call your attention to the plasma values for sodium, potassium, chloride, CO_2 and pH. She received insignificant amounts of potassium until the ninth day, and on that day her pH was 7.59, her chloride was 75 mEq/L and her CO_2 was 40 mEq/L. Even during the period when she received potassium, unfortunately she continued to get tap water by mouth which was withdrawn by nasogastric suction. This patient's course illustrates the fact that it is possible for a child who has not had gastro-intestinal surgery to get into difficulties due to improper fluid management. It is probable that if this child had received proper parenteral fluids from the beginning, parenteral fluids containing potassium, she would have survived her surgery.

The next problem to concern us is that of the management of patients with an ileostomy. In this situation there is one important rule to remember: you must treat the patient and forget preconceived ideas of fluid and electrolyte requirements. Fluid losses in an ileostomy may be enormous. Figure 1 indicates how much water can be lost by a patient with an ileostomy and how much replacement may be necessary. This was a child who weighed three kilograms and was followed for about forty days with an ileostomy. At times, during that period, it was necessary to provide as much as 70 per cent of his total body water by parenteral fluids and oral fluids simply to meet ileostomy losses. The chart indicates that in spite of these enormous amounts of fluid, he did not gain commensurate amounts of weight and hence, the fluid given simply replaced what was lost. Our experience with this patient led us to the conclusion that ileostomy losses should be treated the same as the losses by nasogastric suction. They should be replaced ml. for ml. by the solution being used for parenteral maintenance.

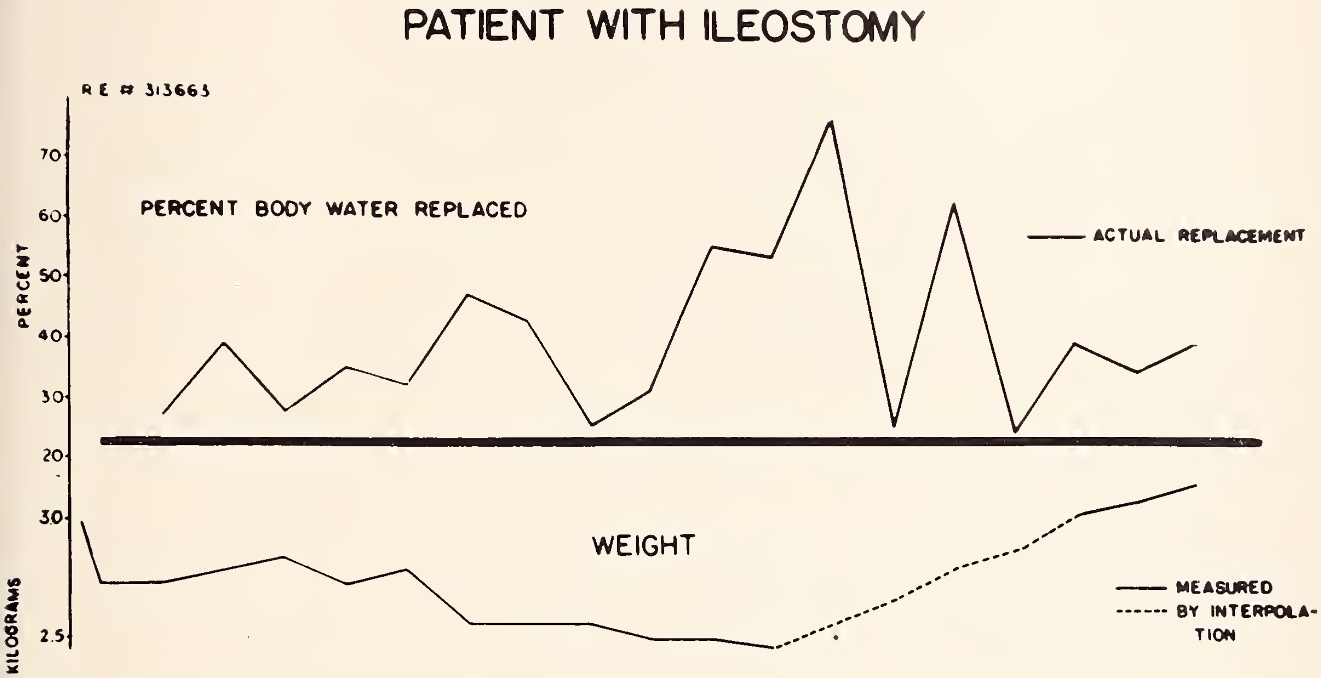

Fig. 1.

The child whose data are noted in Table XI had an ileostomy. He was a six-months-old infant weighing eight and one-half kilograms, who had a volvulus that required resection, and he required maintenance with an ileostomy for thirty days. During these thirty days, we followed the general plan outlined above using P.I.S. #2. Some of the details of therapy for the first eighteen days are indicated on Table XII. During this period we tried to make out a balance sheet based on theoretical considerations to compare actual intake and anticipated needs. Whether or not we were correct we will never know because we have no true balances, but we have the values of the plasma electrolytes and these usually reflect the efficacy of parenteral therapy. This child, who weighed eight and one-half kilograms, required as much as 2,000 ml a day by vein, which was as much as 50 per cent of his total body water; but,

Table XI.

MANAGEMENT OF ILEOSTOMY, INFANT

Pt: D.H. BCH #176 364

Age: 6 mo.

Wt: 8.5 Kg.

S.A: 0.4 M^2

D_x: Volvulus with resection

Parenteral fluids administered: 30 days

except for a period between the sixth and ninth day (the resection was done on the second day), the electrolyte values in the serum remained near normal. We collected the ileostomy output just as we would have collected nasogastric tube contents, measured the volume and replaced it ml for ml with the type of fluids outlined. This child was managed entirely with P.I.S. # 2.

We felt that we could have given less fluid to this patient if we had used a more concentrated solution. Therefore, in subsequent patients we have used a solution more concentrated with respect to electrolyte. Its composition is indicated on Table XIII.

The data which follow demonstrate what can be done in comparing anticipated requirements based on the assumptions mentioned above with actual needs as measured by balance data. Table XIV indicated the clinical data of a child with acute ulcerative colitis who had a resection of

Table XII.

MANAGEMENT OF ILEOSTOMY, INFANT

Pt: D.H. BCH #176 364

18 days

Theot. water req., L	20.3
Act. water intake, L	22.6
Theot. Balance, ml/day	+130

Day	1	2	3	6	9	11	18	27
Weight, Kg	7.0			8.7	8.9	8.1	8.0	7.4
Plasma, meq/L								
Na		139	135	132	125	131		
Cl	107	100	99	80	82		100	103
K		3.7	3.0	3.2	3.3	5.1		
CO_2	22	19.7	27	34	35		30	27.5

Table XIII.

SPECIAL SOLUTIONS FOR PATIENTS WITH ILEOSTOMIES

meq/L	
Na	100
Cl	75
K	30

Table XIV

MANAGEMENT OF ILEOSTOMY

Pt: J.G. BCH #199 321

Age: 8 yrs.

Wt: 20 Kg.

S.A: 0.75 M^2

D_x: Acute Ulcerative Colitis

Parenteral Fluids Administered: 14 days

Total, L. (12 days)

Intake:			37.9
Output:	Urine	15.4	
	I.W.L. (est.)	4.2	
	Ng. Tube	0.65	
	Ileost.	15.7	
			35.95

her colon and an ileostomy and required parenteral fluids for fourteen days. During this period her intake was 40 liters and her output was 35 liters. Her ileostomy loss during that period was about a liter per day. You can see that her replacement needs were very large. We used the solution indicated in Table XIII exclusively.

Table XV records some of the data concerning her course. I would like to call your attention to the fact that her CO_2 remained almost normal throughout the total period of parenteral fluid administration. Her chloride fell at one point but then rose toward normal and on the twelfth day was almost normal.

Table XV.

MANAGEMENT OF ILEOSTOMY

Pt: J.G. BCH #199 321

Day	1	2	3	4	5-9	10	11	12
Theoret. Water Balance	-300	+269	+270	+510	+ 10	+ 25	+1395	- 85
Body Wt., Kg.	19.7	18.5	20	20	19.5	19	19	
Plasma, meq/L								
Na		135	140	142	143	133	140	137
Cl		93	93	95	92	87	94	97
K		4.1	6.2	5.7	5.7			
CO_2		29.6	33	26.5	27	24.5	26	26
pH			7.29	7.44	7.25	7.32	7.30	7.42

Table XVI.

MANAGEMENT OF ILEOSTOMY

Pt: J.G. BCH #199 321

Intake, Day		1 - 7		1 - 12	
		Act.	Theot.	Act.	Theot.
H_2O	L	21	18.8	37.9	35.86
Na	meq	2098	1915	2978	3587
Cl	meq	1577	1436	2349	2684
K	meq	630	574	1172	1075

Theot. Daily Balance		1 - 7	1 - 12
H_2O	ml	+300	+170
Na	meq	+ 26	- 51
Cl	meq	+ 20	- 28
K	meq	+ 8	+ 8

Table XVI is an attempt to make a balance of her data. The intake and balance per day are recorded in two grouped periods; days one through seven and one through twelve. The theoretical data represent what we think she should have had, and the actual data represent what we succeeded in providing. This discrepancy is the result of technical difficulties such as needles coming out of veins, etc. The division into two periods was necessary because the patient started to eat after the seventh day, and her food was largely milk. Unfortunately, milk is a poor source of electrolyte at least when it is used to replace salt lost from an ileostomy (see Tables II and III) and the negative theoretical balance is the result of this. The theoretical balance represents the difference between what we think this patient needed and what she actually received. During the first seven days, she should have been in a positive balance with respect to water and electrolyte. As soon as milk was substituted for a portion of the parenteral intake she went into a theoretically negative balance of sodium and chloride, but, since milk is fairly high in potassium she probably did well with respect to that ion.

Table XVII.

MANAGEMENT OF ILEOSTOMY
BALANCE DATA

Pt: J.G. BCH #199 321

Cumulative Balance, Days 2, 3, 4	Retention/24 hrs Theot.	Actual
H_2O, ml	+349	+281
Na, meq	+ 33	+ 10
Cl, meq	+ 26	+ 30
K, meq	+ 10	+ 7

Fortunately, we were able to collect and analyze excreta and obtain accurate balance data on days 2, 3, and 4. Table XVII* is a tabulation of this information. We avoided trying to measure losses on the day of surgery because we would have had no way of estimating the electrolyte which was lost when the colon was lifted out of the body; therefore, we started our balance studies on the second day and have charted them as cumulative data. Theoretical retentions taken from Table XVI are compared with the measured retentions. You will see that there is a rather surprising agreement between these two sets of data. In other words, on the basis of theoretical thinking, we were able to calculate the right amount of salt and water for this patient. We have data like this on other patients which lead us to believe that it is possible on the basis of theoretical assumptions to come quite close to approximating the needs of children during the postoperative period and after ileostomies.

Conclusion

In closing, I would like to recapitulate the rules of thumb which we use for managing postoperative infants. I think the most important one is to treat a patient, not chemical values of plasma. Measure intake precisely and output if possible. Irrigate nasogastric suction not with tap water but with either a polyionic solution or, if necessary, with a half normal saline solution. Remove nasogastric suction tubes as soon as possible. Patients do better without a nasogastric tube in place. The blood or plasma administered should not be included as a component in calculations of water intake. Replace nasogastric or ileostomy losses ml for ml with whatever solution has been deemed necessary for that patient. Weigh a patient initially and daily. Weights are very simple things to get and are helpful in trying to check the efficacy of a fluid regimen. Use dilute solutions; do not use normal saline. We think half normal saline is probably too concentrated for many patients; third normal saline is better. Try to use a solution in which the ratio of sodium to chloride is similar to that which exists in the plasma. In general it is possible to use the same solutions and volumes for acidotic and alkalotic patients. For maintenance requirements of newborn infants without unusual losses, use a solution similar in composition to colostrum, like P.I.S. #1; for older children use P.I.S. #2, and for patients with an ileostomy make up a solution similar to that on Table XIII. For following a patient's electrolyte balance it is probable that it is sufficient to determine the CO_2 and chloride content of plasma.

Give 3,000 ml/M^2 for a dehydrated patient dur-

(Continued on Page 32)

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*Taken from unpublished data: Lowe, C. U. and Calcagno, P. L.

Development and Correction of Electrolyte Disturbances Associated with Salt Retention

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FROM the standpoint of salt and water metabolism, the body may be regarded as a series of differentiated fluid phases separated by discriminating cell membranes. For clinical purposes, it is convenient to divide these fluid phases into an intracellular phase and an extracellular phase.

The extracellular phase, in turn, consists of the blood volume and the interstitial fluid volume. Edema represents an expansion of one component of the extracellular phase, the interstitial fluid. It always develops under circumstances where, for one reason or another, the kidneys retain excessive amounts of salt and water that have either been ingested by mouth or administered parenterally. Under these circumstances both salt and water accumulate in the body, selectively become deposited in the interstitial fluid, and should the magnitude of this accumulation be sufficient, pitting edema becomes evident. Salt retention under most circumstances appears to be the primary initiating event secondary to which water is retained. The consequences of salt retention, therefore, become very important to evaluate under circumstances where edematous states exist. We know from clinical evaluation of a variety of different disease states that many different disorders are characterized by edema (i.e., cirrhosis of the liver, nephrosis, and congestive heart failure are all, in part at least, associated with massive accumulations of salt and water).

The significance of this salt and water accumulation, however, is not as readily apparent as it would seem at first sight. Everybody agrees that these large accumulations are mechanically deleterious in the sense that they are unsightly. They deposit in the legs leading to ulcers. They may deposit in the gastro-intestinal regions leading to impaired appetite and gastro-intestinal symptoms of one kind or another. It is important to evaluate in addition to these mechanical effects of salt and water retention those effects which may have more profound physiological significance. This is particularly true in the case of congestive heart failure.

In order, therefore, to approach the importance of salt and water retention as well as the measures which may be applied to rid the body of this excessive accumulation of fluid, it is well to evaluate on the basis of current information at least what might be the sequence of events leading to the retention of salt and water.

Initially, in some sense, the heart fails. Precisely what this means today cannot be defined, but it appears at least to be accurately described by the following:

It is clear from Starling's law that the force of systolic contraction is directly proportional to the amount of disastolic stretch up to a point beyond which the force of systole declines with increased disastolic stretching. It is thought by many, therefore, that during the course of heart disease, arteriosclerotic heart disease, or hypertensive heart disease, the myocardial fiber is injured and can no longer expel the blood that is returning to it with the same force as in the normal state. Under these circumstances, a tiny increment of blood remains within the heart, causing diastolic stretching and thereby facilitating a more forceful systole. As heart disease progresses over a period of years, the amount of blood within the cardiac chamber mounts at times to astronomical proportions.

Bing has estimated, for example, that in chronic congestive heart failure there may be as much as 800 or 1000 cc. remaining within the cardiac chamber and in diastole. This large accumulation of blood during diastole permits stretching of the myocardial fiber and thereby permits the heart to expel a normal or near-normal cardiac output. But the price the individual pays for maintaining the cardiac output at near-normal level is a marked rise in the diastolic filling pressure. After a period of years, this pressure rises to a point where the central venous pressure increases. The cardiac output under such circumstances may be normal, may be higher or may be lower. Whatever it is, the key point is the fact that there is a resistance, a high pressure within the ventricular chambers, as a consequence of which the flow of blood toward the heart is impeded and backed up.

The consequences of this increased pressure are clear again from the Starling equilibrium. When venous pressure rises there is a tendency for transudation. This in turn causes a shrinkage of blood volume so that there is a redistribution of extracellular fluid. The blood volume decreases, interstitial volume expands, but the total extracellular volume remains unchanged. At this point, however, as a consequence of something which is not clear, this disequilibrium causes the kidneys to react by retaining salt.

This reaction has been ascribed by some to a decrease in blood volume. Others have implicated a low cardiac output. Still others have considered increased venous pressure as the stimulus to cause the kidneys to retain salt. Whatever it is, the kidneys respond to the congestive state by a tenacious retention of salt and water so that virtually no salt may appear in the urine. And this retention is largely a consequence of enhanced tubular reabsorption of sodium. This enhanced tubular reabsorption of sodium, in turn, leads to a concomitant retention of water, and therefore edema forms. At this point, a series of reactions ensue which are a direct consequence of edema formation. Precisely what the steps are in this reaction are not clear but it has been suggested that in some manner the formation of edema reexpands the blood volume to normal or perhaps supernormal values; this, in turn, increases the central venous pressure, thereby augmenting the magnitude of the congested state so that a vicious cycle appears to be set up by the failure of the heart where edema formation in a certain sense aggravates heart failure.

In the work of the British investigators, Pugh and Windom, an attempt was made to study precisely what effects ridding the body of salt and water alone might have in a patient with severe congestive heart failure. They found that when patients in severe heart failure were given mercurial diuretics, there was a marked fall in right auricular pressure. At the same time, the cardiac output rose and both of these, in turn, were correlated with a rising urine volume.

It appears, therefore, from studies such as these that ridding the body of salt alone by an agent whose sole action, according to present views, is on the kidney, has no direct effect on the heart whatsoever; ridding the body of salt may cause a precipitous fall in right auricular pressure and thereby improve the cardiac output and lead to some relief of the congested state. At present, it appears that in congestive heart failure ridding the body of salt accomplishes more than merely relieving the mechanical effects of fluid accumulation. There is a direct physiological effect. The mechanism may be unclear, but in some manner cardiac function is improved.

In order to approach this problem, it is well to turn to a consideration of those measures which are designed to correct the congestive state as it exists.

Pre-eminent in the treatment of heart failure is the use of digitalis with bed rest. This is true for several reasons. First, digitalis acts only under circumstances where congestive heart failure exists. It is not a diuretic under any other circumstances. Edematous states where heart failure is not present do not respond to the action of this agent. So under these circumstances, a successful digitalis diuresis is strong evidence for the existence of heart failure where a problem of differential diagnosis exists.

Second, a digitalis diuresis is always a physiological one in the sense that digitalis never causes a distorted loss of salt and water. It never disrupts the electrolyte composition of plasma; it never causes excessive loss of fluid. Digitalis, therefore, is a safe diuretic procedure in the sense that it reverses partially or completely the sequence of events which initially led to congestive heart failure.

Supplementing the use of digitalis with rest is a very important measure, because rest facilitates a fall in the work of the heart, for under more nearly basal conditions the individual does not have those demands for a high cardiac output which work imposes. If, therefore, one were to assign some sequence of events in the treatment of patients with congestive heart failure, one would assign pre-eminent importance to digitalis and to bed rest. Bed rest does not mean necessarily in bed; a cardiac chair or some similarly comfortable mode of resting is perfectly adequate.

In many instances, patients with severe congestive heart failure, adequately digitalized and provided with adequate rest, nevertheless fail to deliver their edema. Under these circumstances, one turns to other measures which may promote the renal excretion of both salt and water. Of these measures, mercurial diuretics are perhaps the most important as well as the most potent.

The urine of a prone, dehydrated man shows a marked excretion of sodium about an hour and a half to two hours after 2 cc. of mercurial diuretic has been injected. The excretion of sodium excedes the excretion of water. In other words, mercury is causing here a selective excretion of sodium in excess of water. And as might be predicted, under such circumstances, the concentration of sodium in the serum falls slightly. Later, there is a moderate acceleration of potassium excretion and the excretion of acid (ammonium simultaneously with titratable acidity rises). So, if we turn to the normal individual, then there are several effects which deserve comment.

There is first, as a consequence of mercurial action, an accelerated excretion of salt in water, but salt in excess of water. There is a moderate loss of potassium; there is increased acid excretion into the urine.

Urine output decreases as the excretion of solutes rises more or less in a linear fashion. Apparently mercury causes an accelerated excretion of water as a passive consequence of the excretion of salt. The loss of water appears largely, but not entirely, to be secondary to the accelerated excretion of sodium. A tiny increment of water excretion is due to the fact that because sodium is lost in excess of water, there is a transient inhibition of the posterior pituitary function resulting in a slight water diuresis during the later period. But by all odds, the major cause of the accelerated urine flow is accelerated solute excretion, and this, in turn, consists largely of the accelerated excretion of sodium.

In Wallace's data, the effects of mercury in a patient with congestive heart failure are noted. Here a very salutory diuresis has occurred which is normal in every respect. Sodium is lost with chloride in the same proportions in which they exist in extracellular fluid. There is no negative balance; potassium loss more or less parallels sodium loss. Serum concentrations are normal. Another situation exists, according to Wallace. Again mercury is given under repeated instances and here there is a loss of chloride far in excess of sodium. Accompanying this loss, there is a marked negative balance of potassium and here, in a way similar to Dr. Darrow's studies, there has supervened a marked alkalosis. The serum bicarbonate approaches almost 45 mEq. per liter and the serum chloride falls to almost 70 mEq. per liter. In other words, a mercurial diuresis has been accompanied by potassium deficiency and metabolic alkalosis, and the patient becomes refractory to mercury. This, then, is one of the causes of the refractory state. The cause of this potassium loss and the cause of the alkalosis are not clear, but I should like to mention one possible mechanism whereby this might be effected.

This is illustrated by the effect of the administration of ACTH and salt to a normal man. Under the circumstance where a patient is on a constant diet, there is a marked loss of potassium in excess of nitrogen accumulating to some 200 mEq. after ten days. At the same time, sodium and chloride are retained in the body, but sodium markedly in excess of chloride. This retention of sodium in excess of chloride can be shown, as Dr. Darrow has shown in his rats, to be a consequence apart from the fact that some of this retained sodium is not retained with chloride in the extracellular fluids but penetrates into tissue cells in exchange for potassium. While these changes are going on in the urine, one can detect a fall in the concentration of serum potassium and a fall in the concentration of serum chloride, despite the fact that the balance of chloride is negative.

The most noteworthy observation to which I would call your attention is the supervention of a metabolic alkalosis. The urine concomitantly exhibits a marked acceleration of acid excretion. Actually, after a period of ten days, the loss of acid approaches some 400 mEq. Therefore, it is necessary to suggest some hypothesis which might explain this marked alkalosis and potassium deficiency. Sodium reaches the distal tubule where under the impact of ACTH, cortisone or, as I shall try to argue, under circumstances of mercurial diuresis, it exchanges for potassium, and potassium is lost into the urine. This, in turn, leads to potassium deficiency. When potassium deficiency develops in this way, cells appear to yield their potassium with increasing restraint and, as a consequence, sodium begins to exchange with other cations, the principal one being acid cation.

If we examine what might proceed in the tubular cells, we can imagine the hydration of CO_2 and water under the impact of carbonic anhydrase leading to H_2CO_3. Sodium, in turn, may exchange with hydrogen causing an increased excretion of titratable acid. But the principal way sodium causes acid loss is by an exchange with ammonium. This is, by no means, the only mechanism whereby acid may be lost; there is doubtless a large loss of acid into the tissue cell. But here I would emphasize the fact that under circumstances where potassium deficiency develops, there is a marked loss of acid into the urine and that this loss of acid into the urine contributes in part at least to the development of alkalosis. From the standpoint of mercurial diuresis, this is exceedingly important because in cases where potassium deficiency develops, where alkalosis supervenes, individuals become refractory to the action of mercury. In addition, one has to cope with the deleterious consequences of potassium deficiency itself.

There are a number of symptoms which arise in association with potassium deficiency. First, there are the fairly specific symptoms. These are quite unimportant in most instances by virtue of the fact that they are so rare in their occurrence. If one has to rely on flaccid paralysis, one would practically never diagnose potassium deficiencies.

Second, there are a group of exceedingly important but highly non-specific symptoms which supervene. I say highly non-specific because any sick patient may exhibit these difficulties in varying degree. Perhaps the most important feature, from a clinical standpoint, which should lead to a suspicion of potassium deficiency is a clinical appraisal of those circumstances likely to produce potassium loss. Mercurial diuresis is surely outstanding in this respect in the cardiac patient, but it should be noted that some cardiac patients vomit, or become anorexic and do not eat. Recurrent injections of mercury, starvation and vomiting all are apt to produce varying degrees of potassium deficiency. Electrocardiographic evidence is of a non-specific type, but is helpful. There is prolongation of the QT interval, T wave inversion and ST segment depression, and the presence of a prominent U wave. These evidences of a potassium deficiency are not specific. Many diseases of the myocardium may eventuate in such disturbances, but if they are quickly reversed by potassium administration hypopotasemia is a likely cause.

Chemical evidence, such as a low concentration of serum potassium hypochloremia and alkalosis, is important. It should be pointed out, however, that hypochloremia is not an invariable accompaniment of serious potassium deficiency. Alkalosis, especially when it persists after salt and water replacement, is suggestive of potassium deficiency. This stipulation applies less to the cardiac patient; inasmuch as he is already edematous there is no shrinkage of extracellular volume, rather an overexpansion. But the supervention of alkalosis is one of the most suggestive features pointing to the existence of an underlying potassium deficiency.

Such considerations can probably be applied to the sequence of events in mercurial diuresis. Mercury may act by inhibiting the proximal tubular reabsorption of sodium, and as a consequence excessive amounts of sodium and water are delivered to the distal tubules. In normal subjects and in patients who are responsive to the action of mercury, the capacity of the distal tubule to reabsorb sodium is exceeded, and under those circumstances sodium excretion is accelerated. The accelerated excretion of sodium in turn obligates water leading to an accelerated urine flow. In addition, the slight hyponatremia, which is caused by a loss of sodium in excess of water by this process, may cause some inhibition of posterior pituitary secretion, as a consequence of which a mild water diuresis supervenes.

In patients who are refractory, it can be hypothesized that mercury, which is a tubular poison, may inhibit the sodium reabsorption just as it does in normal subjects but that the capacity of the distal tubule to reabsorb sodium is enormous. The stimulus for sodium retention is very intense, and as a consequence sodium is reabsorbed and part of this sodium is reabsorbed in exchange for potassium. Under such circumstances, potassium deficiency develops and potassium deficiency developing here may have the same consequences as potassium deficiency developing under the impact of sodium and DOCA which Dr. Darrow has earlier described. There is a change in muscle composition, hypochloremia of the serum may occur, and there tends to be a real loss of acid as ammonium titratable acidity in hydrogen leading in part to an alkalosis.

So, if we regard the effects of mercury, there are certain effects which this substance has that are serious and deleterious.

First, there is shrinkage of extracellular volume. It is usual for a mercurial diuresis to proceed to a point where the edema is delivered and then often the patient becomes refractory to the action of mercury. In a sense, this is a protective feature. Occasionally, as in the normal subjects who are practically thrown into salt depletion shock by 2 cc. of mercury, this diuretic agent can shrink extracellular volume below normal levels. Under those circumstances, serious renal impairment may supervene; not due to any poisoning of the tubular cell necessarily, but a consequence of a shrinkage of the extracellular fluid. The safest way to circumvent this complication is simply to stop giving mercury under circumstances where peripheral edema has been delivered, even though perhaps some rales may be in the chest.

Second, there is the low salt syndrome which has not been considered here in detail. This is a consequence of the fact that there is a loss of salt in excess of water, in a net sense, that water is retained, and this sometimes supervenes after a massive mercurial diuresis. Under such circumstances, it is correctable by the administration of hypertonic saline although admittedly patients who tend to develop this disturbance often will develop it even under circumstances where mercury is withdrawn and salt is administered. They tend to retain water despite anything one can do.

Third, potassium deficiency is apt to supervene, and it is important to recognize that one cause of failure to respond to the action of mercury as a diuretic is the supervention of a deficit of potassium.

Finally, a hypochloremic alkalosis may supervene, and this complication is in part perhaps a consequence of the loss of acid into cells and part a consequence, as I have tried to show, of the excretion of acid into the urine. These features of mercurial action are responsible in part, I think, for the so-called refractory state of mercury. And in any patient who fails to develop a mercurial diuresis, it is important to stop the administration of mercury and to evaluate the possible causes which may have led to his refractory condition. These causes, such as are known, have been partially listed here. The diagnosis (and detection) depends again on a high index of suspicion when refractoriness exists. If one thinks of potassium deficiency; if one thinks of alkalosis; then these disturbances can often be readily detected.

Some physicians have been accustomed to administer mercurial diuretics with one or two grams of potassium chloride for a day or two preceding and accompanying the injection of mercury. In other instances, this may not be necessary, of course, if the patient is eating well. In any event, the supervention of a refractory state should lead to a suspicion that this exists.

Should potassium deficiency exist, it is important to correct this. Four grams or so of potassium chloride can be given by mouth, diluted in some suitable vehicle, to supplement the oral intake of potassium in the diet. In addition, it is extremely important to administer ammonium chloride for reasons which are not clearly understood. Some cardiac patients do not correct their alkalosis with potassium alone, and under such circumstances the administration of an acid load may restore the refractory state. Three or four grams of ammonium chloride administered before and during the injection of mercury, that is, under two days preceding and under two days following the injection of mercury, may restore a refractory patient to a responsive state.

It should be emphasized that the mercurial refractory state is in part reversible. For this reason, it is important to evaluate the causes which lead to refractoriness. Prominent among these causes are potassium deficiency, metabolic alkalosis, salt depletion and hyponatremia. By a high index of suspicion, one can often detect the responsible agency and institute measures to correct this effect.

Vehicles and Volume, with Special Reference to Amino Acids and Sugar

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THE SUBJECT of parenteral feeding is a very important one, and the attendance at this meeting attests to the interest that you all have. I suppose, too, in a way it indicates the fact that there is still much we have to learn about the subject.

The United States is known in the rest of the world for many advances in medicine and in surgery, and one of the things that the visitors from overseas like to know about is our use of parenteral fluids. But some, when they come over, get a little confused. One of them, a surgeon from Calcutta, India, wrote me from New York City as follows:

"I came here to find out about parenteral fluids and I visited a professor in one of the big medical schools, asking him how he gives parenteral fluids. He referred me to the resident surgeon and I met the resident surgeon and I asked him the same question, and he referred me to the assistant resident surgeon. And the assistant resident surgeon said, "Well, the intern takes care of those matters," and he referred me to the intern. The intern was very busy and he said he'd like to have me talk to the fourth-year medical student and I talked to several of them and found that their answers were all different."

Now in discussing the subject of parenteral feeding, I first of all would like to pay a tribute to Dr. Carl Rice who, without really direct University support and, initially at least, at his own expense, has been able to add so much to our knowledge of a more complete type of parenteral feeding. He has done this in at least three ways, all based upon careful bedside clinical observation as well as meticulous scientific measurements. For instance, he has added much to the technique of administering parenteral fluids with the least discomfort and danger to the patient. Second, he has shown that the addition of alcohol is not only feasible and practical, but achieves much higher caloric intake than is possible without its use. Third, as an able physician and surgeon, he has demonstrated the beneficial bedside influence which followed the maintenance of a good nutritional intake despite the fact that the patient cannot take anything by mouth. The last objective, of course, is very obvious to anyone who believes firmly that starvation is not only not harmless but is actually harmful, but yet this simple statement needs continuous emphasis.

I would like to emphasize, too, that parenteral feeding never has and never will compete with oral feeding. It competes only with starvation as a temporary method really designed to enable the patient to eat sooner. While too much parenteral fluid tends to be used, the remedy is to use these fluids only when indicated in as small amounts as necessary but to make a mixture as complete as is feasible.

Now in participating in this symposium it was suggested that I discuss some of the newer observations which we have made, particularly with the use of invert sugar and fructose. I am afraid I won't be able to say much about invert sugar because we have made few observations. However, since it contains 50 per cent fructose and glucose, my own feeling is that it somehow has effects half way between those of glucose and fructose.

However, I would like to expand my discussion in order to bring to you some of the fundamental studies made by others as well as by ourselves on the important question of how many calories are needed in order to utilize amino acids. I say this particularly because statements have been made in the literature that there is no use infusing amino acids unless you give the patient full caloric intake. Now this argument may seem academic, but it has resulted, I am afraid, in a widespread belief that until intravenous fat is available to fill out the normal caloric intake, there is no use in adding amino acids to the usual glucose which still forms the basis for most

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programs of intravenous feeding. These statements, however, have completely ignored and overlooked the work of Dr. Rice and his co-workers, as well as observations by many others, showing that nitrogen balance can be achieved with less than a full caloric intake and that even if it is not achieved, the degree of negative nitrogen balance is considerably reduced when amino acids are added despite a suboptimal caloric intake. I would like to express this by saying that if you can't give a whole loaf as Dr. Rice does, at least a half a loaf is better than no loaf.

NUTRITIONAL COMPOSITION OF THE HUMAN ADULT MALE*

Total Weight	70.6 kg.
1. Water	48 kg.
2. Protein	10 kg.
3. Fat	9 kg.
4. Ash	3 kg.
5. Carbohydrate**	0.6 kg.
6. Plus Vitamins	

* Approximate values from Mitchell et al. J.B.C. 158:625, 1945
** From Everett, Medical Biochemistry (Hoeber) p. 306

Fig. 1. Nutritional composition of the human adult male. (From Elman, R.: Surgical Care. New York: Appleton-Century, Crofts, 1951)

Now first I would like to show in Figure 1 the nutritional composition of the human adult. Any time a patient does not receive an adequate amount of food of any kind he subsists only by using his own tissues. When you look at this table you can see that there is really only one dispensable food material and that is adipose tissue. There is actually very little carbohydrate in the body and although there is considerable protein, it is generally agreed that the use of protein for energy leads to functional impairment.

For example, liver and plasma protein, and hemoglobin are used from the very beginning of the time that the diet is deprived of protein. The problem of water I might pass over very simply. It has been discussed many times today and it is a simple one because under normal conditions 2 liters is adequate in the average-sized adult under standard conditions. I will not discuss it further except to say that as I see parenteral fluids used in various parts of the country, too much water is usually given. This has been well-emphasized in the comments of Dr. Ariel. Our objective in preparing any diet is to prevent or correct important deficiencies in the body and the one I am going to consider this afternoon is protein.

There are three degrees in this objective: first, to spare protein, *i.e.,* to maintain nitrogen balance; second, to build protein if there is a deficiency, *i.e.,* to achieve positive balance, and, third, to lose as little protein as possible by reducing the degree of negative nitrogen balance. All of these objectives are easy if you are able to give a full intake of say 4,000 calories and 200 grams of protein, but such an intake is impossible in at least two conditions about which I know. One is the severely malnourished patient unable to take so much food by mouth (at least in the beginning), because of severe gastrointestinal disturbances, and the other, during parenteral feeding. Now nitrogen balance within certain limits is a fairly good measure of protein loss or utilization, but other methods have also been used. In the following discussion I would like to point out that while an adequate caloric intake maintains the best type of positive nitrogen balance, there are many other factors which are also important in promoting the synthesis of protein tissue as measured by this or other means. One of them is the state of nutrition of the patient.

NITROGEN BALANCE IN 13 MALNOURISHED PATIENTS
ALIMENTED PARENTERALLY ON A SUBCALORIC DIET

2 liters daily of 5% amigen + 5% glucose
total caloric intake = 800 calories

	Body Weight in % of Normal	Daily Nitrogen Intake (Gms.)	Daily Nitrogen Output (Gms.)	Daily Nitrogen Balance (Gms.)
Average	65.4	12.6	7.1	+5.5
Range	55-78	12.6	4.5-11.1	+1.5-+8.1
Standard Deviation	6.8	0	2.1	2.0

Fig. 2. Nitrogen balance during malnutrition on a low caloric intake. (From *Surgery,* 34:874, 1953)

The first data are from J. B. Allison (Fed. Proc., 10:676, 1951). They represent nitrogen balance on an inadequate caloric intake containing protein. Dogs which are malnourished at the start go into positive nitrogen balance immediately whereas well-nourished dogs go into negative balance. They both tend to approach zero as time goes on. Nitrogen balance on a protein free intake in either case would, of course, result in a marked negative balance.

We have confirmed this phenomenon as shown in Figure 2 of our own data in a series of patients. Thirteen malnourished patients were on the subcaloric intake containing 12.6 grams of nitrogen as hydrolyzed protein. Note that all of these patients were in positive nitrogen balance to the extent of +5.5 with a range of 1.8 to 8.1.

To show the influence of malnutrition, we repeated the same experiment on three patients after they had become repleted. You can see (Fig. 3) that the positive nitrogen balance either became less or became negative. That doesn't mean that the amino acids were not being utilized, because if they had had glucose alone, they would have been in a —6 to —8 grams of negative nitrogen balance. The point is that the more malnourished the patient is, the more able he is to utilize amino acids despite the fact that we cannot give him a complete caloric intake.

COMPARATIVE NITROGEN BALANCE IN 3 MALNOURISHED PATIENTS ALIMENTED PARENTERALLY ON A SUBCALORIC DIET BEFORE AND AFTER PARTIAL REPLETION

Case	Body Weight in % of Normal	Average Daily Nitrogen Balance	
		Before Partial Repletion	After Partial Repletion
G. S.	75	+ 2.1	- 3.3
N. Y.	64	+ 1.5	- 2.3
N. B.	78	+ 7.7	+ 1.7

2 liters daily of 5% amigen + 5% glucose
total caloric intake = 800 calories

Fig. 3. The effect of repletion on nitrogen balance on a low caloric intake. (From *Surgery*, 34:874, 1953)

Figure 4 deals with a time factor on which I will spend just a few moments. It shows that if you give calories and protein, they must be given simultaneously. That is true whether you give them by mouth or parenterally. This was an experiment on a very malnourished patient with terminal cancer. We gave him one liter of 10 per cent glucose morning and afternoon; the negative nitrogen balance was marked. In the middle period we gave 5 per cent glucose in amino acids mixed together morning and afternoon and now nitrogen balance was positive. When we gave the same amount of material divided into 2 liters of 10 per cent amino acids and 10 per cent glucose, one in the morning and one in the afternoon, and you can see, even though the total amount of nutrition was the same, the patient was in negative nitrogen balance because the two were not given together. Interestingly enough, you will note that glycosuria was absent when the two were given together; when they were given separately or when glucose was given alone, the loss of glucose in the urine was considerable. So I don't think anybody now would give amino acids and glucose separately. But I find that physicians still give amino acids and glucose in the morning and glucose alone in the afternoon. Glucose given in the afternoon will not help the utilization of amino acids given in the morning.

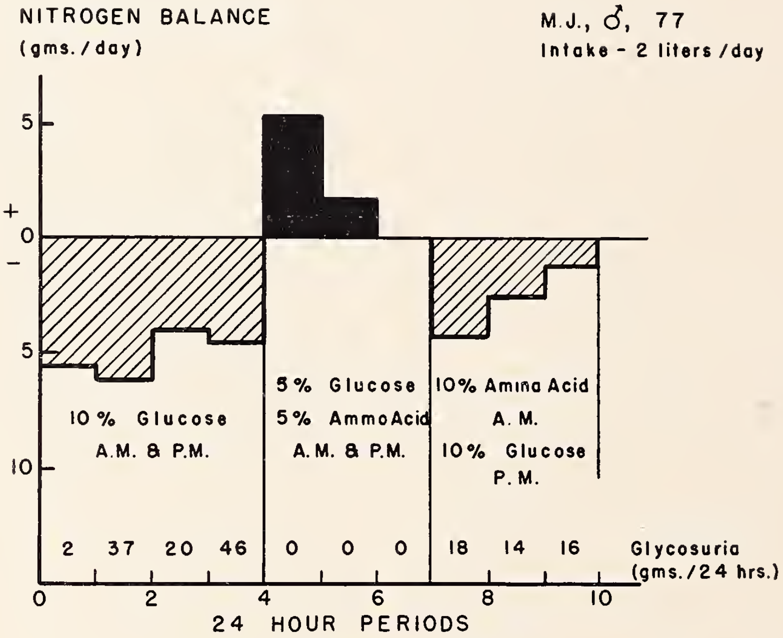


Fig. 4. A time factor in the administration of amino acids and glucose. (From Elman, R.: Surgical Care. Appleton-Century-Crofts, 1951.)

One really should then give the complete mixture both in the morning or in the afternoon, or preferably by continuous twenty-four-hour infusion with an indwelling catheter as described by Dr. Lowe this morning.

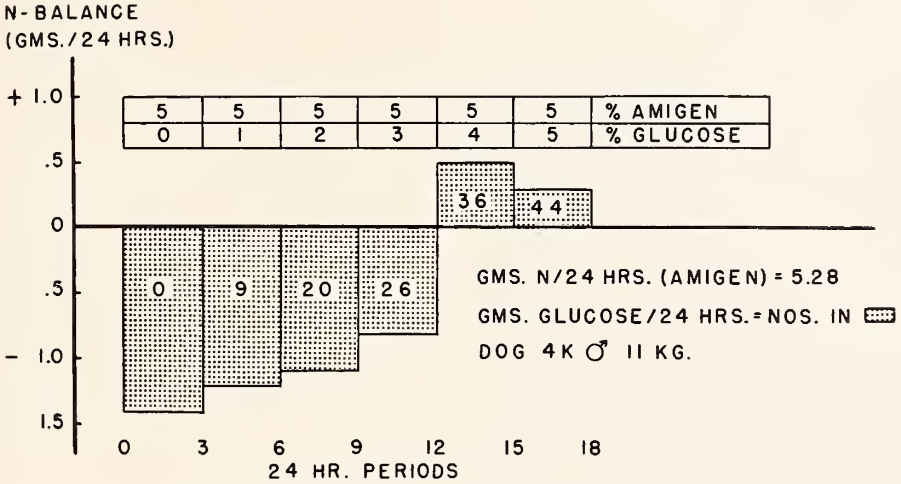
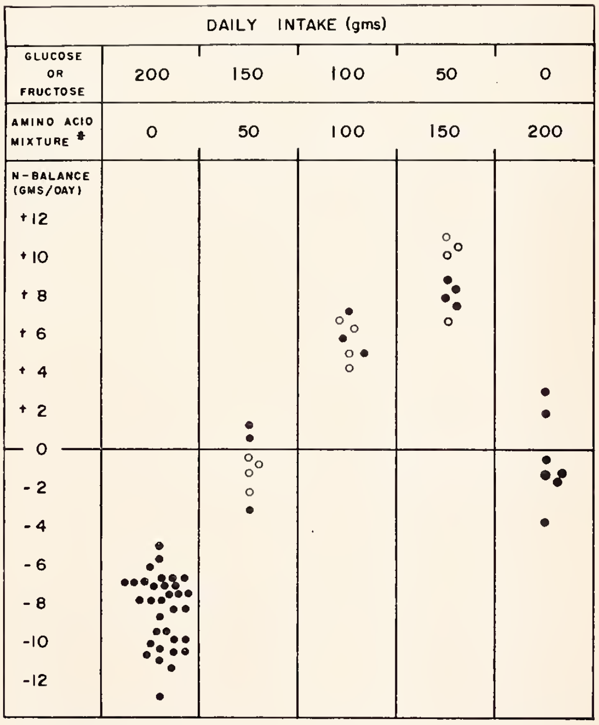


Fig. 5. The effect on nitrogen balance of adding increasing amounts of glucose to amino acids.

Of the several advantages of fructose over glucose, one is its better utilization in diabetics. Insulin apparently is not as necessary for the metabolism of fructose as it is for glucose. Another very interesting thing we have found was that there is no post-infusion hypoglycemia with fructose. In other words, if you give an infusion of glucose even to a normal individual or any other type of patient, the stimulus of the insulin production or whatever it may be that clears the blood of glucose, is so great that it continues after the infusion is ended. Sugar continues to be removed from the circulation and the blood level therefore drops to as low as 40 mg. per cent. With fructose this low post-infusion hypoglycemia does not occur.

Proportion of Amino Acids and Carbohydrate.—Our next studies deal with another factor in the utilization of amino acids, which is rather important and certainly interesting—"What proportion of amino acids and glucose shall one give?" In our first work we hit upon 5 per cent of each on the basis of an experiment which we never published, but I thought you would be interested in seeing. Nitrogen balances were measured in three-day periods in a dog (Fig. 5). During the first three days we gave 5 per cent amino acids and no glucose; nitrogen balance was negative. Then we added 1 per cent glucose and it became less, with 2 per cent it became still less, 3 per cent still less. When we got to 4 per cent positive balance followed. We didn't see any more improvement when the glucose was increased to 5 per cent so we went no further. But really this was a very simple yet incomplete experiment. We finally got around to doing a more adequate



* AMINO ACID MIXTURE OF HYDROLYZED PROTEIN (BRANDNAME-AMIGEN)

○ = FRUCTOSE USED INSTEAD OF GLUCOSE

Fig. 6. Nitrogen balance on varying proportions of carbohydrates and amino acids. (From *Transactions of the American Surgical Association,* Cleveland, Ohio (April 27) 1954)

experiment in humans. We made a systematic study of 29 patients by giving daily infusions containing 200 grams but with varying proportions of amino acids and glucose.

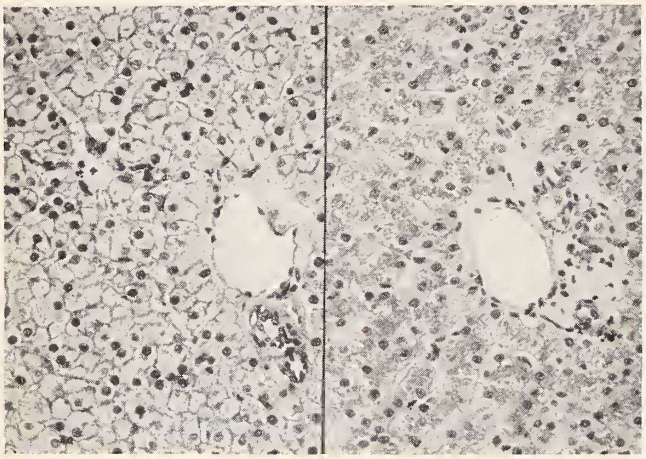


Fig. 7. The effect on the appearance of the liver and its protein content of adding amino acids to intravenous glucose. On the left is the animal receiving only glucose; on the right, the one receiving glucose plus amino acids as discussed in detail in the text. (From *Science,* 108:441, 1948.)

The results of this experiment are shown in Figure 6 in terms of nitrogen balance. When glucose or fructose was given alone, 200 grams with no amino acid mixture, all were in negative balance ranging from —12 grams up to about —5 or —6 grams per day which, of course, was what one would expect.

By contrast, when the intake contained no glucose or fructose at all, only amino acids, two of the patients were in positive nitrogen balance and even in the ones that were in negative nitrogen balance none were as much negative as the ones receiving glucose alone. So from that alone you would say if you are going to give a pure 10 per cent solution, in terms of nitrogen balance, you would be better off giving 10 per cent amino acids instead of 10 per cent glucose. Yet it is said that 10 per cent glucose is preferable to amino acids alone. These figures certainly would deny it. However, when you use a mixture, the figures are even more interesting. If you give 2½ per cent amino acids and 7½ per cent glucose, balance is around zero. If you give 5 per cent of each, it is between +4 and +8 grams per day, but the best nitrogen balance occurred when we gave 7½ per cent amino acids and 2½ per cent glucose or fructose. Actually the statistical difference in the latter two groups is not very great. But the data give confirmation of the fact that an equal mixture of amino acid and glucose really gives better nitrogen balance than 2½ per cent amino acids and 7½ per cent glucose.

Now I know that many of you perhaps have said that conclusions based on nitrogen balance are open to criticism. What we would like to know is whether these amino acids are actually utilized to build body tissue. A number of years ago we did an experiment which is very pertinent in this discussion. It is shown in Figure 7.

One group of dogs received amino acids and glucose and the other received glucose alone. Figure 7 shows the condition of the liver after two weeks of daily infusions. When glucose alone was given (photomicrograph on left), the liver is vacuolated, there is no stainable cytoplasm, the sinusoids are almost obliterated. This is what the pathologist actually calls hydroptic degeneration. This is a liver that contains a large amount of glycogen but has a very small content of protein, by analysis, about 2.8 per cent nitrogen. Next to it is a comparable section around a central vein in a similar experiment in which amino acids were substituted for part of the glucose. Note that the cytoplasm stains normally, the sinusoids are not crowded and actual chemical analysis showed that the nitrogen content was 3.3 rather than 2.8 grams per cent. In other words, here we have I think fairly good histological proof that amino acids are converted into liver protein and that glucose alone is not.

The next series of slides illustrate an important principle I would like to emphasize. The first data comes from the Rutgers group (J. Nut. 36:733, 1948) and represents percentage of nitrogen absorbed and utilized by the body. These are animal experiments. Note the different caloric intakes. The greatest amount of nitrogen absorbed, between 30 and 40 per cent, occurs when the dog got a high caloric intake. Note, however, that with a 50 per cent subcaloric intake, he was still utilizing 10 per cent of nitrogen whereas with no protein at all the figure would be zero.

The next two slides show similar findings from the work of Dr. Warren M. Cox. In this case protein synthesis was measured by actual analysis of nitrogen in the carcass of the animal as the caloric intake increased. Note that when glucose alone is given, there is no increase in the protein of the carcass, whereas on a 50 per cent subcaloric intake with protein there is a definite increase.

(Such an intake is analogous to the one we use clinically, namely 5 per cent amino acids, 5 per cent glucose, 2 liters a day plus vitamins which thus becomes an almost complete parenteral diet.) That a considerable amount of protein is laid down in the carcass illustrates again that you do not have to have full calories to get some good out of the protein that is added.

The next slide (not reproduced) shows a similar type of experiment—in this case based upon the weight gain of depleted animals given various mixtures of protein and carbohydrate. When there is no protein intake, of course they continue to lose weight. With full calories the weight gain is impressive, but note the gain on a 50 per cent subcaloric intake. They don't gain as much weight as they would on full calories, but they are certainly better off than with no protein at all.

Figure 8 illustrates this principle in a diagrammatic way. Each physician has the opportunity of giving a parenteral feeding mixture to his patient in any way he wishes and I find that by and large most patients receive 2 liters of 5 per cent glucose or 10 per cent glucose. Now if you give 5 per cent glucose, the average loss of nitrogen is about 6 grams per day. The patient will therefore be in 6 grams negative nitrogen balance. If you decide to give them more calories as 10 per cent glucose (you add 100 grams of glucose) you will find that he still puts out 6 grams of nitrogen and still has a —6 grams of nitrogen deficit. What you have done, then, with the added calories, obviously, is to spare tissue fat from which the body gets the rest of its energy. Since the body cannot exist without energy, our problem is to decide whether to give them exogenous energy or let them use the energy from their own body tissues. If they have to get energy from protein tissue, they lose protein, which is physiologically undesirable. But if they get calories from tissue fat, it is not physiologically undesirable; in fact, there are many of us in this room who would like to increase their physio-

HALF A LOAF IS BETTER THAN NONE.

Daily Intake	N. Output (grams)	N. Balance (grams)
1. Basic intake (2 L. 5% glucose)	6	- 6
2. Plus 100 grams glucose (2 L. 10% glucose)	6	- 6
3. Plus 100 grams amino acids (2 L. 5% glucose 5% amino acids)	15	- 2

Fig. 8. Decrease in negative nitrogen balance following the addition of amino acids. The table represents a theoretical calculation illustrating that added glucose does not necessarily influence negative nitrogen balance whereas added amino acids may.

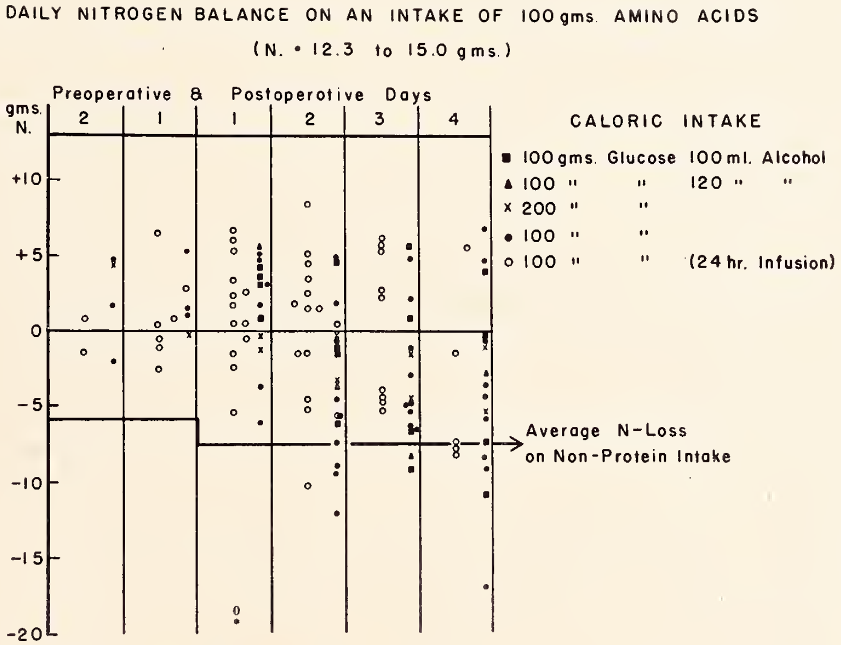


Fig. 9. Nitrogen balance before and after operation. The scattergraph represents specific data in a number of surgical patients undergoing major abdominal procedures and maintained on completely intravenous fluids for two preoperative and four postoperative days.

logical efficiency by getting rid of some of this adipose tissue.

What happens if, instead of adding 100 grams of extra glucose, we add 100 grams of amino acids. Now the output of nitrogen goes up which means that not all of the nitrogen given is utilized. But even under the worst circumstances you will get a reduction in the negative balance, say to —2 grams per day which is a saving of 4 grams of nitrogen. This doesn't make much difference in a well nourished patient and I won't quarrel about it. Moreover, it won't make any difference for one day or two days or three days, but the point is that these facts illustrate a principle. If protein is not added to the parenteral diet or if it is not added to the oral diet, the patient will be in more negative nitrogen balance than if you did add protein at any level of caloric intake. If you don't mind losing protein tissue, all right; but if you do, then you must add protein regardless of the method of administration and regardless of the state of nutrition. There is one exception. That is the well nourished patient during the first few days of the subcaloric intake.

Figure 9 is a scattergraph showing nitrogen balances before and after operation when amino acids were used. When we gave more calories, of course, they had better nitrogen balance as Dr. Rice and others have shown. I would like to point out that this line—this straight line across here—represents the average nitrogen loss which would have occurred if they had no protein intake. Actually, most, I would say 80 per cent, of the points were above that line and indeed many of these patients both before and after operation were in positive nitrogen balance. The worse nitrogen balance was this little spot down here (—23 grams a day) and I was a little disturbed about it until I looked up the data on the patient and found that this figure happened to occur in one who, unlike all the rest of them, did not get amino acids on that day, but simply got glucose alone.

In conclusion, I would like to say that my main theme is a very simple one—that food is necessary for life, for the sick just as it is for the healthy. By food I mean each of the known needed elements in the diet. Moreover, if you can't give all they need of any element, less than all is better than none at all.

I would also like to take this opportunity of expressing my thanks to the Hennepin County Medical Society and the Hennepin County Chapter of the American Academy of General Practice for this invitation to speak and, as a member of the audience, to all of the other speakers whose presentations I have so enjoyed hearing.

PEDIATRIC ELECTROLYTE PROBLEMS

(Continued from Page 20)

ing the first twenty-four hours, and 2,400 ml/M^2 thereafter. When patients are not dehydrated, use 1500 ml/M^2/25 hours. For initial therapy, do not give potassium-containing solutions. Use one third normal saline, 360 ml/M^2 administered in forty-five minutes. Most patients will void in forty-five minutes at this rate of delivery. If they do not void, change that rate of administration to 2,400 ml/M^2/24 hours, but do not use potassium-containing solutions until a patient voids.

I think that the data discussed summarize our present thinking about this problem. Much more could be said about details of management; however, in the last analysis each child has unique problems, demands compromises and improvisation, and the alert physician treats him as a law unto himself.

Discussion

Dr. Edmund Flink: It has occurred to me that some of you might have been confused by the use of the word "normal solution" because of the fact that physiological salt solution or isotonic salt solution is sometimes erroneously called normal salt solution. Isotonic salt solution is neither physiological nor normal. Dr. Helmer was speaking in terms of the chemical definition, namely, an equivalent weight of a particular salt per liter of solution. Both Dr. Helmer and Dr. Darrow have indicated the utility and the clarity of the designation of electrolytes in terms of milliequivalents. This is just as reasonable as recording your personal assets in terms of dollars and cents rather than in terms of quantities of goods. It is just as easy to learn the normal values of these in milliequivalents as it was to learn the heterogeneous units of measurement.

I wish to re-emphasize Dr. Lowe's remarks about keeping information on patients who have fluid and electrolyte problems. It is vitally important to obtain daily weights of these patients and to keep a careful record of all intake and output. Although it is often difficult to keep a record of the output, this is very necessary in order to handle a complicated problem.

To enlarge a little bit on the arithmetic of the problem, I wish to remind you that the molecular weight of sodium chloride is 58; thus 5.8 grams of sodium chloride contain 100 milliequivalents. Nine grams of sodium chloride, as is present in an isotonic solution, contains 155 milliequivalents. Potassium chloride has a molecular weight of 74. The ampule which contains 40 milliequivalents, therefore, will have approximately 3 grams of potassium chloride.

While I am talking about the quantities of potassium chloride to be administered, I wish to call your attention to Dr. Darrow's remarks about the requirements of potassium. This fits in very well with our observations on patients with alkalosis of vomiting. Potassium must be administered in a quantity sufficient to replace the deficiency. We have come to the conclusion that it is necessary to administer from 120 to 160 milliequivalents of potassium per day (8.4-12.0 grams KCl) in order to get anywhere in the correction of a metabolic alkalosis. This amount fits very well with Dr. Darrow's figure of 3 milliequivalents per kilogram of body weight per day. This amount of potassium chloride would be required to correct a potassium depletion in a patient with pyloric obstruction. Inadequate amounts of potassium will tide the patient over for a short period of time but will not get at the basic difficulty, as Dr. Lowe has so clearly shown in the patient who had an orthopedic operation and developed a serious potassium deficiency.

I certainly think, as Dr. Lowe has mentioned, that wherever possible it is wise to administer fluids throughout a twenty-four-hour period when the patient is seriously depleted. In addition, it is well to put a polyethylene tube in the vein and thereby have a continuous infusion. I also wish to warn you of the fact that one must not administer a concentrated solution of potassium intravenously by syringe, although the concentration in the body is not very great, for the concentration in the coronary circulation could be lethal. Rapid administration of solutions of potassium chloride, therefore, must be considered to be dangerous.

Two tables Dr. Lowe showed may have been a little confusing because the electrolyte concentrations were different from those of Dr. Helmer's. Dr. Helmer showed the concentration of electrolytes in serum, whereas Dr. Lowe's table showed the concentration in the extracellular water. It must be remembered that the extracellular water has no protein, and therefore the concentration of chloride and sodium is slightly higher than that which is found in the serum.

Question: Dr. Lowe, will not the alveolar wall of the lung sustain trauma from ether anesthesia on the first and second postoperative days and cause localization of fluid in the lungs resulting in pulmonary edema and atelectasis, if suction losses are replaced exactly? Apparently the idea is, can one give excessive amounts of sodium chloride in the first few days postoperatively? Dr. Lowe, will you open the discussion please?

Dr. Lowe: The normal intake of potassium for the adult is about 4 grams, 100 milliequivalents. That is equivalent to approximately 8 grams of potassium chloride. We do not use normal saline in treating patients postoperatively. Our experience with one-third and one-fifth normal saline has indicated that we are entirely safe in giving the amounts of fluid which I indicated on the slides in the immediate postoperative period. In following the serum concentration of sodium, chloride and potassium in these cases, we have not found levels to occur which we thought were deleterious to the patient.

Dr. Calcagno of Buffalo has studied the glomerular filtration rate and renal plasma flow in postoperative children and found these to be normal. Dr. Ariel has found the same to be true in adults and furthermore has failed to demonstrate a predictable change in the ability of the kidney to handle sodium and chloride postoperatively, although there is a tendency to retain sodium when normal saline is given.

Dr. Darrow, by his question, has given me a chance to mention some other things that I left out. I would say that the Number 1 rule for adequate surgical management of children is co-operation between pediatrician and surgeon. Nothing prejudices good surgery as seriously as a poorly prepared patient or one whose postoperative management is inadequate. We are willing to watch a patient with pyloric stenosis for as long as five or six days before operation in order to get him into shape and we have followed patients with an intussusception as long as eighteen to twenty hours in an effort to try to get them in better shape for surgery, knowing full well that a resection might be necessary because of the delay. If a seriously ill patient is not in fairly adequate electrolyte balance, he will not survive the operation.

QUESTION: Dr. Lowe, if one chooses the sodium content of colostrum as representing the normal sodium intake in an infant, would you give less than this normal amount after operation on the basis that you might anticipate some sodium retention associated with the stress response? Secondly, please discuss your two polyionic solutions. Are they available?

DR. LOWE: I would say that dilute solutions which have a concentration of electrolyte in the range of that of colostrum would be acceptable for replacement fluid for a newborn child who has required an operation. The volume used should be similar to that which might be taken by mouth in a similar child without operation. With the use of such solutions in probably over a hundred babies, we have never seen postoperative edema unless there were complications, such as sepsis, which of their own accord would cause edema. We have seen it in newborn babies, however, when we have used solutions which contain 40 to 60 milliequivalents per liter. It is my impression, and I think there is experimental evidence to substantiate this impression, that the newborn infant is remarkably capable of handling water and is very capable of controlling his electrolyte excretion.

DR. FLINK: It is always worthwhile to look at a problem in more than one way, and to express it in slightly different words. I wish to emphasize again the utility of expressing electrolyte concentration in terms of milliequivalents because of the clarity and the understanding that is permitted.

Dr. Helmer's illustration of the use of chloride and bicarbonate (CO_2) concentrations to estimate approximately the total electrolyte concentration in the serum is just one example. It would be impossible to get any idea of that if one reported CO_2 in terms of volumes per cent and chloride in terms of milligrams per cent. Some laboratories report chlorides as milligrams of chloride ions and others report it as milligrams of sodium chloride. It is also useful to think of the administration of electrolytes in terms of milliequivalents rather than grams. At the present time, the polyionic solutions that are available commercially have on their labels the number of milliequivalents that are available in each 100 milliliters. Potassium chloride ampuls designed to be added to parenteral fluids are labeled as 20 or 40 milliequivalents.

QUESTION: Dr. Helmer, is calcium completely dissociated in the blood, and if so, is it therefore legitimate to express it in terms of milliequivalents per liter?

DR. HELMER: About one-half of the calcium is bound to protein. Since protein can be expressed in milliequivalents on the basis of its cation binding capacity, I think it is legitimate to express calcium concentration in milliequivalents.

QUESTION: Dr. Helmer, will you review the laboratory steps which are necessary to evaluate the sodium content of the serum when a photoelectrometer is not available? You gave a formula and we would like to have it explained to us.

DR. HELMER: Add the value of the CO_2 combining power (bicarbonate concentration), the normal of which is 27 mqs., to the value of the chloride concentration, the normal of which is 103 mqs. From these you obtain a value of 130 mes. per liter. Then if you subtract that, 130, from the 142, the normal sodium value, you will obtain a difference of 12. On that basis, if you add the chloride value plus the bicarbonate value and add 12, you can estimate the sodium concentration.

QUESTION: Dr. Helmer, if the sum of the CO_2 and chloride is above the normal, can this be interpreted as a significant hypernatremia?

DR. HELMER: Yes, you can expect to have a hypernatremia.

QUESTION: Dr. Helmer, what happens to the value of the sum of CO_2 and chlorides in metabolic acidosis or uremia?

DR. HELMER: In metabolic acidosis or uremia this calculation will not be valid. In metabolic acidosis there is an accumulation of organic acids and in uremia there is an accumulation of phosphates or sulfates along with elevation of the other anions.

QUESTION: Dr. Darrow, in a patient with prolonged loss of hydrochloric acid as in a case of an obstructed duodenal ulcer, isn't it probable that an actual chloride deficiency supervenes?

DR. DARROW: I'm sure that it does sometimes, but ordinarily I think most of our chronic alkaloses are intermittent in the sense of a loss of chloride if water does get into the body, and I think that if large amounts of chloride are actually lost from the body in the sense of an absolute deficit, you will find that the concentrations of sodium in the serum are low. I know that theoretically it can occur without that, but I think practically it will be usually with a low sodium concentration.

'DR. FLINK: I wish to emphasize again what Dr. Seldin has mentioned, namely, the importance of recognizing the possibility of an electrolyte imbalance when a patient fails to respond to a mercurial diuretic. When this occurs, one should investigate the electrolyte status in an effort to determine the reasons for that failure to respond.

Question: Dr. Seldin, can you comment on the prognosis of patients with a low salt syndrome which is refractory to therapy?

Dr. Seldin: Patients with a low salt syndrome are presumably patients who have a low concentration of sodium in the serum and who are edematous at the same time. I think one can distinguish two types of a low salt syndrome. In one type, the low salt syndrome develops chronically without any acute onset. A cardiac patient will retain water in excess of salt, thereby leading to a severe hyponatremia. Under those circumstances, one can correct the hyponatremia with hypertonic saline but almost invariably water will again be retained, diluting the sodium down to its previous values. The correction of chronic dilution hyponatremia in congestive heart failure often eventuates in no clinical improvement.

In contrast to this, there is the problem of acute dilution hyponatremia as in the cardiac who also begins to retain excessive amounts of water. Under these circumstances, the administration of hypertonic saline may be life-saving. These states in heart failure should be contrasted with similar states in nephrosis or cirrhosis. Chronic dilution hyponatremia as low as 120 milliequivalents may be tolerated by the cirrhotic patient with surprisingly little ill effect. There is, however, a form of acute dilution hyponatremia in cirrhosis which demands immediate attention. This develops under circumstances where a cirrhotic develops a paracentesis while he is on a salt-free diet. Here water is retained, but no salt is available. Severe hyponatremia supervenes, transudation occurs into the cirrhotic sac and as a consequence of this the blood volume shrinks, peripheral vascular collapse occurs and a lethal outcome may follow. Cirrhotics who are tapped completely of their ascitic fluid are in a precarious state when they are on a salt-free diet and should they become drowsy, disoriented, and so forth, hyponatremia should be suspected and hypertonic saline administered.

Dr. Darrow has raised the question of the concentration of the replacement solution. This should be either 3 or 5 per cent saline in the cirrhotic. Where congestive heart failure is not a problem, 5 per cent saline can be given in amounts which can be estimated from the sum of the CO_2 and the chloride or directly from the concentration of serum sodium. In a cardiac patient where the problem of a massive intravenous load is a formidable one, one should give the solutions slowly in divided doses.

Question: Dr. Seldin, what is the effect of potassium deficiency on the action of digitalis in the treatment of congestive heart failure?

Dr. Seldin: Potassium seems linked in some manner with the action of digitalis in a reciprocal fashion. In order to potentiate a response to digitalis, potassium deficiency must be corrected.

Fluid and Electrolyte Requirements of the Surgical Patient as Influenced by the Post-Traumatic Response

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THE TITLE of this paper is particularly timely because the body's response to trauma—the so-called alarm reaction or reaction to stress—now dominates surgical thinking, especially in regard to defining it and to devising repair solutions to counteract the deleterious effects of this reaction. No one can deny the importance of this reaction but, as so frequently occurs in surgical thinking, certain styles prevail for a given period and, as with ladies' hats, change frequently causing yesterday's style to be shunned and discarded. Today emphasis is upon reaction to stress and the literature abounds with attempts to explain all postoperative phenomena as stress reactions. Throughout the history of surgery, each era has had a particular feature which it has emphasized as being the all-important one for the safe conduct of a patient through an operation. At first when the surgeon was a mere tissue technician, no special efforts were made as regards preoperative and postoperative care. Were it not for the results of infection, it is highly probable that many of these surgical patients would have survived because their homeostatic mechanisms, developed as a result of evolutionary improvements, would have functioned to help convalescence and restore health.

With the emphasis placed on the importance of saline solutions by O'Shaughnessy in 1831, an era prevailed in which saline administration was promiscuously practiced. Coller and Maddock were great exponents of the saline regime for surgical patients and contributed greatly to a better understanding of crystalloid balance in patients undergoing operation.

Over-enthusiastic administration of saline solution by many surgeons was contributory to further lines of thought and practice in treating the patient postoperatively. In 1936, George Hoyt Whipple focused attention upon the importance of protein metabolism and the serious consequences of hypoproteinemia. One of these consequences, edema, was markedly aggravated by the administration of saline. This brought attention to the necessity of supplying proteins to the surgical patient, and some of the greatest advances in this field were made by Elman who carefully studied nitrogen balance in the surgical patient and presented methods of supplying proteins, including the intravenous administration of amino acid mixtures.

The dangers of saline infusions caused certain surgeons to avoid completely any crystalloids during the immediate postoperative period. Among these was Wangensteen, who freely administered ionic-free water postoperatively. This afforded me an opportunity to study the effects of water administered to the surgical patient. A rather high incidence of water intoxication resulted which was not appreciated as such, and a number of postoperative patients suffering convulsions and coma were, therefore, diagnosed as having cerebral vascular accidents.

The recent brilliant studies of Darrow emphasized the important role of the potassium ion in the maintenance of a normal electrolyte structure in patients, demonstrated its defect in infants suffering from diarrhea, and showed that the administration of this ion would at times be lifesaving. He and his associates further developed an explanation of the intermediary metabolic reactions between the various ionic compartments of the organism. It was but a short step to apply these data to certain complications suffered by postoperative patients which resist the ordinary methods of treatment. In this respect I must admit with a certain amount of embarrassment that in a paper written by myself in conjunction with Doctors Abels, Pack, and Rhoads, we described a form of hypochloremia which resisted sodium chloride administration. Under the influence of the "protein age," we attributed the fundamental defect to hypoproteinemia. In all probability we were describing hypokalemia, for the clinical and chemical studies we described in that paper are very similar to those which are the result of potassium deficiency.

With this introduction, I should like to describe some of the reactions which occur in the patient subjected to an operation in an attempt to identify the nature of the ensuing developments.

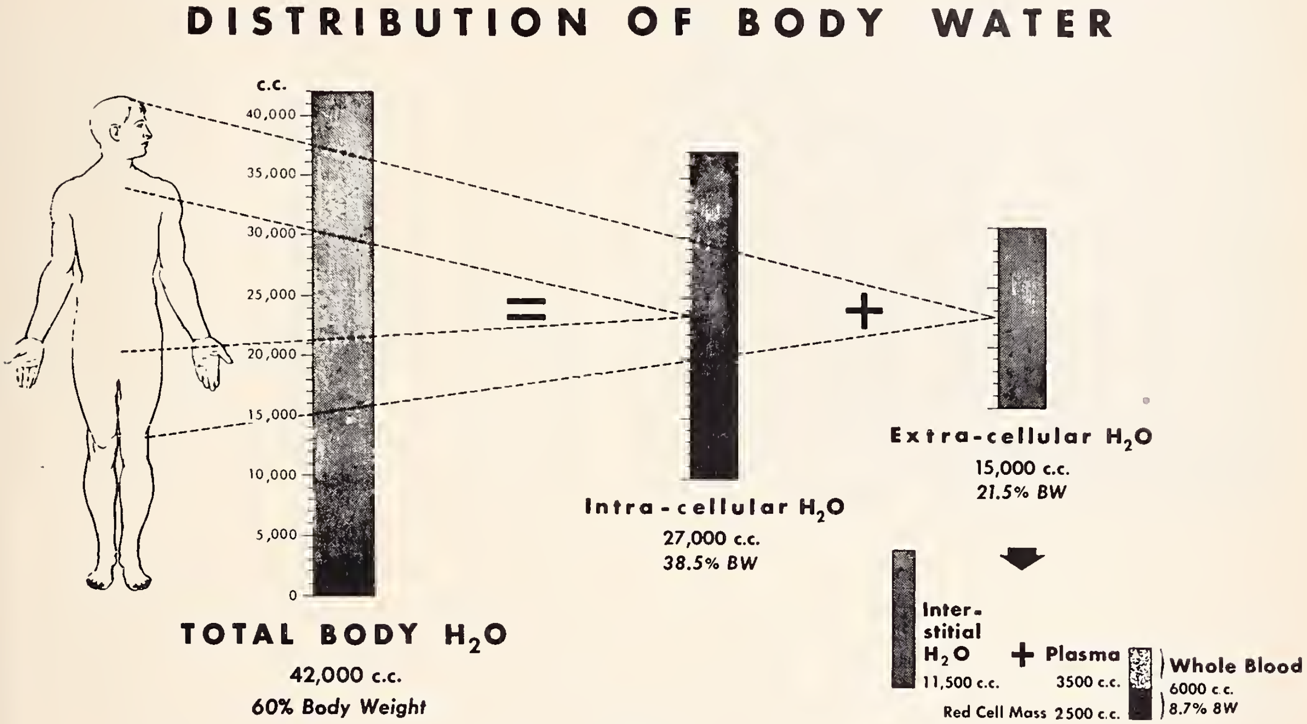

Fig. 1.

Numerous other factors compete for high priority as regards their importance to the postsurgical patient. Among these are vitamins, blood, plasma and plasma expanders, early ambulation, and others. Doctor Carl Rice and his associates deserve credit for focusing attention upon the caloric requirements of the surgical patient, indicating the substandard caloric content of most postoperative hospital regimes, and devising methods of supplying calories, including the use of intravenous alcohol solutions combined with other calorie-containing ingredients.

In reviewing the physiologic aspects of surgical convalescence, it becomes apparent that no single feature is omni-important. Rather, each contributes a necessary link in the maintenance of homeostasis, and a deficiency of any one substance produces a break in the chain of metabolic reactions, the clinical sequelae of which can be disastrous. Nevertheless, during each of the ages certain of the preceding factors dominated surgical thought. Each was considered the all-important item by certain physicians, and heated arguments were not infrequent at medical meetings.

TABLE I.
CLASSIFICATION OF EDEMA AND DEHYDRATION

Bound Water (Osmotically Inactive)	Osmotically Active Water	
	Extracellular Compartment	Intracellular Compartment
1. After injection of certain hormones (estrogens).	1. Edema	Normal
2. Three and one-half grams of water deposited with each gram of protein.	2. Edema	Dehydration
3. Two ± grams of water deposited with each gram of glycogen. (Fat is deposited dry.)	3. Edema	Edema
	4. Dehydration	Normal
	5. Dehydration	Dehydration
	6. Dehydration	Edema
	7. Normal	Edema
	8. Normal	Dehydration

In order to interpret the effects of surgery upon the patient we must first appraise the condition of the patient as he comes to operation. It is not infrequent for the patient to be in a deplorable state of fluid and electrolyte balance and to come to the operating table in that particular state. Then, as a result of the operation, another insult is added to the metabolic trauma that the patient has suffered and the sum total is interpreted as a result of surgery. A few of these alterations found in patients coming to surgery are expressed in the first few figures.

Figure 1 is an expression of the distribution of water, indicating that water constitutes about 60 per cent of the body weight divided into intracellular and extracellular components. We must think not only of loss or gain of water but also of the distribution of the water within the body.

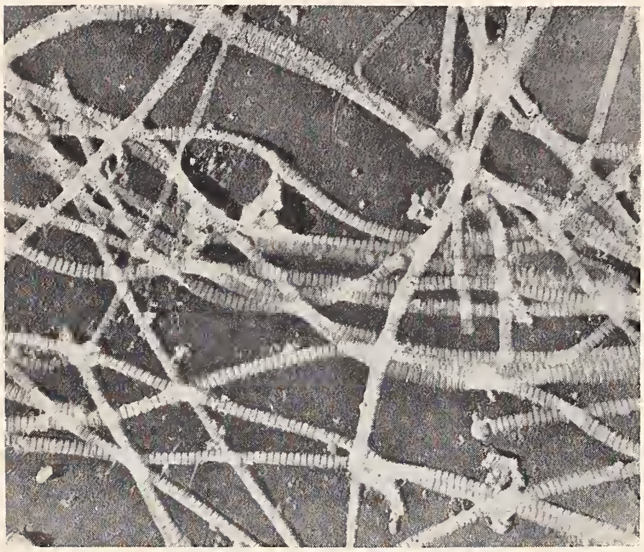

Fig. 2.

Table I demonstrates the various kinds of abnormalities of water distribution that can be produced. The administration of so-called normal saline causes extracellular edema; giving hypertonic saline produces extracellular edema with an intracellular dehydration. We observed extracellular dehydration and intracellular edema in our patients who developed hypochloremia as a result of withdrawing gastric juice. If saline solution is given rapidly to a patient in hypochloremic alkalosis before he has a chance to adjust his mechanism, one of the worst types of edema, namely, intracellular and extracellular edema, results.

We have become accustomed to using the term "extracellular edema" or the "extracellular space" very loosely. Figure 2 shows the electron microscopic appearance of the extracellular space. We see the various fibers and the matrix of the space. We must recognize that this is not a space. It is not a simple space in which fluid goes in and out, but it is an organ which has its special form, structure, and function. Thus we must visualize the body as consisting of two types of circulation, (1) the blood circulation as we know it, and (2) the circulation of the extracellular space. We must also think of the response of the patient to operation in terms of (a) response to the external environment, i.e., abnormal losses and abnormal gains of the various waters and electrolytes; and (b) internal balance, i.e., the shifts which occur between the cell itself and this extracellular space. When alterations occur in this particular space, they have direct reflections upon the cell. We must always bear in mind that any substance given to treat an intracellular defect must first be given into this extracellular space. Ingested or injected fluids and electrolytes must be deposited here, exert their influence here. This influence is then transmitted to the cell.

Table II shows some of the changes produced by water and salt loss preoperatively which, if unrecognized, will result in operating upon a metabolically imbalanced patient. In patients who lose gastric fluid, there is a marked difference between those with ulcers who have a hyperacidity of their gastric contents and those with gastric cancer who usually have a gastric anacidity. The fluid withdrawn from these patients in a period of three to five days of constant gastric lavage contained 629 mEq. of chloride in patients with hyperacidity and 369 mEq. of chloride in the patients with anacidity. Sodium loss was less in the first group in comparison to sodium loss in patients with anacidity. There was a significant amount of potassium withdrawn. Thus, in patients with hyperacidity the gastric aspirate contained a marked excess of chloride in relation to sodium, whereas in patients with gastric anacidity this excess was much less and the loss was one of almost a total salt, sodium and chloride.

The indicated changes are reflected in the

TABLE II. THE CONTENT OF CHLORIDE, SODIUM, AND POTASSIUM OF THE FLUID WITHDRAWN BY GASTRIC ASPIRATION IN PATIENTS WITH GASTRIC CARCINOMA

Case No. Patient	Quantity of Gastric Fluid Aspirated, Liters	No. of Days of Withdrawal	Chloride		Sodium		Potassium	
			mEq./l.	Total Amount Aspirated, mEq.	mEq./l.	Total Amount Aspirated, mEq.	mEq./l.	Total Amount Aspirated, mEq.
1—L.G.	3.5	6	85	297	80	280	10	35
2—C.V.	5.4	4	96	518	92	487	12	65
3—B.B.	4.5	5	76	342	74	333	15	67
4—J.C.	5.3	5	62	329	59	323	17	90
5—M.L.	5.5	4	65	357	60	330	8	44

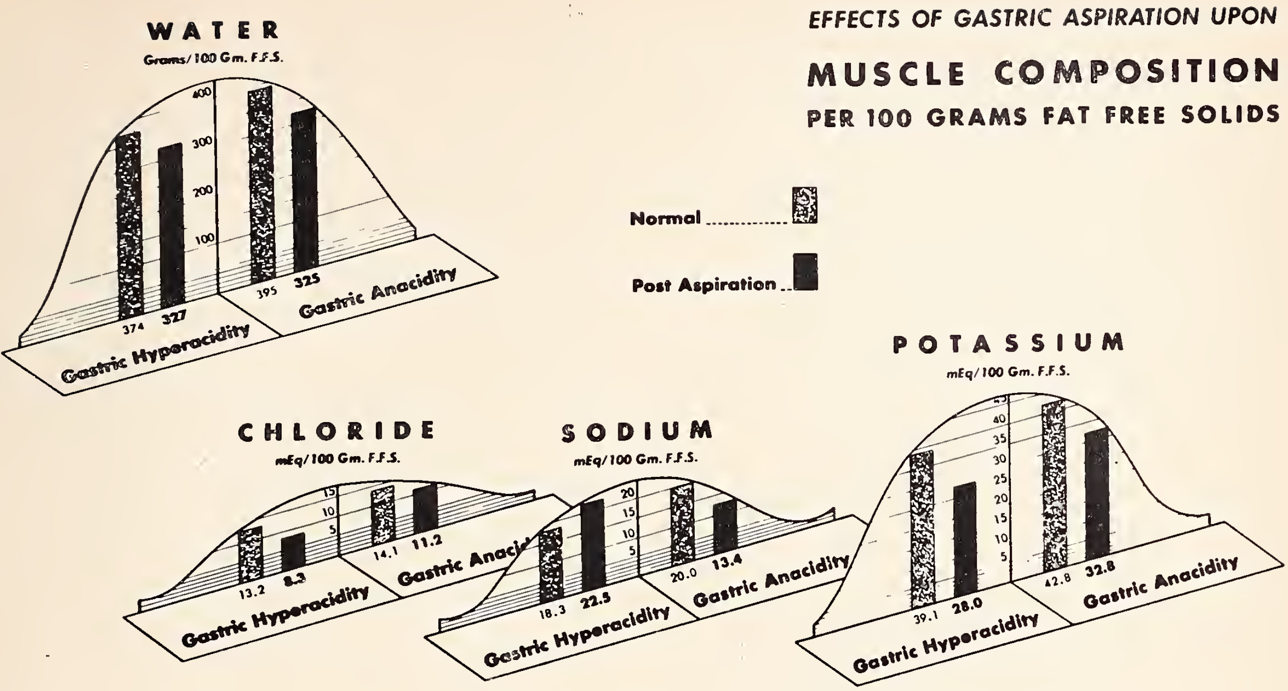
EFFECTS OF GASTRIC ASPIRATION UPON
MUSCLE COMPOSITION
PER 100 GRAMS FAT FREE SOLIDS
Normal
Post Aspiration
WATER
Grams/100 Gm. F.F.S.
400
300
200
100
374
327
395
325
Gastric Hyperacidity
Gastric Anacidity
CHLORIDE
mEq/100 Gm. F.F.S.
15
10
5
13.2
8.3
14.1
11.2
Gastric Hyperacidity
Gastric Anacidity
SODIUM
mEq/100 Gm. F.F.S.
20
15
10
5
18.3
22.5
20.0
13.4
Gastric Hyperacidity
Gastric Anacidity
POTASSIUM
mEq/100 Gm. F.F.S.
45
40
35
30
25
20
15
10
5
39.1
28.0
42.8
32.8
Gastric Hyperacidity
Gastric Anacidity

Fig. 3.

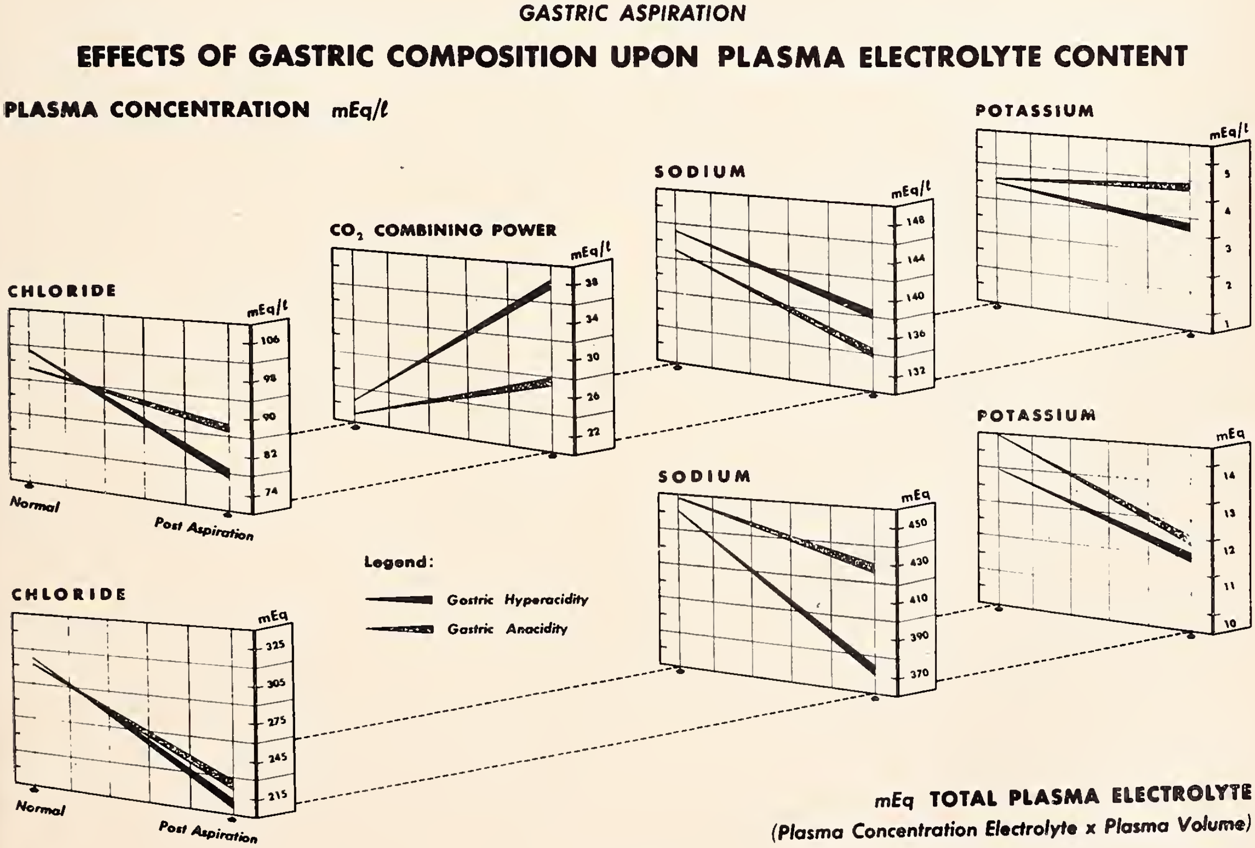
GASTRIC ASPIRATION
EFFECTS OF GASTRIC COMPOSITION UPON PLASMA ELECTROLYTE CONTENT
PLASMA CONCENTRATION mEq/l
CHLORIDE
mEq/l
106
98
90
82
74
Normal
Post Aspiration
CO_2 COMBINING POWER
mEq/l
38
34
30
26
22
SODIUM
mEq/l
148
144
140
136
132
POTASSIUM
mEq/l
5
4
3
2
1
Legend:
Gastric Hyperacidity
Gastric Anacidity
CHLORIDE
mEq
325
305
275
245
215
Normal
Post Aspiration
SODIUM
mEq
450
430
410
390
370
POTASSIUM
mEq
14
13
12
11
10
mEq TOTAL PLASMA ELECTROLYTE
(Plasma Concentration Electrolyte x Plasma Volume)

Fig. 4.

TABLE III. AVERAGE ALTERATIONS IN SERUM CONCENTRATION OF HEMOGLOBIN, PROTEIN, UREA NITROGEN AND CERTAIN ELECTROLYTES DURING ABDOMINAL OPERATIONS

	Pre-operative	Post-operative	Next A. M.
Hemoglobin (Gm. %)	14.9	14.4	14.2
Hematocrit (%)	46.3	47.6	45.3
Total Serum Protein (Gm. %)	7.0	6.6	9.9
Blood Urea Nitrogen (mg. %)	9.9	17.3	11.9
Serum Electrolytes (mEq./l.):			
Chloride	97.1	101.9	90.9
CO_2 Combining Power	25.6	22.5	25.3
Sodium	142.2	148.2	139.0
Potassium	4.3	4.2	4.7

muscle biopsies (Fig. 3). I am grateful to Dr. Cooke of Dr. Darrow's laboratory for assistance in these calculations. We see here the marked increase in the intracellular sodium in patients with the gastric hyperacidity, and in patients with anacidity a loss of sodium.

Figure 4 shows the changes in the plasma concentrations of these two groups of patients. In the group of patients with gastric hyperacidity the chlorides dropped markedly, the CO_2 combining power rose, sodium fell, and potassium decreased slightly. Those with gastric anacidity had similar changes, but not as marked, and I emphasize this because the loss of the total salt may produce metabolic alterations that will not be reflected in plasma concentrations. In short, the loss of chloride over sodium will produce a typical hypochloremic alkalosis; a loss of the total salt will result in a marked diminution of all water and salt and the patient may not show the typical hypochloremic alkalosis we see when chloride alone is lost. Therefore, great care must be exercised in judging the plasma values of patients with gastric cancer.

The effects of the operation upon the metabolic status of the patient will dictate the quantities and types of repair solutions to be given during the operation and during the postoperative period. The study of metabolic alterations which occur during intraabdominal operations is hindered by variables which are difficult to control. Such factors as anesthesia, the trauma of operation *per se*, and the body's reaction to the operation, each contribute a part in the overall picture of the metabolic response to the alterations incident to intraabdominal operative intervention. In addition, the administration of blood and other fluids during the operation further complicates the picture.

The following data were obtained by measuring metabolic responses to intraabdominal operations performed during warm summer months. Only relatively small amounts of glucose in distilled water were administered to the patients during the operative procedures. The values referred to as operative losses indicate amounts lost during the actual operation; those listed as postoperative losses indicate losses from the completion of the operation until the following morning. (Tables III and IV).

TABLE IV. ALTERATIONS IN SODIUM BALANCE OF PATIENTS DURING ABDOMINAL OPERATIONS

	Pre-operative	Post-operative	Balance	Next A.M.	Balance
Plasma Sodium, mEq./l.	142	148	+6	139	−3
Total Circulating Sodium, mEq.	440	385	−55	431	−9
Total Interstitial Sodium, mEq.	1944	2055	+111	2129	+75
	During Operation				
Urinary Excretion of Sodium, mEq.	28	66			

There are two phases of the body's reaction to an operative procedure. The first consists of the balance between the individual and his external environment, and the second the alterations of balance between the cells and their environment (the extracellular compartment).

Balance Between the Individual and His External Environment.—This consists of a negative balance of water, certain electrolytes, and blood. The quantity of water lost varies in different individuals and in different surroundings, depending upon the conditions prevailing at the time of operation. Thus, in the warm summer months under heavy operative drapes, the individual loses significant quantities of water (two to four liters).

Contrary to the belief of many, urine output continues, sometimes in significant amounts, in certain patients during an operative procedure and during the immediate postoperative period. Although a number of individuals will manifest evi-

TABLE V. EFFECTS OF ABDOMINAL OPERATION UPON TOTAL BODY WATER, PLASMA VOLUME, RED CELL MASS AND INTERSTITIAL VOLUME
Average Values

	Pre-operative	Post-operative	Next A. M.
Total Body Water—D_2O (liters)	36.3	35.1	36.0
Plasma Volume (liters)	3.1	2.6	3.1
Red Cell Mass (liters)	2.6	2.3	2.6
Interstitial Volume (liters)	13.5	13.7	15.1

dence of water retention during the immediate postoperative period, this situation does not prevail for all patients, and certain individuals will excrete large quantities of urine. The reason why some patients will continue to excrete urine and others will not remains enigmatic.

It has been demonstrated that there is no significant kidney damage during operation as determined by measurements of renal clearance. The average renal plasma flow, renal blood flow, glomerular filtration and tubular absorption were not significantly affected during the abdominal operative procedures. These findings are summarized in Figure 5. If the operative trauma is great, and if anoxia and hypotension occur, renal damage may occur and contribute to oliguria. However, in most surgical patients it must be assumed that some extrarenal mechanism functions to cause water retention in those patients who receive adequate quantities of water, but who retain that water postoperatively. It would be erroneous to assume that water is retained by all individuals and therefore that water should be withheld postoperatively. It would be equally erroneous to feel that for those patients who manifest evidence of water retention, a greater quantity of urine can be produced by the liberal administration of water. The amount of water given to an individual postoperatively should depend upon the amount lost during the operation and during the postoperative period, which varies from individual to individual.

It is difficult to measure the quantity of water lost during the operation. A rough correlation exists when three methods of estimating total fluid loss are compared. Total body water as measured by deuterium dilution decreased by an average of 1.2 liters in a series of patients subjected to intraabdominal operations. Body weight decreased an average of 0.9 Kg. A small portion of this loss was accounted for by the resected specimen in some instances. A positive water balance of 105 ml. was noted during the operation when fluid intake and urine output were measured. An unmeasured quantity of water was lost by perspiration. Since each of these measurements has an inherent factor of error, it may be assumed as a practical guide that approximately one liter of body water is lost during the three to six hours for intraabdominal operative procedures without undue trauma. There was an average loss of 300 ml. of red cell mass (Table V), which is equivalent to approximately 650 ml. of whole blood lost, or a plasma loss of 350 ml. (computed on the basis of the preoperative hematocrit level).

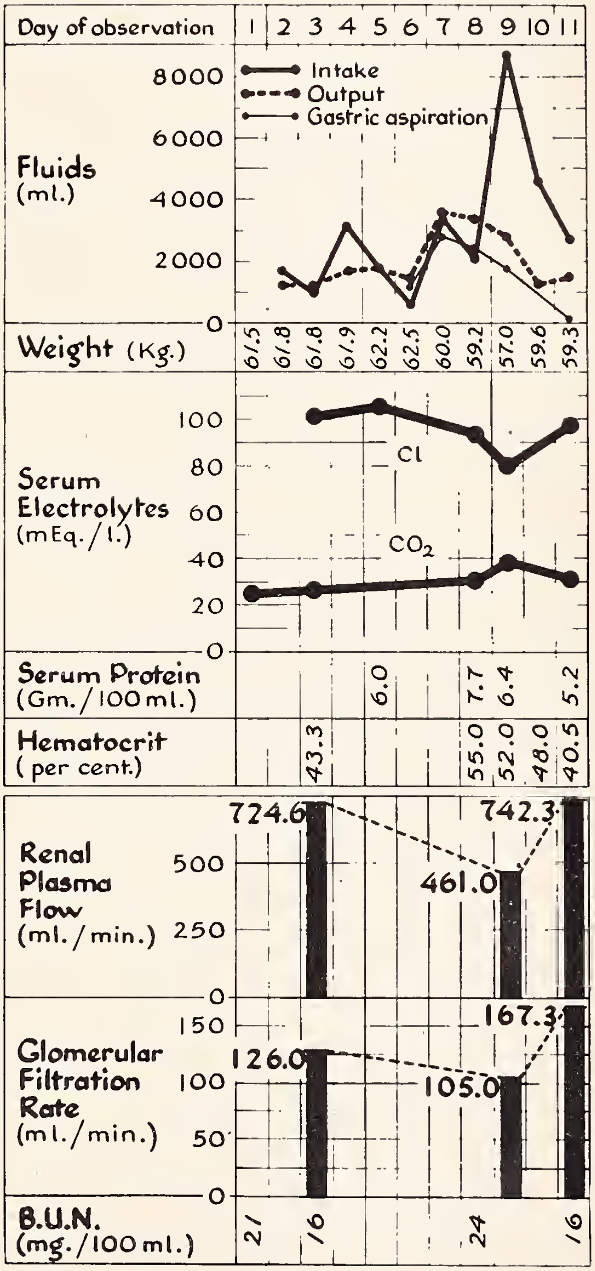

Fig. 5.

TABLE VI. AVERAGE URINARY EXCRETION OF ELECTROLYTES DURING ABDOMINAL OPERATIONS

	During Operation	Postoperative
Chloride (mEq.)	32	67
Sodium (mEq.)	28	66
Potassium (mEq.)	16	67
Nitrogen (grams)	1.4	8.7
K:N Ratio	13:1	8:1

Interstitial Fluid. Such wide fluctuations occur in the postoperative values for the interstitial spaces as measured by the sodium thiocyanate method that it must be concluded that the method is unreliable. It must thus be assumed that an unknown quantity of water was lost from the interstitial spaces and the remainder from within the cells. The urinary potassium lends support to this supposition as does the decrease in muscle water noted postoperatively on analysis of muscle tissue.

Electrolyte Loss During Operation.—A rather close correlation exists between chloride and sodium excretion, 32 mEq. of chloride and 28 mEq. of sodium having been excreted during the intra-abdominal operations. During this same period 16 mEq. of potassium was excreted. The postoperative urinary electrolytes of 67 mEq. of chloride, 66 mEq. of sodium and 67 mEq. of potassium undoubtedly represent excretory manifestations of trauma, although a slight amount could conceivably be accounted for by the 800 ml. of whole blood administered during this period (Table VI).

There does not appear to be any correlation between the total volume of urine and the amount of electrolytes excreted. It would thus appear that the excretion of electrolytes is not dependent upon the same factors which influence the water excretory mechanism. Thus one individual may produce a great deal of ion-poor urine while another may produce a small quantity of urine heavily laden with electrolytes.

Nitrogen Excretion.—Protein loss was manifested by excretion of 1.4 gm. of nitrogen during the operation which is equivalent, if all this nitrogen represents protoplasmic breakdown, to 46 gm. of tissue (Lusk's coefficient: 1 gm. N = 33 gm. of whole tissue), and during the day postoperatively 8.7 gm. of nitrogen was excreted, representing 287 gm. of tissue destroyed if all the urinary nitrogen is the end product of tissue catabolism.

All of the above described alterations reflect changes between the individual and his external environment. They probably represent the effects of the operative trauma upon the organism as well as a reaction of the organism to the stress— the alarm state. In addition to changes between the patient and his environment (alterations in balance), internal changes also occur, i. e., alterations between the cell and its environment—the extracellular space. These internal alterations are indicated by the marked change in the total circulating plasma protein with an egress of the protein from the plasma during the operative procedure and by changes in permeability of the cell membrane to chloride and sodium in certain instances.

This shift of electrolytes and proteins in and out of the plasma in answer to metabolic demands demonstrates some of the reactions to the alarm state. Whether any replacement therapy is indicated for these shifts is not known. The data further demonstrates the fallacy of utilizing serum concentration of various metabolites exclusively as criteria for replacement therapy.

Thus a patient may lose a small amount of sodium and chloride in the urine during an operation and the immediate postoperative period, but as the result of metabolic stress there may be a shift of the sodium and chloride out of the plasma into the interstitial spaces, inducing a decrease in the plasma content of these electrolytes. If one is guided exclusively by concentration values of the serum, one would be tempted to administer quantities of sodium and chloride sufficient to bring the value to normal. Although the exact method of coping with this abnormal shift is not known, it is believed that the excess administration of saline solution would be detrimental in such instances.

The adrenal response to the stress of a surgical operation will favor the excretion of potassium and nitrogen and retention of sodium and chloride. If an excessive response occurs which is further complicated by administration of sodium chloride solutions, abnormal retention of these electrolytes may become clinically dangerous. Moore and Ball have ascertained that an initial response to an operation (six day period) results in the normal release of 875 mg. cortisone, 10 mg. percorten and 300 cc. escharten.

Other factors which must be considered in the overall appraisal of the effects of surgical intervention upon body metabolism are the quantities of energy used during an operative procedure and manifested by utilization of body carbohydrate and fat. A previous study in which the effects of operation upon liver fat and glycogen were measured in liver biopsy specimens taken preoperatively and postoperatively demonstrated an average loss of 2 gm. per cent hepatic glycogen. This amounted to a decrease of 45 per cent of the total hepatic glycogen, and in some cases the hepatic glycogen decreased 50 and 75 per cent below the preoperative level. There was also an increase of hepatic lipids during an intraabdominal operation, sometimes to rather large amounts.

Therapeutic Applications

The demonstration of certain defects which develop as a result of intraabdominal operations with a decrement of water, blood, electrolytes and nitrogen, some of which have been quantitatively measured for this study, indicates the quantity and type of repair solutions which should be administered during the operative and postoperative periods.

Water.—The observation that an average loss of one liter of fluid occurred during operation suggests that this quantity of fluid administered during an intraabdominal operative procedure should be well tolerated. The fluid loss, however, can be much greater, especially under surgical sheets during hot summer months, during which time the patients' temperatures have been noted to rise to 102 to 104 degrees (Fahrenheit). In such instances larger quantities of fluid are indicated.

It is impossible to generalize concerning the quantity of water to be administered. Certain surgical patients will develop an abnormal retention of administered water with an associated oliguria. In such patients water must be given cautiously; otherwise symptoms of water intoxication will develop. Because of this phenomenon, some surgeons have cautioned against fluid administration to patients during the operative and immediate postoperative period under the mistaken belief that all surgical patients retain water. This concept is in error. The ability of the surgical patient to excrete water or to retain it abnormally is an individual characteristic and must be evaluated for each patient.

Blood.—In a group of patients investigated by the author, average blood loss as determined by alterations in plasma volume and red cell mass, amounted to 650 ml. of which 300 ml. were red cell mass and 350 ml. plasma. These alterations are greater than the blood loss determined by extracorporeal methods (weighing sponges, etc.). The maintenance of an adequate blood volume is one of the greatest essentials of surgical practice, and it is felt that a minimum of 500 ml. of whole blood would be indicated in these patients during the operative seance.

There is a prevalent tendency to administer too much blood during operation. The most frequent guide is the patient's blood pressure, and any drop regardless of cause is considered a signal for continued blood administration. This is harmful and we have seen patients develop surgical polycythemia from such a practice. There is no good method of determining blood volume serially during an operation, hence caution must be exercised against the prevalent policy of promiscuous administration of blood. A mild vasoconstrictor to combat vasodilation, the use of plasma expanders, and gentle handling of tissue will reduce the necessity for administration of large quantities of blood.

Electrolyte Replacement.—Sodium, chloride and potassium are excreted during the operative procedure (32, 28 and 16 mEq. respectively) and during the immediate postoperative period (an average of 67 mEq. for each ion). Although the loss of this quantity of electrolytes should in itself not hamper convalescence, it is felt, nevertheless, that every effort should be made to maintain the preoperative balance, and replacement therapy should begin at the time of operation. If this is done electrolytes lost during the stress of the operation will be replenished. A dilute, polyionic solution such as described by Fox, et al., which contains 140 mEq./l of sodium, 103 mEq./l of chloride, 10 mEq./l of potassium, 55 mEq./l of bicarbonate, and small amounts of calcium and magnesium, would seem to be indicated. The administration of an electrolyte solution would also serve to buffer against the convulsions and coma of water intoxication which would occur if an excess of ionic-free fluid were administered and retained. One liter of this solution administered postoperatively would replenish the sodium and chloride losses as measured in this study. The potassium decrement would not be significant, provided potassium replacement were instituted within four to five days after operation.

Fear of administering electrolytes to patients during the immediate postoperative period grips certain surgeons as the result of a preceding era when large quantities of 0.9 per cent sodium chloride were routinely administered to all patients postoperatively. Intelligent administration of electrolytes to replace those lost during operation is a more physiologic practice than fear and avoidance of these ions during and immediately after operation. Our study indicates that an estimate of electrolytes lost is a far safer indication for replacement therapy than serum concentrations of the electrolytes. In fact, abnormally low serum levels may ensue because of an abnormal distribution of the available electrolytes, and one should never strive heroically to raise a subnormal serum value to a so-called normal level by administration of massive quantities of repair solutions.

Nitrogen.—The loss of 1.4 gm. of nitrogen during the operative procedure and 8.7 gm. during the immediate postoperative period is not considered clinically serious, and it is felt that it does not warrant any immediate replacement therapy. A healthy convalescence with oral intake of protein foodstuffs will adequately replenish this loss. The plasma protein concentrations cannot be used as an index for protein replacement therapy because a large quantity of protein is mobilized from the plasma in answer to the metabolic demands of stress. In addition, the hydrodynamics of the plasma will mask the true plasma protein content.

Internal Balance.—A number of changes occur during an operation which reflect differences in distribution of certain metabolites. Such changes as the emigration of protein from the plasma or the abnormal ingress of sodium and chloride into the cells in certain patients reflect little understood responses of the organism to trauma, and nothing is known as to what therapeutic attempt is indicated if any.

Energy.—The expenditure of energy with the utilization of carbohydrates and fats during an operation demands caloric replacement. The body can withstand this short period of negative energy balance provided it does not progress over a prolonged period.

Postoperative Care

The patient immediately after an operation must be regarded as a traumatized individual in whom three sets of responses are occurring: (1) those responses which are the direct result of the injury to the organism, (2) normal responses by the organism in reaction to the traumatizing actions, and (3) abnormal responses due to the inability of the individual to cope with the trauma either because the trauma was too extensive and/or the individual's response mechanism is defective. An example of severe trauma would be massive hemorrhage and of the defective response mechanism Addison's disease. It is the surgeon's responsibility to define each type of reaction, measure those abnormalities of balance, and to correct those which demand correction. He must always stand humble, recognizing the fact that his replacement efforts are at best a poor substitute for nature's reaction to stress.

When a well-nourished, otherwise healthy individual has been subjected to a moderately traumatizing surgical procedure the immediate reactions will consist of (1) a mild fever (for which antibiotics are not indicated), probably the result of foreign protein reaction, (2) a brief period of starvation, and (3) an increased catabolism which is a result of the operation. The losses as described under *Effects of Operation* are not great and careful attention must be given to balance, avoiding the over-administration of any intravenous solution, encouraging early ambulation and early active respiratory exchange. Certain changes which have been unduly stressed by some investigators may occur. These include oliguria or anuria, an excessive excretion of potassium, retention of sodium or chloride, or excessive nitrogen excretion. In the author's experience these abnormalities may occur occasionally but represent the exceptional individual variations. When urine excretion is suppressed, except in the occasional instance, it has been shown to be transient, lasting during the day of operation and the first postoperative day. It is never harmful and it is considered advisable not to attempt to encourage urine excretion. It has been found that most complications in this type of patient are the results of over-enthusiastic mal-administration of various intravenous solutions. Administration of too much five per cent dextrose in distilled water to patients with transient urinary suppression will produce hypotonicity of body fluids with resultant convulsions, saline over-administration will produce edema, and over-administration of blood will embarrass the cardio-respiratory system. It is considered better to err on the side of under-administration because the body can cope with such situations.

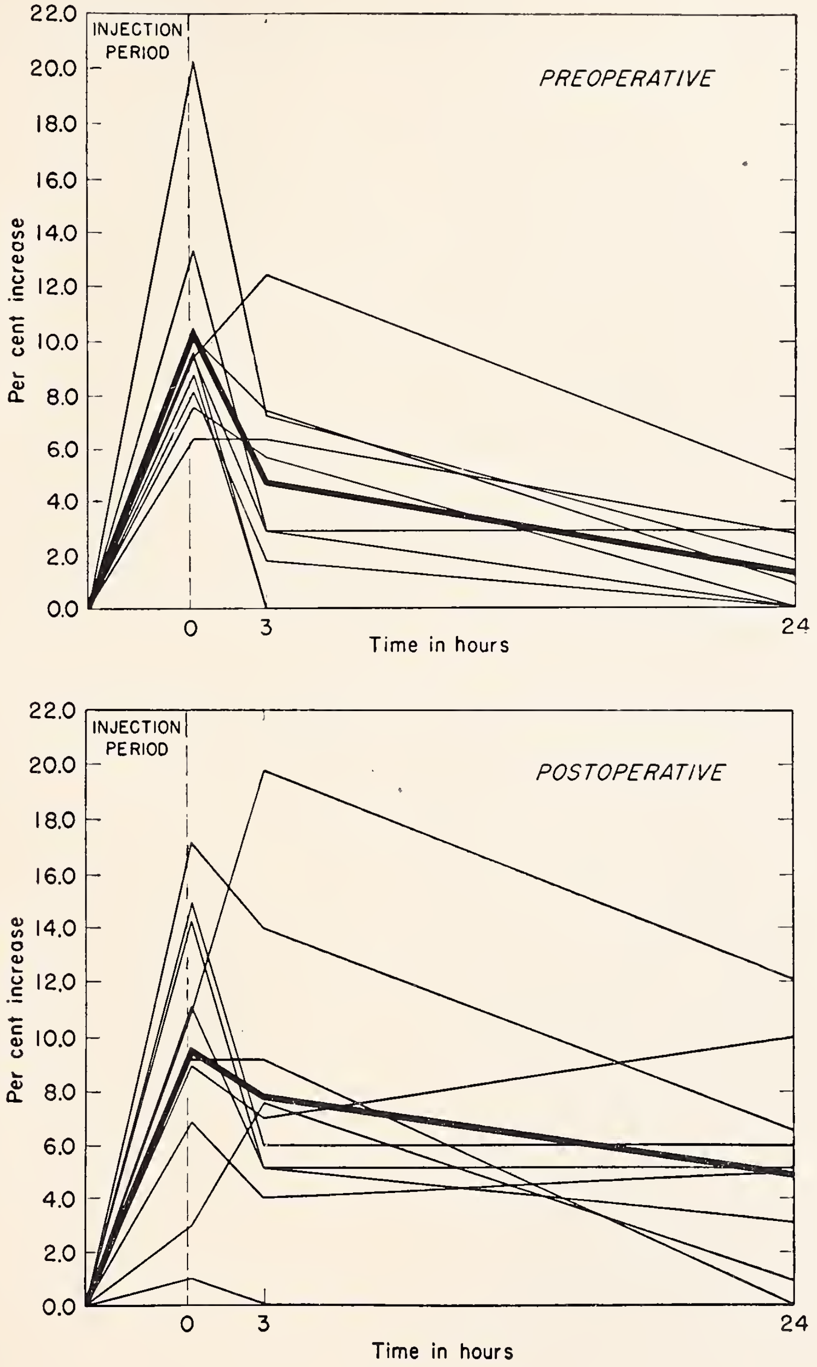

Fig. 6.

Studies of the adrenal response in such patients will demonstrate first a diminished amount of circulating eosinophils and an increase in the urinary 17-ketosteroids. It is stressed that the organism can tolerate the trauma and post-traumatic period by its well integrated adaptive mechanisms. A hands-off policy is indicated for the first two to four days replacing only estimated or calculated losses. If the patient has not recuperated to the point of ambulation and oral ingestion of foods by this time, a program of intervention must be charted. It is essential during this period to respect the body's ability to withstand the trauma and to watch carefully for the development of any complications. Blood volume must be maintained, careful attention must be given to hemoglobin, hematocrit, and protein levels realizing that these measurements are only measurements of concentration. Continued blood loss will become evident by diminishing hemoglobin and hematocrit; marked water loss due to unmeasured perspiration will be reflected by an increasing plasma protein concentration. It may be mentioned that in the patient receiving antibiotics the orthodox symptoms and signs of abscess formation and peritonitis will frequently be absent. The only indices of their presence will be a continued low grade fever beyond the first two postoperative days, a reluctance to ambulate, and hypoproteinemia due to loss of plasma proteins into the abscess. These complications should of course be treated by appropriate measures.

Fluids and crystalloids given to patients during the immediate postoperative period will be handled differently in many instances from that given to an unoperated person. It has been demonstrated by this author that sodium chloride solutions will be spilled in the interstitial space in greater quantity and for a longer period than the same salt load given preoperatively. It has been shown that normally proteins are mobilized into the plasma subsequent to a salt load in an effort to maintain normal osmotic relationships. In the postoperative patient this mechanism is faulty and the saline solution diffuses into the interstitial spaces. Figure 6 shows the plasma chloride concentration subsequent to a load of 27 grams of sodium chloride. The difference between the preoperative curve from that which occurred postoperatively is manifest. If hypertension or anoxia supervenes during the operation, the cell membranes become disrupted and sodium and chloride enter the cell, producing further derangements with a lowering of the plasma concentration levels of these ions.

The handling of a water load by patients postoperatively depends upon their renal excretory capacity. In a group of twenty patients given an intravenous water load of three and one-half liters during the immediate postoperative period, eight maintained good urinary excretory ability and twelve retained the administered water. In the later group, a few presented convulsions and other signs of acute water intoxication with dilution of the various plasma crystalloids. The cause for the difference of response remains enigmatic but indicates that each patient must be individualized regarding his personal reaction to a water load postoperatively.

The Complicated Response

If the surgical trauma has been extensive, if blood loss has been great and replacement inadequate or delayed, or if anoxia has occurred during the operation, the surgeon's task of conducting such patients through the immediate postoperative period will be great. Some patients who have been subjected to shocking traumatization will rebound with gratifying agility. In such instances a conservative therapeutic policy is indicated. In most such patients, however, the postoperative period is punctuated by various complications. Distressing sequelae consist of prolonged oliguria or anuria with retention of nitrogen (which produces uremia), sodium and chloride retention, and the liberation of potassium from the cells into the plasma which exerts deleterious effects especially upon the heart. The exact mechanism of this complication is not known, but it lasts usually from three to seven days after which there is a profound diuresis with urinary excretion of electrolytes. The therapy for this complication consists of a hands-off policy during the period of oliguria, supplying only such water as is lost. A mild anemia is best left untreated because some of these patients are sensitized and will develop unexplained hemolytic reactions to administered blood. The surgeon must be constantly on the alert in such instances to treat boldly by administering large quantities of electrolytes and water when diuresis ensues.

The response of the malnourished, weakened patient differs from that of the robust individual in many respects. It may vary from utter failure to institute a reaction to stress such as seen in the Addisonian patient to an effective alarm reaction but with an extreme proclivity to development of postoperative complications. In such patients a preoperative test of adrenal function as advocated by Thorn may be advisable and if the test indicates hypoadrenalism and an operation is nevertheless mandatory, the cautious administration of cortisone during the surgical period may be indicated and a postoperative regime instituted similar to that utilized for patients after adrenalectomy.

In those mal-nourished individuals who had been prepared for operation by an intensive preoperative regime with the aid of surgical measures (gastrostomy, enterostomy for feeding, etc.) it has been my experience that the nutritional status of such patients is sometimes more apparent than real and after operation the "bottom drops out," with the development of hypoproteinemia, poor wound healing, increased susceptibility to infection, delay in development of peristalsis with resultant gastrointestinal distention, etc. In such patients it is advisable to push nutrients by every means possible, to avoid over-hydration and excessive electrolyte administration, to keep alert for the development of any complication, e.g., hypochloremia, and to correct it instantly before the chain reaction of metabolic disturbances develops.

It is essential to measure all losses from the body (vomiting, Wangensteen suction, diarrhea, exudates and transudates) and on the basis of their content of electrolytes and proteins to replace these, volume for volume. The surgeon can have a better index for replacement therapy by measuring the content and composition of body losses than measurements of chemical composition of the blood.

Acknowledgment

Permission has been given for publication of the following tables and figures: Table I, Ariel, Freier and Kremen,[9] courtesy of W. B. Saunders Company; Figure 2, Courtesy Dr. Jerome Gross; Table II, Figures 3 and 4, Ariel,[5] by permission of *Surgery, Gynecology and Obstetrics;* Figure 5, Ariel and Miller, reproduced from *Surgery* through courtesy of C. V. Mosby Company; and, Figure 6, Ariel and Kremen,[6] reproduced from *Annals of Surgery,* courtesy of J. B. Lippincott Company.

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Blood Transfusions and Plasma Volume Expanders

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THE ENORMOUS value of blood transfusions in anemia and in states where blood volume is depleted is well known. The indications and hazards in the use of blood are also too well known to bear repetition. The serious question may be rightfully asked, how well do we adhere to the known indications?

In an attempt to obtain an answer to this question, the transfusions given in a small general hospital in 1953 were reviewed. During this year, 284 patients received transfusions. The total number of transfusions given was 1,244. Of this group, the records of 75 patients, who received a total of 256 transfusions, were carefully reviewed.

TABLE I. INDICATION FOR TRANSFUSION.

Hemorrhage—surgery		57
No shock	49	
Shock	8	
Given postoperatively	9	
Hemorrhage—gastrointestinal		14
No shock	12	
Shock	2	
Hemorrhage—traumatic		5
No shock	2	
Shock	3	
Hemorrhage—nasal (shock)		1
Anemia		5
Hypoalbuminemia		1

TABLE II. EVALUATION OF TRANSFUSION.

Indication	*Number Not Indicated*
Hemorrhage—surgery	
Given postoperatively	5
Given at surgery	4
Anemia	2
Hypoalbuminemia	1
Total	12 (16%)

The indications for which the blood was given are shown in Table I. It will be noted that most of the blood was given for hemorrhage. Usually this was hemorrhage occurring during surgery. Of the patients who received blood during surgery, only eight were in shock at the time. In Table II, all of the transfusions were evaluated as to whether they were indicated. It will be seen that there were nine patients who received blood for hemorrhage incident to surgery in which the reviewers thought that the blood was not indicated. Four of this group received blood during surgery. None of the patients were in shock. They had minimal blood loss, and postoperatively were found to have a polycythemia which we presume was induced by the blood transfusion. The patients given blood postoperatively had hemoglobins, at the time they received the blood, of 14.6, 14.3, 13.9, 12.9, and 11.9 grams. The two individuals who received blood for the treatment of anemia where it was considered not indicated had hemoglobins of 12.5 and 12.7 grams. One patient received blood for the chemical finding of hypoalbuminemia, although he had no symptoms related to the lowered protein content of the blood. Sixteen per cent of the total patients receiving transfusions then received blood when not indicated. In addition to this, there were another thirty patients in which blood was given at surgery where there was no shock; where the surgeon described minimal blood loss; and where it was impossible to ascertain whether or not the blood was indicated or not.

Of the total group of patients, forty-four received one or two transfusions and 31 received three or more. Thirty-eight of the patients receiving only one or two transfusions received their blood during surgery.

The reaction rate in this group of seventy-five patients was 5.3 per cent. There were two pyrogenic reactions, one allergic reaction, and one

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The studies of blood transfusions were done in collaboration with Jack Riggall, M.D., and Edward Schneider, M.D. The dextran studies in collaboration with Ben Heller, M.D., and Richard Ebert, M.D.

TABLE III. FOUR GROUPS OF PLASMA VOLUME EXPANDERS.

1. Blood derivatives
    - Albumin
    - Globin
    - Plasma
2. Modified Proteins
    - Gelatin
    - Oxypolygelatin
    - Isinglass
3. Plastics
    - Methyl Cellulose
    - PVP
4. Polymerized Carbohydrates
    - Acacia
    - Pectin
    - Dextran

hemolytic reaction. It is worthy of mention that the patient who had the only serious transfusion reaction, the hemolytic transfusion reaction, was one of the individuals who received blood postoperatively where it was considered that blood was not indicated. The man had a fracture of the left tibia and fibula and right femur in an automobile accident. Surgery was performed on November 19. On December 4, his hemoglobin was 11.9 grams. Despite the only moderate reduction in the hemoglobin, he was given a transfusion on December 10. After 200 cc. of blood he developed back pains, vomiting, chills, cyanosis, and tachycardia. Investigation proved that it was a hemolytic transfusion reaction. The error occurred when the pilot tubes on two bottles of blood were interchanged.

From this review, then, it is apparent that we do not always follow the indications for the transfusion of blood as closely as we should.

There are certain difficulties with the use of blood that make it advisable to have other substances available. Blood, of course, is difficult to procure and in addition to that it is difficult to store. In the event of a disaster, it would be impossible to insure an adequate supply of blood. The onset of the Korean War intensified interest in the use of other substances. These should be termed plasma volume expanders and not substitutes for blood. In Table III is a list of the various plasma volume expanders. They are divided into four main groups: blood derivatives, modified proteins, plastics, and polymerized carbohydrates. Many of these substances have received extensive clinical or experimental trial. Albumin, globin, and plasma, like blood, are difficult to procure. In addition, the incidence of hepatitis following plasma transfusion is appreciable. In the patients receiving plasma transfusions during the Korean War, the incidence of hepatitis was approximately 20 per cent. It is possible that further development of the storage of plasma at room temperature will obviate the high incidence of hepatitis following such transfusions. However, this remains to be proved and until definite proof is available, it would seem advisable to limit the use of plasma transfusions. The various blood derivatives derived from animal blood have been tried. The objective to these substances is the high incidences of reaction. Of the modified proteins, oxypolygelatin would seem to have the greatest promise. This particular substance is a modification of gelatin and does not gel at room temperature. Isinglass has been used and found to have a high incidence of reaction. Both of the plastic substances mentioned, methylcellulose and polyvinylpyrrolidine, are stored in the tissues. Methylcellulose has been found to produce renal injury. Of all of the substances listed in this table, the two that would appear to have the greatest promise are dextran, which is a polymerized carbohydrate, and oxypolygelatin. Since more information is available regarding dextran, the remainder of our discussion will be limited to the use of dextran.

Dextran is a water soluble glucose polymer related to glycogen. It is produced by the action of Leuconostoc mesenteroides on sucrose. The raw dextran has a very high molecular weight and is highly toxic for animals. By acid hydrolysis this may be degradated into material of suitable molecular weight. This material has a low incidence of reactions. It has certain other attributes which indicate that it would be worthwhile to consider as a plasma volume expander. It produces no alterations in the pulse, blood pressure, or electrocardiogram. It has no toxic effects on the kidney or liver. It does not interfere with blood typing, cross-matching, or Rh determinations. It has a low incidence of reactions. When the reactions occur, they are chiefly of an allergic type with itching, urticaria, joint pain, headaches, vasomotor collapse, and respiratory distress. The incidence of reactions has varied greatly. In Sweden, where dextran was first used, the incidence of reactions was approximately 1 per cent. In England the reaction rate was 2 per cent. At Brooke Army General Hospital utilizing the Swedish dextran, a reaction rate of 40 per cent was found.[3] More recently, with the American products, the reaction rate has been extremely low and is probably a fraction of a per cent.

If any substance is to be used by the practicing physician, it is important that he know not only what it will do, but also what happens to the material. In order to more accurately ascertain this, certain experiments were performed.[2] In the first part of the experiment, one liter of 6 per cent dextran was given to normal, healthy subjects. The plasma volume, hematocrit, protein concentration, dextran concentration, and urinary dextran excretion were determined at one, six, and twenty-four hours. In the second portion of the experiment, young, healthy, male subjects were bled one liter and then were given a liter of dextran. Similar determinations were performed.

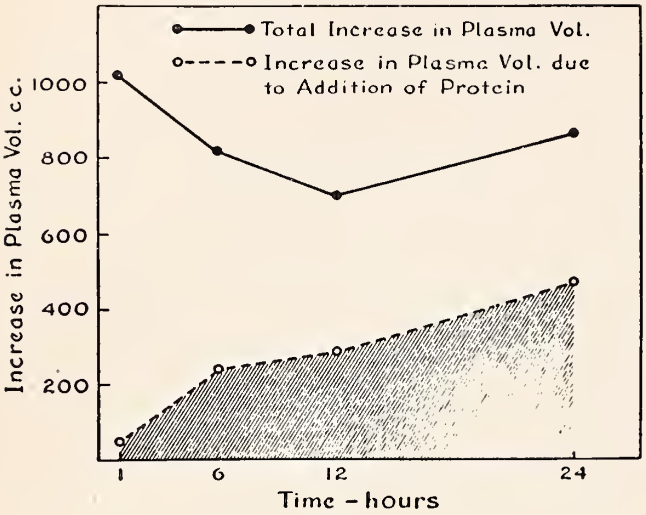

Fig. 1

The dextran concentration in both groups was slightly over 1 gram at 1 hour and then decreased slowly. At the end of forty-eight hours, the dextran was still detectable in appreciable quantities in the blood. In those subjects who were not bled, dextran was excreted rapidly; 23 per cent in 1 hour and a total of 48 per cent in twenty-four hours. In the subjects who were bled, similar results were obtained. Twenty-six per cent was excreted in one hour, and in twenty-four hours 60 per cent had been excreted. Of the dextran administered in those subjects who were bled, 40 grams was still circulating at the end of one hour and 25 grams at the end of six hours. A small per cent of the dextran could not be accounted for by the circulating dextran and the dextran excreted. This per cent of dextran unaccounted for gradually increased with time. This experiment supplies no information as to the fate of the dextran which could not be accounted for. However, other experiments have shown that dextran diffuses freely into the lymph and this may explain the discrepancy at 1 hour. Dextran is metabolized, which explains the loss at other time intervals. Studies utilizing dextran tagged with radioactive carbon have shown that 90 per cent was eliminated in 10 days. Of this, approximately two-thirds is excreted in the urine and one-fourth is expired as CO_2. It is assumed that dextran is metabolized to glucose and other experiments have substantiated this notion.

The administration of 1 liter of dextran produced an increase in the plasma volume of 750 cc. in one hour, which then decreased to 530 cc. in the subjects who were not bled. In those individuals who had been bled, the plasma volume after the administration of dextran was sustained as indicated in Figure 1. The shaded portion of Figure 1 indicates that portion of the increase in plasma volume which was related to the addition of native protein to the circulation. It is this addition of native protein to the circulation that accounts for the sustained plasma volume despite the excretion of dextran. It is important to determine whether the addition of native protein to the circulation proceeds at a normal rate despite the presence of dextran in the circulating blood. At the end of one hour, 3.3 grams had been added to the circulation; at the end of six hours, 15.7; and at the end of twenty-four hours, 30.6. These figures are similar to those obtained by Ebert, Stead, and Gibson[1] in experiments in which no plasma volume expander was given in subjects who were bled one liter. It then appears that dextran does not inhibit the addition of native protein to the circulation. Changes in the hematocrit and protein concentration were accounted for by the increase in plasma volume while the red cell volume remained constant. As would be expected on a percentage basis, the protein concentration decreased more than the hematocrit concentration.

The requirements for a satisfactory plasma volume expander have been outlined by the Subcommittee on Shock of the National Research Council. These requirements are: (1) maintenance of satisfactory colloid osmotic pressure; (2) constant composition, (3) suitable viscosity, (4) stability with temperature changes, (5) stability in storage, (6) ease of sterilization, (7) freedom from pyrogens, (8) absence of immediate adverse effects, (9) absence of immediate or delayed organic derangement, (10) absence of antigenicity, and (11) reasonable price. It would appear that dextran meets all of these requirements with the exception that it may possess some antigenicity. In addition to these requirements, an additional requirement should be listed. This is that dextran or any plasma volume expander should have no effect on the addition of native protein to the circulation. Were it to inhibit this addition and still be excreted, the plasma volume would rapidly decline.

TABLE IV. FLUID PREFERRED

	Whole Blood	Cells	Dextran	Plasma	Other
Anemia	S	B	O	O	O
Acute poisoning	S	B	O	O	O
Blood dyscrasias	S	O	O	S	S
Immunotherapy	S	O	O	S	B
Hypoproteinemia	S	O	S	S	B
Shock, hemorrhagic	B	O	S	S	S
Shock, burn	S	O	S	S	S
Shock, infection	S	O	S	S	S
Cerebral edema	O	O	S	O	B

B—Best S—Satisfactory O—Not Indicated

In Table IV are listed the preferred solutions for the indication given in the left hand column. By other preparations in this table, I refer to such things as the various protein fractions. I would like to point out that dextran or any other plasma volume expander is not indicated in anemia, acute poisonings associated with reduction in the oxygen carrying capacity of the blood, blood dyscrasias, and immunotherapy. It is evident from this table that dextran may not be considered a blood substitute. Considerable experience has been accumulated with the use of dextran in the treatment of hemorrhagic shock. It is in this circumstance that it would appear to have its greatest usefulness. It has also been effective in shock associated with burns and infections, but less effective than in shock related to hemorrhage. In circumstances where there is edema related to hypoproteinemia, dextran in a salt free solution may be effective in inducing a diuresis. In cerebral edema, concentrated dextran in a salt free solution may be effective. Such a preparation is not available commercially. The commercially available dextran is a 6 per cent concentration and is in 0.9 per cent salt solution.

In summary, then, blood is the ideal solution to use when the blood volume is depleted, particularly when that depletion is secondary to hemorrhage. A review of the records of a general hospital indicated that blood had been used unnecessarily in 16 per cent of the patients. In an additional 40 per cent it was impossible to ascertain whether a clear indication existed for the use of blood. While blood is the ideal solution, in the event of a disaster it may be impossible to obtain adequate supplies of blood. Under such circumstances dextran would seem to be a satisfactory plasma volume expander. In emergencies, particularly in small hospitals that do not have large reserves of blood, dextran again may be used while waiting to obtain blood or even as the sole agent if the blood deficit is not too great. Again, I would like to emphasize that dextran is not a blood substitute. In circumstances where only small quantities of blood seem indicated, perhaps dextran could be used, but under those circumstances nothing may be better than something.

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Acute Renal Insufficiency

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IN A BASAL state in health approximately one fourth of the cardiac output, or 1,200 cc. of blood, perfuses the kidneys each minute. This amount of blood is equivalent to a renal flow of 700 cc. of plasma per minute. About 19 per cent of the plasma water that perfuses the kidney each minute is filtered by the glomeruli, resulting in a glomerular filtration rate of 130 cc. per minute.

Numerous events may precipitate an intense and persistent decrease in renal blood flow and glomerular filtration rate, and acute renal insufficiency may ensue. During the past twelve years, a syndrome of acute renal insufficiency associated with a wide variety of precipitating factors has become recognized with increasing frequency. It is of the utmost importance that the syndrome be recognized early and proper treatment instituted, as errors in diagnosis and treatment may lead to death of the patient.

Acute renal insufficiency may be defined as a syndrome of rapid, but *reversible,* loss of renal function due to renal ischemia and disseminated areas of tubular degeneration. Synonyms that have been used are acute renal failure, acute tubular necrosis, lower nephron nephrosis, shock kidney, crush syndrome, hepatorenal syndrome, toxic nephrosis, burn nephritis, acute parenchymatous degeneration and extrarenal uremia. The term "lower nephron nephrosis," which implies localization of pathologic change to the distal tubule, is a poor one despite its wide acceptance, since the renal lesion is disseminated.

Pathologic Physiology

1. Shock.—The majority of patients with acute renal insufficiency will have experienced shock. In those patients who do not manifest the clinical signs of peripheral vascular collapse, hemoconcentration and a deficient circulating blood volume frequently will be present. The studies of Lauson and associates[2] indicate that the kidney participates to a pronounced degree in the vasoconstrictive phenomena of shock. The renal ischemia is proportionately greater than the reduction in cardiac output, and the glomerular filtration rate and tubular excretory function are greatly reduced. Organic tubular damage, suppression of urine and uremia may be the sequelae. If the patient recovers, normal renal function may not be established for weeks or months.

2. Pigment Casts.—Hemolytic transfusion reactions and other disorders associated with a hemolytic process are commonly followed by acute renal insufficiency. The distal and collecting tubules are frequently obstructed by hemoglobin casts, but obstruction *per se* is often an inadequate explanation of the oliguria. The precipitation of the casts appears to be secondary to intense renal vasoconstriction and reduction of glomerular filtration rate. Flink[1] has demonstrated that the value for hemoglobin in the plasma is an important determinant in the degree of tubular damage and the development of uremia. The crush syndrome, in which large amounts of myohemoglobin are released from striated muscle, occurs through a similar mechanism.

3. Tubular Toxins.—Tubular necrosis and hydropic degeneration are caused by numerous substances. Chief among these are carbon tetrachloride, mercuric bichloride and the sulfonamides. Experimental work indicates that the reduction in renal blood flow and glomerular filtration rate is secondary to the tubular damage. Poisoning with carbon tetrachloride is a frequent cause of oliguria. The diagnosis is frequently overlooked since the patient may appear to present the problem of primary liver disease with jaundice and hepatomegaly. In the absence of liver involvement, an incorrect diagnosis of an acute exacerbation of chronic glomerulonephritis may be made. Since 1947, an average of two cases of poisoning with carbon tetrachloride have been seen at the Minneapolis Veterans Hospital each year.

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4. Mechanism of Anuria or Oliguria.—Renal vasoconstriction and reduction of glomerular filtration, together with back diffusion of that filtrate which is being formed, account for the suppression of urine. Back diffusion occurs because of damage to the tubular cells.

Clinical Course

The clinical syndrome may be divided into three phases.

1. Shock.—This has been discussed already.

2. Acute Renal Insufficiency.—There is rapid development of progressive renal insufficiency ushered in by fever, nausea and vomiting, abdominal distention, occasionally jaundice, and pain in the costovertebral angle. Diminution in urinary output to 400 cc. per day or less is the general rule, but occasionally this syndrome may develop in spite of a daily urinary output of 500 to 800 cc. per day. Anuria is uncommon. The urine may be frankly bloody or smoky for the first several days; it is acid in reaction and contains large amounts of albumin. The initial urine may have a specific gravity of 1.017 to 1.020, but concentrating ability is rapidly lost and the specific gravity becomes fixed at 1.008 to 1.012. The urinary sediment contains erythrocytes, leukocytes, pigment casts, and hyaline and granular casts. A rapidly progressive normocytic normochromic anemia develops that may continue for several months.

As the oliguria persists there is a great tendency for the development of signs of congestive heart failure with pulmonary edema. This is especially true if salt and water are administered injudiciously. Hypertension may develop after the first few days, the systolic hypertension being more pronounced than the diastolic. Hypertension is not invariable. Six of twenty unselected patients seen at the Minneapolis Veterans Hospital did not have hypertension.

Gastrointestinal symptoms may continue in this phase, with abdominal distention, nausea and vomiting, and diarrhea. These symptoms, as is true of all others, may vary greatly from patient to patient.

Cerebral symptoms are common. There may be restlessness, delirium, stupor, and coma. Convulsions occur but are not common.

Early deaths are usually due to the primary disease, but fatal pulmonary edema may occur early if incorrect therapy is given. Deaths after the tenth day are due to pulmonary edema, uremia, or hyperpotassemia.

Laboratory tests reveal: a rapid fall in hemoglobin to 8 to 10 grams per 100 cc. of blood, with stabilization at this level; a rapidly rising blood urea nitrogen, decreased serum sodium, chloride and bicarbonate; and varying elevations of serum inorganic phosphorus and potassium. Potassium commonly rises to levels that are neurotoxic and cardiotoxic, especially if the serum sodium is low. Muscular weakness and pain, respiratory paralysis and flaccid paralysis of the extremities may occur when the serum potassium is at levels above 7.5 to 8 mEq. per liter. The first electrocardiographic abnormality is the presence of high peaked T waves, with a narrow base, noted initially in the precordial leads (usually V_2 first) and then in the limb leads. This may quickly be followed by depression of the ST segment, varying degrees of auriculoventricular and intraventricular block, disappearance of the P wave, and ventricular fibrillation. The sequence of events may be so rapid as to catch the unsuspecting physician off guard.

3. Recovery Period.—As tubular regeneration occurs, there is a gradual increase in urinary volume and diuresis occurs. The data of Swann and Merrill[3] indicate that diuresis is related to retention of water during the oliguric phase, this water probably coming from the oxidation of fats. There are some patients in whom depletion of the volume of extracellular fluid and circulating blood will be followed by circulatory collapse, but this is not the general rule. The blood urea may continue to rise for several days after diuresis occurs. This is usually not cause for alarm and steady improvement in the clinical course will be noted. In some instances, however, symptoms and signs of acute renal insufficiency will progress in spite of diuresis. After clinical recovery, normal renal function may not be reestablished for many months.

Differential Diagnosis

The following diseases must be considered in differential diagnosis: acute glomerulonephritis, acute exacerbation of chronic glomerulonephritis, acute or chronic pyelonephritis, ureteral obstruction from calculus, sulfonamide crystalluria, metastatic cancer and renal vascular lesions, such as malignant hypertension and renal infarction. As a general rule, in the absence of a history of an event known to precipitate acute renal insufficiency, the diagnosis will usually prove to be another form of renal disease.

Treatment

Principles.—There are several important principles governing therapy:

1. Renal ischemia produces a *reversible* renal shutdown. Tubular necrosis is potentially reversible, and the renal lesion may heal completely.
2. The kidneys cannot be "flushed out" by large amounts of salt and water in the presence of oliguria or anuria.
3. The principal cause of death is pulmonary edema.
4. Hyperpotassemia results from intake of potassium as well as from breakdown of tissue.
5. In a 70-kg. man with sudden cessation of renal function, loss of 1,000 cc. of insensible water occurs daily. The preformed water of oxidation is about 470 cc. daily, leaving a daily deficit of 530 cc.
6. Administration of carbohydrate will spare breakdown of protein.
7. The phase of diuresis cannot be accepted as one of complete recovery, and the patient must be carefully observed in this phase.

Measures.—Therapeutic measures in keeping with the principles just noted may be outlined as follows:

1. Combat shock promptly with effective measures.
2. Evaluate urinary output in any patient in whom the diagnosis is suspected before ordering parenteral fluids.
3. No more than 500 to 750 cc. of a 10 to 15 per cent solution of glucose should be given daily. In the usual case 500 cc. daily will suffice.
4. If the patient is able to tolerate fluids orally, either emulsions of fat (Ediol) or a modified Borst regimen of butter soup and butter balls may furnish sufficient calories in a small amount of fluid to decrease cellular breakdown with its attendant biochemical abnormalities.
5. Keep in mind the susceptibility to pulmonary edema. Whole blood is *not* indicated to correct the developing anemia. Avoid use of sodium chloride even in small amounts. Do not give sodium bicarbonate unless metabolic acidosis threatens life.
6. Frequent serial electrocardiograms and measurements of serum potassium, when available, are necessary to evaluate hyperpotassemia.
7. Insulin and glucose (1 unit for each 3 to 5 grams of glucose), may be used as an emergency measure to combat hyperpotassemia. In critically ill patients such treatment may be followed by hypoglycemia.
8. If the patient is being treated properly during the phase of acute renal insufficiency, he will lose about one-half pound per day. If body weight is maintained or is increasing, overhydration is present.
9. In the early recovery phase parenteral fluids must be given with caution since there is a delayed diuretic response to water. Pulmonary edema and cerebral symptoms may recur with injudicious use of salt and water. After twenty-four to forty-eight hours of the early diuretic phase, daily loss of salt and water may be safely replaced over a period of twenty-four hours. Urinary volumes and the concentrations of electrolytes in urine and serum may serve as guides to replacement therapy.
10. With the onset of recovery, a moderate-protein, high-carbohydrate diet is indicated.
11. As noted by Swann and Merrill,[3] any sensible plan for restricting water, sodium, chloride, potassium and protein and providing calories as carbohydrate will result in low morbidity rates and recovery. In some severely-ill patients, especially those with hyperpotassemia, the artificial kidney may be lifesaving.

Prognosis

Patients with acute renal insufficiency secondary to chemical toxins appear to have a milder course and to regain normal renal function more rapidly than do those whose condition is secondary to traumatic, oligemic or postoperative shock. This may be correlated with the degree of negative nitrogen balance and cellular breakdown, which is more intense in the latter group. The mortality rate has been extremely high (80 to 90 per cent) in the past. With a sounder understanding of the reversibility of the renal lesion and the pathologic physiology, better treatment has sharply decreased this mortality rate.

(References on Page 59)

Alterations of Carbohydrate Metabolism Following Trauma

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IT HAS been known for years that injury may produce abnormalities in carbohydrate metabolism. As early as 1854, Goolden[30] reported that patients who sustained an injury not infrequently had considerable sugar in the urine, accompanied by a diuresis. As the patient's condition improved the glycosuria disappeared, in many instances within ten days. Following these observations, Claude Bernard and others referred to transitory hyperglycemia and glycosuria following various types of trauma. In 1911, Cannon, Shohl and Wright[14] showed that an emotional glycosuria could be produced in animals but it did not occur after an adrenalectomy with a comparable stress. In 1914, Epstein and Baehr[27] reported an elevation in the blood sugar concentration following hemorrhage, and two years later Epstein and Aschner[26] concluded that anesthesia also played an important role in the production of hyperglycemia. Several years later, Keeton and Ross[33] found about a 100 per cent increase in the blood sugar concentration in normal dogs after they had been under ether anesthesia for two hours. Cannon[11] and Aub and Wu[8] demonstrated hyperglycemia following traumatic shock, and Underhill and co-workers[46] described the same finding in patients following a thermal burn.

In 1925, Davidson and Allen[21] reported studies on numerous patients following head trauma. Figure one is a composite summary prepared from their data. They noted that blood sugar concentrations, after the intravenous administration of 125 ml. of 25 per cent dextrose, were higher in patients following a concussion of the brain than in the same patients later during convalescence or in normal control subjects. Twenty-four to thirty-six hours after a skull fracture the blood sugar values were even higher and "the fall of the curve to the fasting level was still more delayed." They concluded that concussions of the brain and fractures of the skull produced a profound, though temporary, derangement of carbohydrate metabolism and that the more serious the injury, the more striking was the disturbance in the blood sugar levels following the administration of glucose.

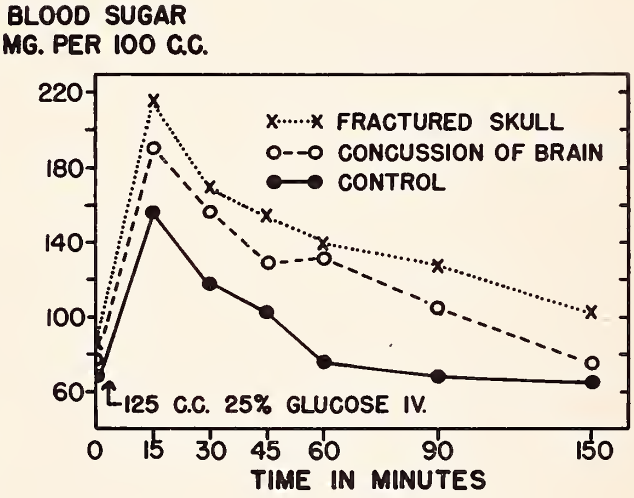

Fig. 1.

It was studies such as these and the results of the nitrogen and chloride balance data reported by Davidson and his associates[20,21] on burned patients, and the reports of other investigators[12,19,44] over the next few years, that eventually led to our present-day concept of the pituitary-adrenal relationship to the findings noted after injury.

In 1938, Thomsen[45] reported his results of extensive studies on traumatic diabetes. He concluded that post-traumatic disturbances in the carbohydrate metabolism, manifesting themselves by spontaneous or alimentary glycosuria and hyperglycemia, were evident in a considerable number of previously normal persons. He also noted that the kind and location of trauma was not of prime importance since there was no difference

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between head trauma and peripheral injuries. He concluded that "a physical trauma may give rise to an exacerbation of existing diabetes, but the exacerbation manifesting itself immediately after the trauma is only temporary."

During the past twenty years, more information concerning the role of the pituitary and adrenal glands has been published. In 1940, Long, Katzin and Fry[36] noted that adrenalectomized rats, who refused food and sodium salts, or were forced to fast, show a marked depletion of liver and muscle glycogen. These workers noted that such changes could be prevented by the administration of adrenal cortical extract and that under this therapy liver glycogen could be increased well above normal, even in fasted animals, thus confirming previous observations of Britton and Silvette.[10]

When the adrenocorticotropic hormone and cortisone became available, numerous other significant contributions to our understanding of the metabolic alterations noted following trauma or stress appeared. In 1948 and 1949, Conn and his associates[17,18] reported the results of their metabolic balance studies of three normal young adults who were given ACTH intramuscularly for eight to ten days, following a control period. These subjects demonstrated hyperglycemia and glycosuria, as well as a plateau type glucose tolerance curve. Two of the subjects exhibited a negative nitrogen balance and lost large amounts of body protein, despite an adequate oral food intake.

We have been concerned with the metabolic alterations following burns and lesser forms of *trauma* for a period of twelve years and are particularly interested in the possible therapeutic measures which might be employed to reduce the morbidity and mortality of the post-traumatic patient. More recently, we have been interested in carbohydrate metabolism, since with the advent of fructose and invert sugar new materials are available which are metabolized differently than glucose.

Time does not permit a review of numerous contributions[1,9,15,16,22,28,50] which led ultimately to the clinical use of fructose and invert sugar during the past few years.[23,24,38,41,47,48,49] Because recent studies[38,41] indicated that the metabolism of fructose differed from that of glucose in both the normal and diabetic subject, and because the metabolism of fructose in the person with diabetes, even in the absence of insulin, is similar to that seen in a normal subject, it was decided to undertake studies in postoperative patients to determine whether the two sugars would be utilized differently. The results of these studies have been presented previously.[23,24]

Price, Cori and Colowick[42] proposed that insulin acts on the hexokinase, which converts glucose to glucose-6-phosphate, whereas the phosphorylation of fructose is under the control of a different hexokinase which is not affected by the lack of insulin. It therefore seemed important to determine if the hyperglycemia and glycosuria seen postoperatively was due to an impairment of the glucokinase activity. If such were the case, the metabolism of fructose in such patients should be normal. Accordingly, fructose and glucose in amounts of 1.0 gm. per kilogram was administered intravenously as a 10 per cent solution over a period of sixty minutes to patients on alternate days preoperatively and postoperatively. Five otherwise normal adult males, undergoing a unilateral herniorrhaphy, under spinal anesthesia, were used for this study, and blood samples drawn periodically were analyzed for glucose, fructose, pyruvic acid and serum inorganic phosphorus. The results indicated that while a higher glucose tolerance curve was obtained postoperatively than preoperatively, the postoperative fructose tolerance test was essentially the same as that noted preoperatively. The blood pyruvic acid levels and serum inorganic phosphorus concentrations indicated a better utilization of glucose preoperatively than postoperatively and also that the metabolism of fructose was essentially unaltered by operation. The four-hour excretion of hexose in the urine during and immediately after the tolerance tests indicated that more glucose was spilled postoperatively than preoperatively and that more hexose was present in the urine following the administration of glucose preoperatively than occurred postoperatively after fructose.

In subsequent studies,[4] glucose tolerance curves were obtained in patients undergoing a more severe type of stress or operative trauma. In such patients, the glucose level in the blood, observed after the intravenous glucose tolerance test, was higher than that noted following a herniorrhaphy, and the abnormality persisted for a longer period of time. In patients undergoing a gastrectomy, even though the glucose was infused at a slightly slower rate, it was not unusual to find 5 to 45 gm. of sugar (equaling from 2 to 30 per cent of that infused) in the urine per twenty-four hours during the first five to six postoperative days.

Other workers have made similar observations and noted that fructose and invert sugar could be better utilized than glucose.[47,48,49] Several groups [0,25,40] have also presented evidence to show that the utilization of nitrogen might be enhanced by the simultaneous administration of a hexose (especially fructose). While more work along this line must be done before definite conclusions can be reached, these observations may be of importance in helping to minimize the wasting of the body tissues following a severe injury.

Albright[7] has reported, in detail, the metabolic alterations that occur in patients with Cushing's syndrome. He presented glucose tolerance curves in four such patients. They all showed a very high fasting blood sugar level and a typical diabetic type of curve. Recently, glucose and fructose tolerance tests were done in a patient with Cushing's syndrome preoperatively while she was a diabetic and again several months after a total adrenalectomy.[3] The diabetes which had been present for several years was completely alleviated by the adrenalectomy. A typical diabetic glucose tolerance curve was obtained preoperatively, and a normal curve was present several months postoperatively. The preoperative and postoperative fructose tolerance curves were similar, indicating no interference with this hexose in hyperadrenocortical states. Since such patients presumably exhibit these changes because of an excess of the sugar or S hormone it is conceivable that a comparable derangement of endocrine activity exists temporarily following trauma.

The problem of therapy is still somewhat confused, but because of the information which has accumulated during the past ten years, some specific recommendations can be made. We feel that it is not desirable to force-feed, either orally or parenterally, the very ill or severely injured patient for the first few hospital days. These conclusions were reached after attempting early forced feeding in patients following a severe thermal burn.[2,31] Detrimental effects were noted by forcing a high caloric, high protein diet during the period of acute illness. However, after several days in the more severely injured patient and in patients exhibiting lesser forms of trauma, such as that induced by most surgical operative procedures, an adequate well rounded diet containing all necessary nutrients should be given.

If this cannot be accomplished orally, or if the giving of food by mouth is undesirable, intravenous alimentation should be used. Our studies[4,5,35] done during the past two years indicate that considerable loss of body weight and negative nitrogen balance might be prevented, at least in part, by the giving of 1,600 to 3,200 calories and 12 to 15 grams of nitrogen. While an insufficient number of patients have been studied, we have noted a better maintenance of weight, and a smaller loss of nitrogen if an intravenous fat emulsion and 250 to 350 grams of sugar are given in conjunction with an adequate protein intake. We noted that patients with peritonitis who have received amino acid solutions intravenously, along with other nutrients, showed considerably less nitrogen deficit than others who were maintained solely on intravenously administered carbohydrate. These impressions are in keeping with the results reported by Madden and Clay[37] who did nitrogen balance studies before and after the production of a turpentine abscess in dogs. They found that an increased catabolism of protein was responsible for an accelerated loss of nitrogen in the urine and were able to reduce the nitrogen deficit by supplying large amounts of protein.

Our studies[5] also tend to confirm the experimental work of others[32,39] that marked abnormalities in electrolyte balance *per se* will induce a negative nitrogen balance or a catabolic response. Non-operated patients with pyloric obstruction exhibited glycosuria and increased nitrogen loss which could be nearly or even entirely negated by the administration of the proper electrolytes, without need of additional calories or protein. Although recently[13,29,34,43] there has been considerable emphasis on the role of potassium and its possible relationship to carbohydrate and protein metabolism, in our patients we have been impressed with the alteration noted following a sodium deficiency. We do not wish to minimize the importance of potassium, but since more rapid and extensive deficits of sodium frequently occur, the importance of this ion should not be overlooked. This is especially true since we are passing through an era of sodium restriction. In many instances, in our opinion, this is well beyond that which is good for the patient. The importance of sodium was recently re-emphasized during studies[5] on patients with pyloric obstruction who were losing considerable quantities of fixed base (especially sodium). During a seven day period one of these patients, who received moderately large amounts of sodium and potassium, developed cumulative deficits of over 500 mEq. of sodium and a 108 mEq. of potassium. In spite of an adequate intravenous caloric and protein intake, he showed a negative nitrogen balance and glycosuria. In previous studies[4] we were unable to demonstrate any effect on protein and carbohydrate metabolism when deficits of 100-400 mEq. of potassium existed. In spite of deficits of this magnitude (which represented only a 3-12 per cent decrease of the total body potassium) our patients showed a normal glucose tolerance curve and no significant effect on protein metabolism. The administration of 100 to 150 mEq. of potassium daily for several days, after permitting deficits of the aforementioned magnitude to accumulate, again showed no demonstrable effect.

It now is well established that the first aim in parenteral therapy is a restoration of blood volume and the correction of an abnormal electrolyte pattern (extracellular or intracellular compartmental deficits or excesses and acid-base balance). The prompt institution of such therapy constitutes an emergency and should take precedence over all other types of nutritional supplementation. At the same time, however, after specific immediate therapy is begun, there is no need to neglect the nutritional aspects of convalescence when there are now available parenteral solutions which contain adequate amounts of nitrogen, fat and carbohydrates, and by employing combinations of these to which the necessary minerals and vitamins have been added, one can provide complete and adequate nutrition. The many and varied facets which characterize man's response to stress must be considered as a whole and the therapy planned to adequately correct or protect as many of these features as possible. In this manner one can approach an ideal type of treatment which will minimize the effects of the stress, improve the convalescence, and diminish the mortality rate.

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Discussion

Dr. Jacobson: It has been previously pointed out that there is an apparent difference between the hyperglycemia following use of adrenal corticotropic hormone and that of true diabetes. Dr. Conn has shown that glutathione is increased in the blood in cases of ACTH-induced hyperglycemia, whereas this substance is not elevated in true diabetes. Other authors have differentiated so-called "steroid diabetes" and "pancreatic diabetes." At least one other difference between these two types of diabetes is the degree of insulin sensitivity. It has been shown that patients with "steroid diabetes" are relatively insensitive to insulin. I would like then to ask Dr. Abbott to discuss observations to which he has made with regard to the degree of sensitivity to insulin in postoperative hyperglycemia. I would also ask him to describe the role of insulin in the treatment of this type of hyperglycemia.

Dr. Abbott: In regard to the question concerning insulin, we have not had a great deal of experience with its use in non-diabetic postoperative patients.

The glucosuria and hyperglycemia that we have seen after major operations have not been, on the whole, severe enough or persistent enough to warrant use of insulin. A few studies have been started in an endeavor to determine the answer to some of the problems that Dr. Conn and others have pointed out. Dr. Rice says that he has used insulin in the treatment of patients exhibiting postoperative hyperglycemia. I have been satisfied that utilization of some of the other sugars (invert or fructose), with slow administration, will prevent severe hyperglycemia and the glycosuria will be relatively mild.

Dr. Beal recently gave 400 to 500 grams of sugar per day in the form of 20 to 25 per cent solutions of dextrose. It was administered at the rate of about 1 gram or less per kilogram of body weight per hour. In those patients he observed less than a 10 per cent spillage of sugar.

I enjoyed the paper Dr. Heller presented dealing with anuria. We have been interested in this problem for the past ten years and I think that with the artificial kidney which we have used (Skeggs-Leonard kidney) we have been able to extract as much as 1,200 cc. of water per hour, so that over a period of five or six hours it is not difficult to remove six or seven liters of fluid. We have noticed that patients with pulmonary edema and overhydration show great improvement when water has been extracted. These patients have been then returned to a normal state of hydration, and have shown a more dramatic clinical improvement than almost any of the other types of patient.

At Walter Reed Hospital, I recently had occasion to observe a patient who was severely traumatized. Within two days he showed extremely high values for serum potassium and blood urea nitrogen so that he had to be dialyzed three times in a period of about nine days in order to alleviate the effects of hyperakalemia. Most of the potassium was coming from a crushed necrotic muscle of his lower extremity. The condition was greatly relieved when an amputation was performed. The patients we see clinically, such as those with chronic renal disease or acute renal shutdown following a transfusion reaction, or after poisoning with bichloride of murcury of sulfonamides, are much easier to care for than those we encountered during the war in Korea, because of the lack of trauma. The existence of an associated injury and the catabolic phase that follows greatly accelerates the danger and rapidity of hyperkalemia.

Question: Dr. Elman, do you hesitate to give amino acids in the presence of liver damage?

Dr. Elman: Originally I did, because I have observed a number of patients suffering from severe liver disease in whom the level of blood amino acids was high. Thus I reasoned that amino acids were not being utilized. Subsequently, Dr. Stewart of Buffalo gave amino acids to a number of patients with jaundice and other evidences of severe liver disease; as far as he could tell clinically and chemically, amino acids were utilized just as well as in the normal individual. Later, the Thorndike laboratory in Boston obtained evidence to show that intravenous administration of amino acids, along with albumin, was beneficial in severe liver disease. We have seen a number of patients suffering from hepatic coma. I had one patient who was extremely grateful when she came out of hepatic coma by responding beautifully to the intravenous feeding of glucose and amino acid as well as pure albumin. All the work done on liver disease and liver regeneration indicates the nutritional value of protein as well as carbohydrate in the resistance to hepatotoxins and the ability to overcome liver damage.

The old experience with chloroform poisoning proved that the administration of carbohydrate to patients with damaged livers enabled them to withstand the effects of such poisoning. In fact, that was the way patients were prepared for operation when chloroform was to be used. When protein as well as carbohydrate is added, the beneficial effect on the liver is increased. As a result of all these observations, I have changed from my original opinion and now I feel that protein is an extremely important part of nutritional therapy in patients with severe liver disease.

I would like to emphasize again that parenteral nutrition in any form to be used only when patients cannot obtain nutrition by mouth.

Question: Dr. Elman, will you compare the relative values of blood and amino acids in correcting protein deficiency?

Dr. Elman: I think we are dealing with two entirely different types of deficiency. One group of proteins comprises the circulating proteins, namely hemoglobin and plasma proteins. The other consists of the fixed proteins, or tissue proteins.

If there is a deficit in circulating proteins, which may occur even though the concentration is normal, the simplest, most direct and quickest way to correct that deficiency is to give enough blood to eliminate the deficit. The tissue or fixed proteins, normally are not replaced by that method. The physiologic way in which a deficiency of tissue protein can be restored is to enable those tissues to make their own protein by presenting them with the proper mixture of amino acids and polypeptides.

This can be done by letting the patient make his own amino acids from the protein that he ingests and digests normally, or amino acids can be administered parenterally. Tissues can also utilize intravenously injected whole blood (hemoglobin and plasma protein) by a roundabout way that is to a certain extent unphysiologic because it does not happen normally. If enough blood or plasma is given for this purpose, and some workers have advocated this as a method of feeding protein, the excess leaves the blood stream. The tissue proteases break down this protein and hemoglobin and the resulting amino acids are picked up and fabricated into tissue protein. This may begin rather soon after the injection has been given, but it is rather prolonged process: it takes about ten days for completion, according to studies of Fuller Albright and his co-workers.

I feel that since it is really unphysiologic and delayed, and since, in most cases by the end of ten days the patient should be able to take food by mouth, this method of furnishing the tissues with amino acids should not be used. Furthermore, it is more expensive than giving amino acids directly into the vein.

I prefer to give patients blood to restore the deficiency of the circulating proteins. As soon as patients can tolerate food by mouth, I prefer that they receive as soon as possible large amounts orally; usually 200 to 300 grams of protein per day, with 4,000 to 5,000 calories given if a severe deficiency is to be corrected rapidly.

Question: Dr. Lowe, would you like to discuss anything further regarding the problem of amino acid therapy?

Dr. Lowe: We feed infants with a mixture of amino acids, protein and carbohydrates. When amino acids are given by mouth, allergic reactions are minimized and digestion is spared; in other words, the body gets an already completely digested protein and does not have to break it down by means of the proteolytic ferments of the stomach and the intestine. The amino acids are available immediately and these can be absorbed directly and rapidly. I imagine there must be certain conditions in which the gastrointestinal tract is only partly disabled and can handle some food, though it may need a little help by being given predigested or completely digested proteins.

We have provided additional calories by substituting alcohol. These children seem to grow adequately on it. We have used the type of mixture which Dr. Rice has given intravenously. I don't think that anybody has been quite successful in keeping a complete nutritional balance by the intravenous use of alcohol. We did this just to see if it would make babies grow. We used the mixture which Dr. Rice has used for his complete parenteral feeding. We found some difficulty in using it because it irritated the veins.

Dr. Elman: I, too, would like to comment on that subject. We have given alcohol intravenously and I don't really know why I don't use it more frequently. The only objection I have found is that the patient's room smells of the alcohol which is excreted in the expired air. That is obviously just a prejudice. I don't know why social taboos should interfere with the progress of science, but apparently in this case they do.

Question: In acute renal insufficiency there is frequently a profound hypochloremia. This does not necessarily mean sodium loss, but is usually a reflection of a shift in electrolytes. A common error in treatment of acute renal insufficiency is to attempt correction of hypochloremia by administration of salt solution. It is now clearly understood that hypochloremia itself is not an indication for the administration of saline solutions. I would like to ask Dr. Heller whether saline solution should ever be a part of the replacement program for patients with acute renal insufficiency. Specifically, should saline be used for replacement of fluid lost by emesis?

Dr. Heller: Sodium chloride should not be replaced during the phases of renal insufficiency or the phase of oliguria, even if the patient is losing small amounts of sodium chloride by vomiting. These patients have a tremendous susceptibility to pulmonary edema. Freeberg, in reviewing the literature, has shown the importance of differentiating between what I call primary heart failure and heart failure that is secondary to renal insufficiency wherein too large amounts of salt and water have been replaced during the oliguric phase. These patients who have decreased sodium and chloride in the serum do not seem to be affected by this decrease, but they can die as the result of small amounts of pulmonary edema. As a matter of fact, a shift of 300 to 500 cc. of fluid into the lungs can be fatal. I think it would be much safer not to replace any salt lost during the phase of oliguria and much safer to give no sodium bicarbonate or sodium lactate unless one feels that the state of acidosis is actually threatening the life of the patient.

Question: It is my impression that while there are complex factors which control the excretion of potassium, the single most important factor is the urinary volume. Even in the face of severe renal damage, hyperpotassemia is unlikely when the urinary volume is more than 500 cc. per day. I would like to ask Dr. Heller to discuss further the factors which are important in the development of hyperpotassemia in acute renal insufficiency.

Dr. Heller: In most instances the hyperpotassemia is related to urinary volume. When patients manifest cardiotoxic and neurotoxic symptoms and signs of hyperpotassemia, the urinary volume is usually less than 400 cc. per day. This, however, is not universally true. Some patients may excrete as much as 800 cc. of urine per day and still have fatal hyperpotassemia. Lethal hyperpotassemia may occur even during the phase of diuresis. Dr. Flink and I have had the opportunity of seeing patients at Veterans Hospital in Minneapolis who developed severe hyperpotassemia after the phase of diuresis had begun and when the patient was excreting more than a liter of urine per day. It has also been noted that in patients with traumatic or oligemic shock—because of the markedly negative nitrogen balance—hyperpotassemia may be a more serious problem than it is in the acute renal insufficiency that occurs after poisoning with carbon tetrachloride.

Question: Recently forced high-fat, low-protein oral feeding has been recommended in acute renal insufficiency. I would like to have Dr. Heller discuss the use of such a forced feeding program.

Dr. Heller: Oral treatment for patients with acute renal insufficiency unfortunately is unsatisfactory because of nausea, vomiting and diarrhea. Many of these patients have abdominal distention. Such patients cannot take any food or fluid by mouth. For those who can take small amounts of oral feedings, the use of *Ediol,* which is a high-caloric-fat-emulsion mixture, or the use of the forced regimen as modified by Dr. Flink's wife through the use of butter soup and butter balls, can be highly successful in patients who can tolerate such a mixture. One of the most ideal patients that I have ever treated received what I now call "Mrs. Flink's butter-ball soup mixture." This is a mixture of cornstarch, glucose and butter that really is quite palatable. This patient had carbon tetrachloride poisoning and was able to tolerate butter soup feedings by mouth. His blood urea nitrogen never rose above 165 mg.; the phosphate did not rise to the extremely high levels so often seen; the potassium did not get above 6 mEq. per liter and the carbon dioxide combining power did not fall below 17 mEq. He made one of the smoothest recoveries I have ever seen. I feel that in a small number of such patients with acute renal insufficiency who can tolerate oral feedings, the sparing of protein breakdown can definitely facilitate recovery.

Question: Dr. Heller, will you please discuss the relationship of preoperative electrolyte imbalance and the age of the patient, as they pertain to the frequency of postoperative acute renal insufficiency?

Dr. Heller: The spectrum of acute renal insufficiency is broad. Renal function may be impaired during and after operations. It is not known how small degrees of impaired renal function, as may be seen during the first twenty-four to forty-eight hours after operation, fit into this syndrome. The role of metabolic alkalosis in this syndrome is likewise unknown. A renal biopsy done on Dr. Hay's service at the Minnesota's Veterans Hospital, on a patient who had metabolic alkalosis from pyloric obstruction appeared to be entirely normal.

If a patient manifests a greatly impaired balance of electrolytes and water preoperatively, we feel that this situation should be corrected. The urinary volume should then be carefully observed in six hours to make sure that acute renal insufficiency is not developing. As already mentioned, the administration of too much fluid to a patient who has oliguria can be fatal because of pulmonary edema.

Question: Dr. Ariel, do you wish to comment on the problem of acute renal insufficiency?

Dr. Ariel: It is extremely difficult to differentiate carefully between acute renal insufficiency and renal insufficiency due to metabolic alkalosis. We see an average of one patient every two weeks, who, after surgery, is suffering from loss of water or electrolytes. Undue loss from an ileac stoma, undue drainage because of Wangensteen suction or failure of proper administration of certain fluids may be the cause. Most of these patients have a high blood urea nitrogen. In addition, we see oliguria and a decrease in chloride. The carbon dioxide combining power is usually elevated and the sodium and potassium values vary from normal to abnormal. These patients are sick; they will not eat because they are nauseated, and they vomit. They must have fluid and electrolytes. Administration of a polyionic solution of electrolytes will relieve them overnight. This solution must be given boldly and with complete appreciation of the defect. As soon as the solution is given, urinary output increases. Blood urea nitrogen comes down within twenty-four hours and the electrolyte status becomes normal. If unattended, I don't know whether or not such patients would go into a state of renal insufficiency. Thus it becomes paramount to differentiate the acute anuric syndrome due to renal damage from that due to metabolic alkalosis. In one case fluid is withheld, as Dr. Heller has said, and in the other, fluid and electrolytes are forced to keep the patient out of danger.

Question: Dr. Hammarsten, will you discuss further your explanation of a patient with acute gastrointestinal hemorrhage and a hemoglobin of 12 grams in whom hemodilution has not occurred?

Dr. Hammarsten.—My awkward six-year-old boy went out to feed the hogs on my farm. The hog feed is mixed with water in a ratio of one to four. After mixing the hog feed, he tripped and spilled some of the mixture. The concentration of feed to water was then determined and found to be the same as it had been previously. He didn't have time to return and add more of the feed, so he restored the mixture to its original volume with water. Then the concentration of hog feed became lower. In this illustration the feed, of course, represents red blood cells and the water represents blood plasma.

Question: Dr. Hammarsten, do you recommend weighing the blood lost at operation and do you recommend full replacement of such calculated blood loss?

Dr. Hammarsten: As an internist, I have criticized the surgeon and anesthesiologist for giving blood when it isn't indicated. Accurate measurement of the amount of blood lost would be of more value than a guess. When only a few hundred cubic centimeters of blood are lost, I do not see that it would necessarily need to be replaced. On the medical service we sometimes withdraw that much blood in doing laboratory procedures.

Dr. Elman: I would like to comment on that problem. I agree wholeheartedly with everything that Dr. Hammarsten has said about the excessive use of blood and other parenteral fluids. However, in massive upper gastrointestinal hemorrhage we previously gave just enough blood to get the patient out of shock, but more recently we found that that policy often led to disaster.

When confronted with massive hemorrhage we aim to give as much as is needed to replace the blood lost. We do not try to restore the hemoglobin to 14 or 15 grams, but rather to a minimum of 10 grams. We would operate if there were evidence of persistent or recurrent bleeding. In about 50 per cent of these cases, the massive hemorrhage stops spontaneously, and then we can operate electively as indicated; in the other half, operations are done as emergencies.

Question: Dr. Ariel, would you like to make some remarks about this problem?

Dr. Ariel: We have given Dextran solutions in place of blood to patients on the operating table. These patients have tolerated the procedure well. Our biggest difficulty, however, has been in estimating the blood volume of the patient the next morning because the Dextran has so diluted the blood that determination of the hemoglobin and hemocrit has lost its value, and it is impractical to determine blood volume routinely.

Question: Dr. Ariel, would you have any ideas about proper estimation of blood volume after operation?

Dr. Ariel: The methods employed today for measurement of blood volume are not accurate enough to provide a good estimate of what the patient's expected normal blood volume should be. If the blood volume is calculated from the height or weight or surface area of the individual, there will be a 10 or 15 per cent error. Studies by Dr. Lyons, and more recently by Dr. Ellison in Columbus, show that many of the cancer patients whom we transfuse come to the hospital greatly depleted. Many of them are malnourished and have blood volumes that are extremely diminished even though the concentrations of plasma protein and hemoglobin and the hematocrit value may be relatively normal. These patients have been anemic for long periods and have compensated for it; yet they are sitting on a keg of dynamite, because they cannot tolerate the shock after a relatively small loss of blood. On the other hand, we should not pour blood indiscriminately into healthy individuals who have no need for it. I believe with Dr. Elman that the loss of a few hundred cubic centimeters is hardly enough to require replacement.

Question: Dr. Seldin, would you discuss the management of a dynamic ileus in a patient with congestive heart failure, who requires continuous nasal gastric suction? Also please discuss the problem of hypochloremia in such states.

Dr. Seldin: It is well known that potassium deficiency may eventuate in *ileus*. To that extent that gastrointestinal suction produces a deficiency in potassium, it is sensible to administer potassium when ileus exists in order to replace the deficit. This may be sufficient to overcome the ileus and the condition may be reversed.

In congestive heart failure, there is massive expansion of extracellular fluid so that gastric suction may eventuate in no overall salt depletion in the sense that the patient may still be edematous even though large amounts of salt, chloride and acid have been removed from the gastrointestinal tract. The administration of potassium may be sufficient to correct the potassium deficit as well as the alkalosis. At times the administration of small amounts of ammonium chloride may help.

This panel has been so blissful and so full of accord on every event, that I hesitate to interject a note of dissent, but I would like to register a gentle protest against the position that almost everyone seems to have agreed upon. Dr. Heller has vigorously and distinctly formulated the idea that salt should not be used in acute tubular necrosis. The impression that one gleans from this discussion is that if one waves five grains of salt beneath someone's nose, pulmonary edema will ensue, with a lethal outcome. I would like to make a few preliminary remarks on this score because I don't think they are irrelevant.

A large proportion of lower nephron nephrosis is a simple consequence of salt-depletion shock. This could have been predicted from the work of Darrow long before potassium became the magic cause of all human ailments. Salt-depletion shock is a well-known entity. The net result of salt depletion is a catastrophic fall in renal plasma flow and the glomerula filtration rate, oliguria and even anuria. In its early stages, salt-depletion shock is completely reversible by the administration of adequate amounts of salt and water. The term "acute anuria" or "acute renal insufficiency" includes at least two distinct stages. In one there has been an anatomic lesion of the tubules leading to a prolonged state of oliguria or anuria even when the initial insult has been rectified on the initial depletion corrected. In the other state, acute oliguria or anuria may supervene, but it is completely correctible when the initial stress has been removed. I wish to confine my remarks to salt depletion. When a patient has had a profound restriction of salt for one reason or another, and has had a major surgical procedure, some period of oliguria or anuria frequently may develop. At times this may be severe. Under these circumstances it is often impossible to say whether or not the patient has suffered significant renal injury. The only way to be certain is to replace what is assumed to be a deficit and await the response. No convenient methods now are available for the measurement of extra cellular volume. The estimation of the deficity must rest on an intelligent appraisal of the clinical condition of the patient and a critical assay of the history that has preceded the period of anuria. It is my belief that it is slightly safer to overestimate the amount of salt and water to be replaced than to underestimate it. Nobody advocates flooding the patient and inducing massive edema. It is simply a question of restoring the volume of extracellular fluids to nearly normal volumes. During the course of lower nephron nephrosis, severe losses of salt and water may take place. This may occur in the form of diarrhea with ulcerative lesions of the colon. It may occur in mercury poisoning with severe diarrhea or it may occur if suction is employed to relieve ileus. Large amounts of salt and water are removed from the gastrointestinal tract in these instances. This can result in a serious fall in renal plasma flow with delay in or prevention of restoration of renal function.

Moreover, the transport of sodium and chloride into the cells does not mean that the extracellular volume is normal. It means that the extracellular space is shrunken. The presence of sodium and chloride in cells doesn't necessarily immunize the body to the effect of depletion of salt. Under these circumstances, it is my view that it is safer to try to maintain the volume of the extracellular fluids at nearly normal values. I would, therefore, disagree with the position that small amounts of saline or bicarbonate are harmful, particularly if they are intelligently administered with a view toward maintaining the composition and volume of the extracellular fluid in a fairly normal manner.

Question: Dr. Darrow, would you like to comment on this problem of salt depletion?

Dr. Darrow: I was not aware that renal damage could be produced by a decrease in the circulation of the kidneys as a result of salt loss. It is possible that most examples of so-called lower nephron nephrosis accompany other types of shock rather than that which is associated with depletion of salt. I don't think Dr. Heller made himself absolutely clear; I understood him to say that at the start of treating anuria he corrected extracellular fluids to more or less what he thought was a normal volume and then started from there, using minimal amounts of salt.

Question: Dr. Heller, can you explain this?

Dr. Heller: I started my talk by saying that in order to combat shock the patient who presents himself with profound electrolyte and fluid disturbances should be brought to a state of balance and then be observed carefully to see whether acute renal insufficiency is going to develop. As to depletion of salt, *per se,* causing the oliguric or anuric syndrome, the evidence is indeed to the contrary. The experiment done by nature in Addison's disease, in which a profound depletion of salt and water occurs, is not followed by anuria or oliguria. Some of the studies in England on salt and water depletion indicate a decrease in renal function, though it is not pronounced. There are no patients in my experience who have died in acute renal insufficiency or dehydration or depletion of the extracellular volume. The invariable finding at necropsy is pulmonary edema, and I think it is well established that these patients are extremely sensitive to replacement of salt and water. They have a tendency to experience pulmonary edema when even small amounts of salt and water are replaced.

To be sure it is desirable to have normal extracellular fluid volume, but it is completely undesirable to have patients dead of pulmonary edema with a normal volume of extracellular fluid. I would also re-emphasize that the spectrum of acute renal insufficiency is broad, in that some patients cannot be fitted into this particular category. I have mentioned the fact that patients who have depletion of salt and water may undergo some impairment of renal function but do not appear to fit exactly into the category of acute renal insufficiency. I agree with Dr. Darrow that acute renal insufficiency rarely follows depletion of water and electrolytes, *per se,* and that is why I divided the etiologic agents into shock, chemical toxins and hemolytic disorders.

Physiologic saline is decidedly unphysiologic in that it is hypertonic with regard to chloride and slightly high in sodium. The losses of electrolytes associated with fulminating diarrhea can be replaced quantitatively but with caution. However, the uncomplicated cases, with nausea and small amounts of emesis and diarrhea, are usually no problem, and I would not replace the salt and water in those instances.

If one becomes too eager in an attempt to replace the losses of serum sodium and chloride, the efforts may eventuate in pulmonary edema, since it takes such a small amount of fluid to precipitate pulmonary edema.

Question: Dr. Lowe, if one chooses the sodium content of colostrum as representing the normal intake of sodium in an infant, can you give less than this normal amount after operation on the basis that you might anticipate some retention of sodium associated with the stress response. Secondly, please discuss your two polyionic solutions and their availability.

Dr. Lowe: Dilute solutions that have a concentration of electrolytes somewhere in the range of colostrum would be acceptable for replacement fluid for a newborn child who requires an operation. With the use of such solutions, in more than 100 babies we have never seen postoperative edema. We have seen it in newborn babies, however, when we have used solutions that contain 40 to 60 mEq. per liter. It is my impression, and I think there is experimental evidence to substantiate it, that the newborn infant is extremely capable in handling water and is able to control both retention and excretion of electrolytes.

The solution that we call "Polyionic Solution No. 2" is now on the market and is manufactured by Baxter Laboratories. The other solution mentioned, also prepared for us by Baxter Laboratories, is not yet commercially available.

Question: Dr. Sprague, would you discuss the use of specific alkali substances in the treatment of diabetic acidosis?

Dr. Sprague: We have not been in the habit of administering solutions of sodium bicarbonate or sodium lactate, *per se.* We have been using solutions that contain an excess of sodium over chloride. There are occasional instances in which early in the treatment of acidosis it is desirable to correct an acidosis rapidly in order to relieve hypertonia. This may occur in patients with severe diabetic acidosis that has been precipitated by a myocardial infarction. They experience air hunger even when lying in bed. In these patients it has been rather urgent to correct the acidosis as promptly as possible, and solutions of sodium bicarbonate have been administered with rapid benefit.

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